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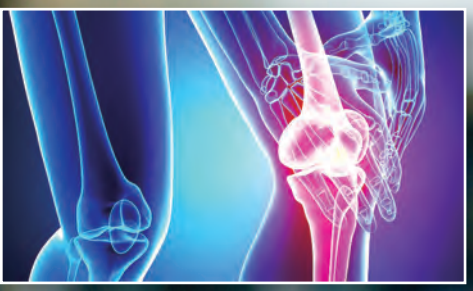
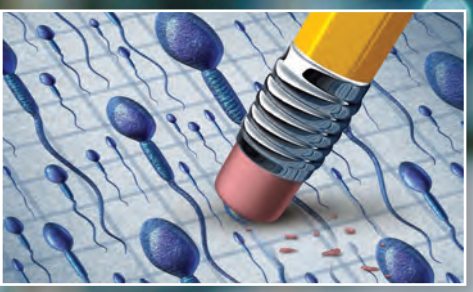
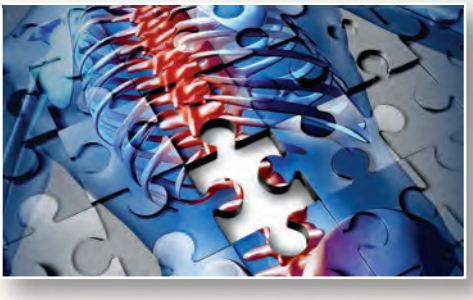


COMMON KNEE PROBLEMS



The management of
INFERTILITY
in Primary Care

FEBRILE CONVULSIONS
The most common seizure disorder in young children



The complexity of
LOW BACK PAIN

BRITISH INTERNATIONAL DOCTORS' ASSOCIATION

www.bidaonline.co.uk

The Journal of the British International Doctors' Association Issue No.3, Volume 20 July 2014 Edition

Annual Representatives Meeting & Annual General Meeting

Friday 10th, Saturday 11th and Sunday 12th October 2014

Thistle Haydock Hotel, Penny Lane, Haydock, Merseyside WA11 9SG
Tel: 0871 376 9044 E-mail: m&e.Haydock.thistle.co.uk

Hosted by THE BRITISH INTERNATIONAL DOCTORS' ASSOCIATION, WIGAN DIVISION

PROGRAMME FOR THE ANNUAL REPRESENTATIVES' MEETING

DR S SARKER *National Chairman* | PROF M S ELJAMEL *ARM Chairman* | DR A K BANERJEE *ARM Vice-Chairman* | DR D TRIVEDI *National President*

Friday 10th Oct

4.00 - 5.00pm	Delegates arrive
7.00pm	Scientific Session: Wigan Division
8.00pm	Dinner - hosted by BIDA Wigan Division

Saturday 11th Oct

The ARM meeting on Saturday 11th between 09:00 & 22:30 is sponsored by **BOEHRINGER INGELHEIM LTD.**

Boehringer Ingelheim will also be exhibiting with a promotional stand.

From 9.00am	Registration & Delegates assemble.
10.00am	Welcome by ARM Chairman, Professor M S Eljamel
10.10am	Proposal by the General Secretary, Dr A Kakkar, to accept the list of representatives for the ARM as the official representatives to the ARM 2014.
10.15am	Presentation of Annual Report by the Chairman, Dr S Sarker.
10.25am	General Secretary's Report, Dr A Kakkar
10.35am	Presentation of Accounts by the Treasurer, Dr B K Sinha.
	Presentation of BIDA Journal Accounts by the Chairman, Dr S Arya.
	Matters Arising from the Chairman's, General Secretary's and Treasurer's Reports.
11.15am - 11.30am	Coffee Break
11.30am - 1.00pm	ARM Resolutions.
1.00pm -2.00pm	Lunch - Hotel Restaurant.
2.00pm -5.00pm	ARM Resolutions followed by 'Afternoon Tea'.
7.00pm -10.30pm	ARM Dinner with Chief Guest.

Sunday 12th Oct

The AGM meeting on Sunday 12th between 10:00 & 12:30 is sponsored by **BOEHRINGER INGELHEIM LTD.**

Boehringer Ingelheim will also be exhibiting with a promotional stand.

10.00am	Delegates assemble
10.05am	Welcome and opening remarks by the National Chairman – Dr S Sarker
10.10am	Introduction of Scientific Lecture. Speaker sponsored by Boehringer Ingelheim Ltd.
10.15am - 10.45am	Dr. Hitesh Patel, Consultant Haematologist, WWL on "The updated NICE AF Guidelines and Anticoagulation"
10.45am - 10.55am	'Question & Answer' session
10.55am - 11.05am	'Open Forum' – Chaired by the National Chairman, Dr S Sarker
11.05am - 11.20am	Coffee Break
11.30am - 12.30pm	**ANNUAL GENERAL MEETING**

Address by the President – Dr D Trivedi

Review of proceedings and Resolutions of the ARM by the General Secretary – Dr A Kakkar

Presentation of Treasurer's Report and Approval of Accounts by the Treasurer – Dr B K Sinha

Presentation of BIDA Journal Accounts by the Chairman of the Editorial Board – Dr S Arya

Election of Association's Auditors and Solicitors for the ensuing year

Awards and Honours

Conference closes

Please note Sunday Lunch is available in the Hotel for those who have given prior notice to Central Office.

THE ANNUAL GENERAL MEETING IS OPEN TO ALL MEMBERS OF BIDA

When time is short, the Chairman will, at his discretion, reduce the length of the discussion

MEMBERS OF THE AGENDA COMMITTEE

Dr D Trivedi	*(President)*	Dr S Arya
Dr S Sarker	*(Chairman)*	Dr A Trivedi
Dr A Kakkar	*(General Secretary)*	Dr A Jha
Dr A K Banerjee	*(ARM Vice Chairman)*	Dr B N Kulkarni
Dr B K Sinha	*(Treasurer)*	Dr R C Rautray
Prof M S Eljamel	*(Chairman of the Agenda Committee)*	

BRITISH INTERNATIONAL DOCTORS' ASSOCIATION

BIDA Journal

Editorial

Contents

Any photographs offered for publication become the property of BIDA Journal and unfortunately cannot be returned.

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Staffing in the NHS

UK has fewer working doctors per head of population than almost all other EU countries. European Commission figures show that the UK has 2.71 practising doctors for every 1,000 people, fewer than countries including Bulgaria, Estonia and Latvia. The UK is ranked 24th out of the 27 European nations, only beating Slovenia, Romania and Poland. By comparison there were more than six doctors per 1,000 people in Greece, which tops the list, nearly five in Austria and just under four in Italy. Although there has been an increase in the number of doctors in the NHS over the recent years, it is still not sufficient to provide safe patient care. Sadly the medical profession is not considered lucrative for many of our youngsters anymore. Doctors in the NHS face increasingly challenging, high-pressured and stressful work environments, often with limited resources and gruelling workloads. Only by making working practices and environments safe and sustainable will the NHS be able attract and retain the required number and mix of doctors.

NHS hospitals under severe staffing crisis

Across the country hospitals are facing a severe shortage of doctors. There are nearly 4000 vacancies, mainly at consultant and Registrar grades. This is raising concerns about patient safety and the ability of the hospitals to cope with the workload. The pressure and demand on the NHS is unrelenting and overwhelming, yet the average vacancy rate for doctors in the NHS hospitals stands at 4.5 per cent. There are not enough doctors in training nor are there fully trained doctors to fill all the vacant posts. These gaps are being filled with locums or by getting staff to work overtime. This is affecting the morale of the medical workforce. The change in the immigration rule is largely responsible for the problem. Urgent attention is needed before more patients' lives are put at risk.

Seasonal variations in coronary heart disease

Coronary heart disease exhibits a winter peak and summer trough in incidence and mortality. In England and Wales, the winter peak accounts for an additional 20,000 deaths per annum. It is likely that this reflects seasonal variations in risk factors. Body weight varies by season, with obesity being more common in winter months. This may in part reflect a higher fat intake in winter. Lower levels of physical activity may also be a factor. Regular physical activity is associated with a reduced risk of CHD, but the benefits reduce within a few weeks of cessation. Many physical activities are only undertaken at certain times of the year and may, therefore contribute to the seasonal variation in CHD risk. In both sexes, overall levels of physical activity are significantly higher in summer than in winter. Interestingly, fasting blood glucose and insulin levels are lower in normal subjects in summer than winter.

Current DVLA guidelines on fitness to drive

It is the duty of the licence holder to notify DVLA of any medical condition, which may affect safe driving. The GMC has issued clear guidelines applicable to such circumstances, which state:

1. The driver is legally responsible for informing the DVLA about such a condition or treatment. However, if a patient has such a condition, you should explain to the patient:

(a) That the condition may affect their ability to drive, and

(b) That they have a legal duty to inform the DVLA about the condition

2. If a patient refuses to accept the diagnosis, or the effect of the condition on their ability to drive, you can suggest that they seek a second opinion, and help arrange for them to do so. You should advise the patient not to drive in the meantime.
3. If a patient continues to drive when they may not be fit to do so, you should make every reasonable effort to persuade them to stop. As long as the patient agrees, you may discuss your concerns with their relatives, friends or carers.
4. If you do not manage to persuade the patient to stop driving, or you discover that they are continuing to drive against your advice, you should contact the DVLA immediately and disclose any relevant medical information, in confidence, to the medical adviser.
5. Before contacting the DVLA you should try to inform the patient of your decision to disclose personal information. You should then also inform the patient in writing once you have done so.

Obesity in the UK

UK is among the worst in Western Europe for level of overweight and obese people. 67% of men and 57% of women are either overweight or obese, with more than a quarter of children also overweight or obese, 26% of boys and 29% of girls. Obesity raises the risk of diabetes, heart disease and cancers. Obesity, defined as a body mass index (BMI) of 30 or more, while overweight, defined as BMI of over 25, has increased by 10% in the UK over the past three decades. The rapid increase in child obesity is particularly disturbing, as being overweight at a young age can set children up for a lifetime of poor health. We need to be seriously thinking now about how to turn this trend around.

Have a lovely summer and hope you enjoy reading another issue of the BIDA Journal.

Dr Sanjay Arya
Editor, BIDA Journal
Consultant Cardiologist,
Wrightington, Wigan & Leigh NHS Foundation Trust

Department of Health

From the Rt Hon Jeremy Hunt MP
Secretary of State for Health

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79 Whitehall
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SW1A 2NS

Tel: 020 7210 4850

10 JUN 2014

P000000860258

Dr Sabyasachi Sarker
National Chairman
British International Doctors' Association
'ODA House'
316A Buxton Road
Great Moor
Stockport SK2 7DD

Dear Dr. Sarker,

Thank you for your letter of 6 May about Alder Hey Children's NHS Foundation Trust.

As you know, both the Care Quality Commission (CQC), the independent regulator of health and social care, and Monitor, the independent regulator of foundation trusts, have been informed about patient safety concerns at the Trust.

I understand that the Trust is addressing issues that were noted in the CQC's report published in February, following an inspection of the theatre department, which found that the Trust was not meeting four of the five standards assessed. A copy of the report can be found at www.cqc.org.uk (by searching for 'Alder Hey').

I can assure you and your members that this Government wants to see a major cultural change around openness and transparency in all health and social care organisations. We expect such organisations to encourage the raising of concerns by staff as a positive step to improving public and patient safety, and for the concerns raised to be investigated and acted upon.

As the report on the Francis Inquiry into Mid Staffordshire NHS Foundation Trust showed, it is essential that both the public and staff should be encouraged and supported in speaking out when they have complaints and concerns about the safety of patients.

That is why in the Government's response to the Francis Inquiry, Hard Truths: The Journey to Putting Patients First, we set out how we plan to build the safe, effective and compassionate culture that patients, the public and the overwhelming majority of staff across health and social care expect, making sure whistleblowers are not just protected, but also praised for their courage and thanked by management.

As you will understand, this is a very ambitious agenda. We are therefore giving further thought to how we can be satisfied that in every hospital, practice and care setting, staff feel able to speak out with their concerns. I will write to you further once we are in a position to share our thinking.

Thank you once again for your association's interest in whistleblowing issues.

Yours sincerely

Jeremy Hunt

JEREMY HUNT

A Joint Statement

by BIDA, RCGP and BAPIO

The RCGP, British Association of Physicians of Indian Origin (BAPIO) and British International Doctors Association (BIDA) have announced that they will be working in close collaboration to address supporting international medical graduates and Black and Minority Ethnic doctors in relation to training and passing the MRCGP

At a very positive and productive meeting held at the College, the three organisations pledged to work together to determine what support could be offered to identify struggling trainees at an early stage and improve their training experiences in order to better prepare them for the MRCGP and for safe independent practice. The RCGP shared some of their specific plans to support trainees and trainers such as developing e-learning resources for Clinical Skills Assessment preparation (based on sociolinguistic research) and reviewing ways to enhance CSA feedback to candidates. BAPIO and BIDA were both very supportive of these initiatives

RCGP Chair Dr Maureen Baker said: "We are very pleased to now be working in partnership with BAPIO, BIDA and other key stakeholders to look at solutions and find the best way of supporting the small number of trainees who fail the CSA component of the MRCGP licensing exam to give them every chance of passing."

This move follows the Judicial Review hearing at the High Court in April in which the Clinical Skills Assessment (CSA) was judged to be lawful.

Dr Baker added: "It was very reassuring to have the High Court judgment rule out the claims of discrimination.

"As the High Court ruling highlighted, patient safety is the key purpose of the MRCGP exam and the College must have total confidence in those who pass the exam, having clearly demonstrated the appropriate skills and clinical knowledge.

"The College is not at all complacent and we are keen to move forward in a number of areas - not just those that were raised in the Judicial Review - for the benefit of patients and trainees. We discussed this with BAPIO and BIDA and received their support.

"We take multiple steps to ensure that our exam is robust but fair to all candidates and the overall failure rate for trainees wishing to enter general practice is very low.

"But as an organisation committed to equality and diversity, we have always been, and remain, concerned that international medical graduates do not do as well in the exam as those from UK medical schools. Indeed, we were the first of the Medical Royal Colleges to publicly raise this issue and have commissioned and supported extensive research to understand what is happening and to try and identify what the causes may be. "We look forward to working with BAPIO and BIDA in a renewed spirit of collaboration and co-operation."

In welcoming the decision to collaborate Dr Ramesh Mehta, President of BAPIO said: "We have had a very fruitful discussion with the RCGP. We are pleased that the Royal College has identified several steps to implement the equality impact assessment. We also discussed the issue of those trainees who have been removed from the training and the possibility of them getting back in to General Practice. We are looking forward to working constructively with the Royal College for fairness and professional excellence in the interests of doctors and patients.

"It is time to make progress and we welcome the proactive approach of the RCGP to provide much needed relevant support to the international medical graduates and Black and Minority Ethnic doctors in relation to training and passing the MRCGP."

Dr Bachi Sarkar, National Chairman of BIDA said: "We are glad that the College is appreciative of the immense contribution of the International Medical Graduates to the General Practice".

20th June 2014

An update about ongoing MRCGP CSA Examination Issues

1. Overview

BAPIO made 3 claims against the RCGP in respect of the CSA

- That the RCGP did not comply with their public sector equality duty (PSED) under Section 149 of the Equality Act 2010
- That the CSA directly discriminates against IMG and BME candidates
- That the CSA indirectly discriminates against IMG and BME candidates

The Judge dismissed all 3 claims against the RCGP on 10 April, but agreed to consider an appeal against the RCGP in relation to PSED, as there were novel points of law within his judgment.BAPIO later decided not to pursue the appeal.

In the course of delivering his judgment, the Judge clarified the College's PSED responsibilities and made some recommendations on future areas to be addressed.

2. Public Sector Equality Duty (PSED) requirements

The Judge ruled that the College has a PSED, but that the RCGP is not a public authority and so its duties are limited to the public functions it exercises, which are defined by its Royal Charter. Since the RCGP conducts and awards the MRCGP, it has the power to determine who meets the standards to be a GP in the UK and this is a matter of public importance because of patient health impact.

He saw differential pass rates as a "long standing problem" for which reviews/research alone might not be sufficient because the Equality Act does not permit such a body: *"to identify the need to eliminate discrimination in one of the public functions it exercises and then do nothing about it. If it acted thus it would not be "having regard" to the need to eliminate discrimination in the exercise of its public function; it would be* ***disregarding*** *a specific need which it had itself identified."*

He concluded that RCGP has identified the respect in which it needs to act to eliminate discrimination in the discharge of its public functions *"and has identified some of the means by which that need might be addressed and fulfilled. The time at which it should act upon the information which it has gathered and analysed has either arrived or will do so very soon. If it does not act and its failure to act is the subject of a further challenge, it may well be held to be in breach of its duty under section 149 for that reason alone."*

3. Judge's recommendations for RCGP actions

The Judge stated that it is well within the scope of RCGP public functions to consider taking steps such as those he identified below, and should consider taking such steps now, otherwise it might be failing to discharge its duties under s149 in future.

He advised:

- An urgent need to try to address the differential CSA outcomes by further reviewing research findings and recommendations.
- GP training is the main area to address, primarily by encouraging and supporting Deaneries/LETBs all to provide high quality training to doctors, familiarisation with the CSA to equip them to deal with the assessment, to provide remedial training for those who have failed at first attempt and special assistance to support struggling IMGs/BMEs
- RCGP should work with other bodies to attract more doctors into general practice and assist them to maintain the highest possible standards
- RCGP should recruit a more representative group of examiners from diverse backgrounds.
- Although there is no legal obligation to formally record EIAs, RCGP should keep records of all such discussions/decisions. (Courts will not interfere with decisions made provided that due process has been followed.)

4. Claims of direct and indirect discrimination

a) Direct discrimination claim

The judge concluded that there were no grounds for a claim of direct discrimination. He ruled that: *"The statistical differences which exist do not of themselves establish direct discrimination."* Rather, that they *"demonstrate that there is a difference of outcome. They do not establish the reason or reasons for the difference, still less that it is because the Royal College, or individuals for whose actions the Royal College is responsible, are subjecting doctors who fail the assessment to less favourable treatment on a prohibited ground."*

b) Indirect discrimination claim

The judge accepted that the CSA was more difficult to be passed by South Asians of either IMG or BME background, but fully agreed that the CSA is "a proportionate means of achieving a legitimate aim", i.e. that it is sufficiently 'high stakes' to guarantee patient safety with GPs licensed to practice in an unsupervised capacity, and that it is necessary to test skills in information gathering, diagnosis and communicating with patients.

He acknowledged that the CSA is the best means of testing these skills yet devised, is in common use worldwide and that the MRCGP has a very small eventual failure rate. He also accepted that the treatment of the measurable error of assessment must act in favour of patients rather than doctors.

He flagged that the external 2013 GMC review of the CSA undertaken by Professors Esmail and Roberts stated that:

"The CSA is not a culturally neutral examination and nor it is intended to be. It is not and nor should it be just a clinical exam testing clinical knowledge in a very narrow sense. It is designed to ensure that doctors are safe to practise in UK general practice. The cultural norms of what is expected in a consultation will vary from country to country... British graduates have much greater exposure, both personally and through their training, to general practice when compared to the majority of IMG who graduate from health systems which are not as dominated by primary care as the NHS. Most medical schools in the UK now have well developed programmes for communication skills training, reflective practice and direct exposure of students to General Practice as a discipline."

He therefore ruled that: *"There is no basis for contending that the small number who fail ultimately do so for any reason apart from their own shortcomings as prospective general practitioners."*

He concluded: *"I am satisfied that the Clinical Skills Assessment is a proportionate means of achieving the legitimate aim identified. For those reasons, the claim against the Royal College must be dismissed".*

However, he added that he was: *"satisfied that this claim has been brought in good faith and in the public interest by an organisation acting for a proper purpose". Also that: "the bringing of this claim is likely in the end to produce something of benefit for the medical profession and so for the public*

Dr.Chandra Kanneganti
MBBS,MRCGP,DFFP,PGc Med Ed, MSc Medical Leadership
Principal GP, Goldenhill Medical Centre & Five Towns GP Surgery
CCG Clinical Director, Community Care, NHS Stoke CCG.
BMA GPC Member
BIDA National Executive Member

generally. The claim was only brought by the Claimant when it felt that no other course was open to it to attempt to rectify an anomaly acknowledged by all to exist. This claim has served a useful purpose and the Claimant has achieved, if not a legal victory, then a moral success."

In the light of his views about BAPIO's actions 'in the public interest', he only awarded £50k against BAPIO to cover legal costs accumulated by RCGP and GMC, with this sum to be split between them as agreed.

BIDA has supported BAPIO in this legal case with BIDA chair Dr.Sarker and National Exec Member Dr.Chandra Kanneganti active involvement.

After the high Court Judgement, Both Dr.Sarker and Dr.Kanneganti along with Dr.Ramesh Mehta and Dr. Joydeep Grover of BAPIO met the college officers including RCGP Chair Maureen Baker, RCGP Chief Executive Neil Hunt, Chief Examiner Pauline Foreman on 12th June 2014.

It was decided in this meeting for RCGP to work collaboratively with BIDA and BAPIO and number of actions agreed to move forward which were later presented to RCGP council on 13th June 2014.

ACTIONS AGREED:

In order to ensure that the RCGP continues to comply with its PSED, the MRCGP team intends to undertake the following specific actions.

1) A formal equality assessment of the MRCGP

2) PSED training for the MRCGP team including examiners

Examiners and role players already have equality and diversity training that is tailored to their specific roles.

Examiner equality and diversity training is completed annually at the examiners' conference and generally focuses on a different protected characteristic each year. .

3) A strategic MRCGP plan to address differential performance

a) Actions which the RCGP can take

The MRCGP team is currently working through a number of recommendations suggested by the GMC/Norcini reviews including:

- Increasing the length of the AKT timing by 10 minutes for all candidates to reduce time pressure, which may particularly benefit candidates whose first language is not English (planned for Oct 14 subject to GMC approval).
- An increase in the number of CSA diets to increase flexibility for candidates and deaneries/LETBs from October 14 (GMC approved).
- New resources for CSA preparation for trainees and trainers based on new sociolinguistic research. Two new e-modules and a book are planned for Autumn 2014.
- Consideration of a move to the borderline regression method of standard setting for the CSA, which is potentially more reliable than the current system (initial feasibility pilots in progress).
- A review of quality assurance of examiner performance.
- Improved formative feedback from the CSA. Summated domain scores will be available to candidates from October 14 in addition to their overall score and the daily pass mark. Two different methods of providing additional formative feedback are currently being piloted.
- Continuing work to ensure a more representative panel of examiners. We have been encouraging applications from IMG/BME examiners from 2010 with some success. We have recently changed the new examiner application requirements so that an AKT pass lasts for 10 rather than 5 years. Our equality analysis screen suggests that this will encourage applications from younger, female, IMG/BME candidates who are relatively under-represented on the panel.

b) Actions with stakeholders

Work with the GMC / HEE / Deaneries / LETBs / COPMED / COGPED to improve the quality of GP training in order to reduce underperformance by IMG and BME candidates in the MRCGP. The focus needs to be on identifying candidates at risk of poor performance, and supporting them. This could be achieved by:

- Supporting relevant joint research proposals, e.g. current GMC project to look at NRO selection data and outcome in the MRCGP.
- Encouraging HEE/deaneries/LETBs to evaluate current training interventions for struggling trainees. Interventions that are already taking place across the UK include:
  - Optimising the training environment, e.g. trainees with the lowest selection score going to the 'best' practices, or advanced training practices
  - Extra skills training, e.g. communication skills training including language skills
  - Extra support, e.g. IMG conferences, facilitated CSA study groups
  - CSA exam practice circuits with feedback
- Support for GP Trainers and Training Programme Directors (TPDs) including:
  - A programme for Trainers to visit the CSA, prioritising deaneries with the highest failure rates.
  - CSA training courses for Trainers run locally through the Faculties
  - A template for running a mock CSA circuit for TPDs.
  - Encouraging Trainers to benchmark their WBPA assessments with other trainers and utilise the new indicators of potential underperformance in the e-portfolio to pick up struggling trainees earlier.
  - Development of a joint statement with COGPED on minimum standards for training for the CSA. This could potentially include guidance on observation and feedback, ensuring an adequate case mix and maximising the opportunity to consult with a diverse group of patients.
- Joint working with the AoMRC / BMA / BAPIO / BIDA / and other relevant stakeholders to highlight research into the cause of differential exam outcomes for IMG and BME candidates and develop effective solutions.

BIDA and BAPIO are thanked by Dr. Maureen Baker for offering to work collaboratively to support the above actions.

Common Knee Problems

Introduction

The knee is the second most commonly injured joint in the body after the ankle. It is also a common joint to develop degenerative conditions such as arthritis, with the population at risk of developing arthritis in the knee being about 1% of the population. As the population ages, the number of individuals with symptomatic arthritis is also going to rise.

Anatomy / Physiology

The knee joint is the largest synovial joint in the body. It is made up by 3 bones comprising the distal femur and proximal tibia, which make up the major articulation of the knee joint. There is a smaller, but equally important articulation between the femoral trochlear and the patella (a sesamoid bone within the quadriceps tendon), which makes up the patellofemoral articulation (Figure 1).

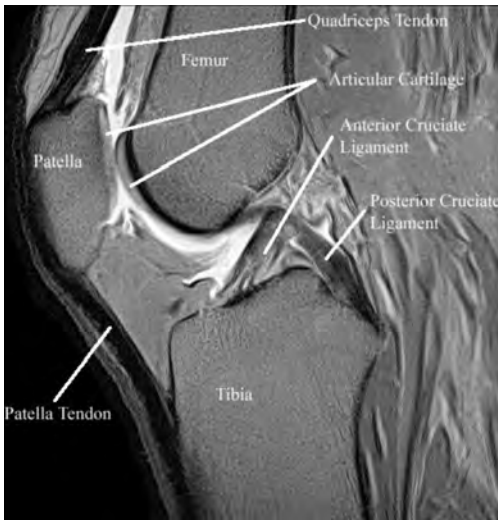

Figure 1 – Saggital MRI image showing anatomy of the knee. (ortho pics – knee pics)

The function of the knee joint is made possible due to various static constraints, such as the articular cartilage menisci ligaments and bones combined with the dynamic functions of the surrounding muscles and tendons (Figure 1).

Any of these structures can be injured or become deranged to adversely affect the function of the knee joint and create the commonly experienced symptoms of pain and swelling, locking, giving way and so forth.

The menisci have a unique role in the knee joint to increase the surface area of the articulation between the femur and tibia and therefore reduce force transmitted from the femur to the tibia due to the innate structure of menisci. The particular anatomy of the menisci that allow this are the collagen fibres that run circumferentially from the anterior horn to the posterior horn (Figure 2). The continuity of these fibres produces the biomechanical features of so called "hoop stresses". This is where a force acting to compress the meniscus is transmitted into a force which is then dissipated through the circumferential fibres around the meniscus and outwards. This function can only occur if the fibres are in continuity and the commonest way that this continuity fails is when menisci tear. This tearing leads to disruption in the hoop stresses, which means the force can no longer be dissipated in that part of the meniscus. The force, therefore, increases in that area between the femur and the tibia. Articular cartilage is not designed to constantly take high force loading, which if occurs, will result in the articular cartilage failing, breaking down and ultimately the articular cartilage will be lost to form an osteoarthritic lesion.

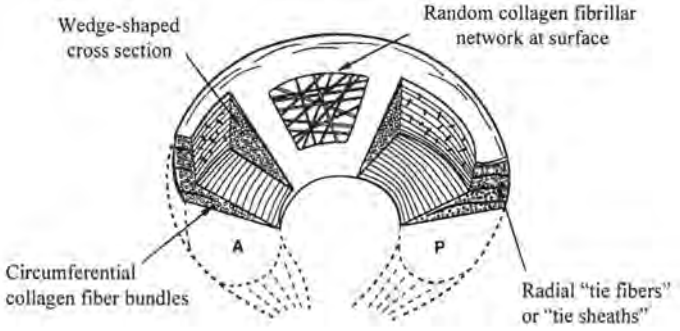

Figure 2: Anatomy of meniscus. (ortho pics - knee pics – knee misc)

This failure of the meniscus in force transmission will occur as soon as the meniscus tears and it is not so much the removal of the meniscus that causes the problem, but the tearing itself. Once the meniscus is torn, it has, in essence, functionally been removed from the knee causing increasing loading in that part of the knee (Figure 3).

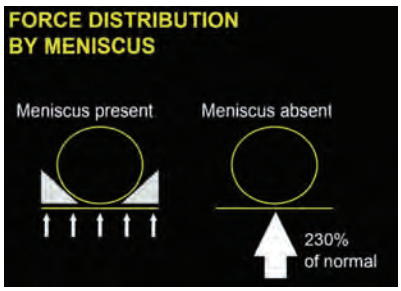

Figure 3: Effect of meniscal tears / removal on force transmission in the knee. (ortho pics – meniscal tears)

There is an innate relationship between anatomy and physiology, as is demonstrated by the above example in the knee.

The patellofemoral joint will take up to 8-10 times an individual's body weight when the individual undertakes activity such as going up and down stairs, or running and jumping. It is, therefore, not difficult to see why the patellofemoral joint commonly is a source of degeneration and symptoms.

Clinical Knee Conditions

Knee pathology can generally be divided into traumatic and degenerative.

The traumatic group can be subdivided in to acute traumatic episodes, such as fractures, dislocations, ligament ruptures and the chronic trauma or overuse group. The overuse group would commonly be conditions such as tendonitis or even meniscal tears.

The degenerative group may occur as a result of previous acute overuse trauma with the final common pathway being osteoarthritic changes within the joint or chronic degenerative changes occurring within the joint.

Before considering individual pathology, it is prudent to comment on diagnosis.

As we have all been taught during our medical school careers, 80% of the diagnosis of any condition is made from the history with a further 10% or so being confirmed on the examination. The same applies to diagnosing common knee problems and therefore an innate part of treating our patients is the ability to obtain a good history in order to provide a diagnosis or at least a differential diagnosis.

In many conditions, the history is not difficult to obtain. For example, in individuals with progressive osteoarthritis, the history will be a progressive increase in pain and reduction in function, together with complaints of swelling, sleep disturbance, limitation of walking and there may be even mechanical symptoms such as locking, catching and giving way. A little bit more challenging is the diagnosis in knee injuries. If an individual is questioned closely on facets of their history it is not uncommon to be able to diagnose their traumatic pathology.

If one is seeing an individual with a knee injury, a few minutes going in to their mechanism of injury can be invaluable diagnostically. The sort of factors to consider include enquiring whether the individual's injured knee had the corresponding foot planted on the ground at the time of their injury, or off the ground. Most serious knee injuries tend to occur when the individual's foot is on the ground and the weight is on their leg.

It is important to enquire whether their knee sustained a direct blow and, if so, from which side. This is a common mechanism for tearing, primarily, the collateral ligaments depending upon the side of injury. If the leg is planted on the ground and the individual is struck from the lateral side, it will commonly be a medial collateral ligament injury and vice versa.

An individual who provides a history along the lines that they were running, suddenly planting their foot to change direction, felt a click or a pop, fell to the ground, the knee swelled up and they were unable to continue playing, is a classical history for an anterior cruciate ligament (ACL) tear. Differential diagnoses would include an acute patella dislocation.

Following a knee injury, one should enquire whether the knee swelled and if so, how quickly. If swelling occurs within seconds to minutes of the knee injury occurring, this would indicate bleeding into the joint (hemarthrosis). 80-85% of acute hemarthrosis with an appropriate history should be considered to be a tear of the anterior cruciate ligament until proved otherwise. Other causes for acute hemarthrosis would be patella dislocations, large meniscal tears or intra-articular fractures.

Injuries occurring when an individual is jumping, landing, or changing directions, are classically an ACL tear or potentially a patella dislocation.

If an individual falls and lands on their knee, enquiry of whether the foot at the time of the landing was dorsiflexed or plantar flexed can provide some diagnostic information. Generally, if the foot is plantar flexed, the individual lands on their tibial tuberosity, which if injuring the knee will cause a tear of the posterior cruciate ligament (Figure 11). If the foot is dorsiflexed the individual will usually land on the patella, sustaining an injury to the patellofemoral joint.

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Knee Pathology

Bursas

There are many bursas around the knee (Figure 4). The common bursas seen include the pre-patellar bursa (housemaids knee) (Figure 5) and infrapatellar bursa (clergyman's knee). The diagnosis is usually self-evident from the swelling. The onset can be traumatic or of gradual onset. With the anterior knee bursas, there is usually history of repetitive kneeling.

The treatment is generally similar with initially rest, ice and anti-inflammatories. Aspiration and injection of steriod can be considered in a clean environment so as to minimise the potential risk of infection. The final option of treatment is surgical removal, which may be done open or arthroscopic. The open route is a more common. It is however important the patient is aware they will be swapping a lump for a scar. Sometimes the scars can be as troublesome as the bursas / lump that were present previously, especially in scenarios where the individual is going to be kneeling. With all treatment methods there is a risk of recurrence, but this is obviously lowest with surgical removal. Generally, if the bursa does not bother the individual then they can be simply observed.

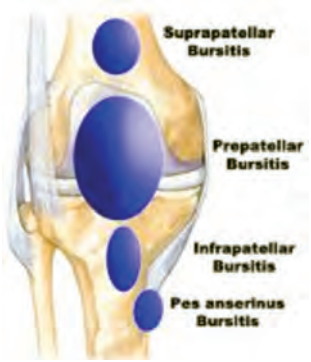


Figure 4: Bursas around the knee (otho pics - knee misc)

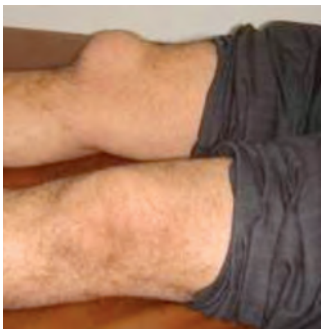


Figure 5: Pre patella bursa right knee (housemaid's knee) (otho pics - knee misc)

Meniscal Tears

Meniscal tears are very common pathology, encountered by GPs as well as Orthopaedic Surgeons. Occasionally a history of acute trauma is provided with an individual either twisting or stumbling on their knee. More often than not, with degenerative meniscal tears, there is no specific history of injury or overuse. Commonly an individual will complain that they woke up one morning and the knee was painful and swollen and they had difficulty walking. Pain is usually localised to the side of the meniscal tear, either medial or lateral. Generally their symptoms will gradually get better and could be treated expectantly with ice, anti-inflammatories, rest and elevation.

Meniscal tears generally have no capacity to heal themselves. The reason being there is no adequate blood supply running through them (Figure 6). Therefore, if the tear does not heal, although the symptoms can improve, generally will tend to reccur when an individual aggravates their knee. The aggravation commonly will occur with activities involving twisting, kneeling or squatting, as these all put rotational forces on the meniscus generating pain and swelling. Therefore, while individual's symptoms can improve, with meniscal tears symptoms tend to recur with the frequency of recurrence becoming more so as time goes on.

It should also be borne in mind that not all meniscal tears are symptomatic. Indeed, it is estimated that about 30% over the age of 40, with no knee symptoms, will be found to have asymptomatic meniscal tears on an MRI scan. Therefore, not all meniscal tears require treatment unless symptomatic.

The key examination findings of meniscal tears are joint line tenderness over the medial or lateral aspect of the knee. It is generally considered that meniscal joint line tenderness tends to occur towards the posterior half of the meniscus, either medially or laterally (Figure 7). The joint line tenderness tends to be maximal at one point.

There are a number of meniscal tests that are described (Figure 8). They all have the common feature of trying to increase an individual's pain by trapping the torn meniscus. Examples include McMurray's test. The key to these tests is to ensure that the patient's knee is flexed deeply and then rotated into different positions of flexion or extension. The key, however, is the deep flexion, as this is the position at which the meniscus gets trapped. A positive test would be reproduction of pain over the sight of maximal tenderness, occasionally with a click, although the click is not mandatory.

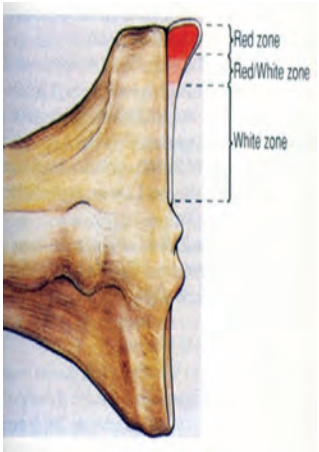


Figure 6: Blood supply of meniscus. There is some blood at the capsular margin of the meniscus (red zone) but this diminishes towards the free edge of the meniscus (white zone). (ortho pics - meniscal tears)

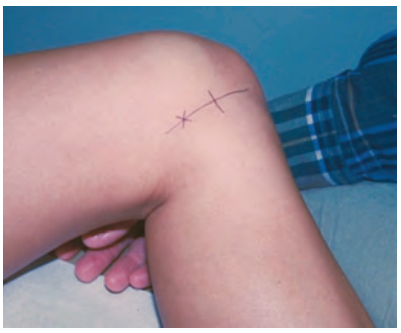


Figure 7: Medial side of Left knee. Clinically, meniscal tear pain on palpation is usually at point X or posterior to the vertical line, which is the half way point of the joint line. Pain or tenderness anterior to the line is not usually meniscal in origin.

The diagnosis can be made on the history and examination in the vast majority of patients with meniscal tears. Indeed, studies have been found to show that experienced clinicians are more accurate a diagnosing meniscal tears than MRI scans. It also should be borne in mind that MRI scans are not 100% sensitive at picking up meniscal tears and can miss up to 10-15% of meniscal tears. However, if the diagnosis is suspected, an MRI scan would be the investigation of choice so as to illustrate the meniscal tear (Figure 8)

There are many types of meniscal tear (Figure 9). The key principal in treating meniscal tears is to preserve as much normal meniscal tissue as possible for the reasons outlined in the previous discussion, ie to try to keep loading forces down in the knee (Figure 3).

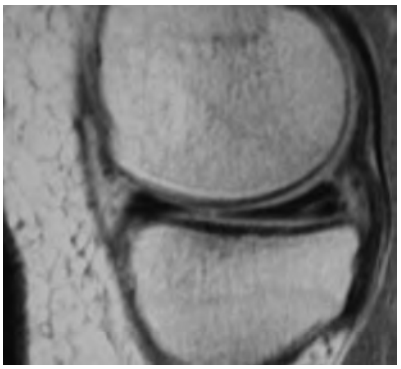


Figure 8: Meniscal tear seen on an MRI scan. The tear is a white line through the black triangle representing the meniscus. (ortho pics - meniscal tears)

Initial treatment options include conservative treatments. Consideration may also be given to a steroid injection intra-articularly, which will reduce an individual's symptoms of pain and swelling. It is usually, however, a temporary phenomenon and sooner or later the symptoms tend to recur once the steroid wears out.

Ultimately, for symptomatic meniscal tears, the treatment tends to be surgical with an arthroscopic partial meniscectomy, leaving behind as much normal tissue as possible. If it is an acute meniscal injury, which appears traumatic on imaging or at the time of arthroscopy and is peripheral and close to the capsular margin (which has a blood supply) (Figure 6), consideration should be given to meniscal repair. The reason being, that if the meniscal is repaired early on, the tear is non-degenerate and goes on to heal, it is possible to return normal meniscal function to the meniscus.

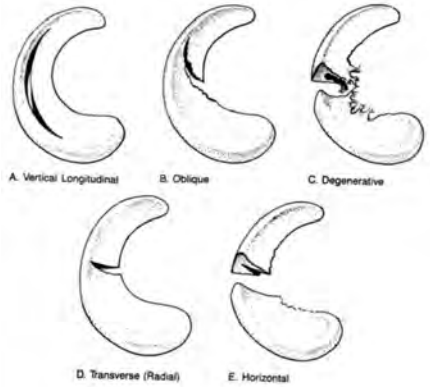


Figure 9: Different types of meniscal tears. (ortho pics - meniscal tears)

Generally, medial meniscal tears are much more common than lateral meniscal tears. The reason being, that most individuals have their body weight axis running through the medial compartment, which will therefore load and more likely injure the medial meniscus.

Occasionally, in young individuals, a large piece of meniscus has to be removed, such as in irreparable bucket handle meniscal tears. In this group consideration can be given to meniscal transplantation to try and preserve knee function.

Common Knee Problems

(continued)

Anterior Cruciate Ligament Tears (ACL)

The ACL is a commonly injured ligament in the sporting environment but can also occur in slips and falls. ACL tears are a major traumatic injury to the knee, as significant forces have to be imparted to the knee joint to tear the ligament. Consequences of an ACL tear are also significant to the extent that an individual who tears their ACL has a ten-fold increase in their risk of arthritis development within their knee.

The diagnosis can be made through a detailed history. The classical history of an ACL tear is an individual running that suddenly stops or reacts, to change direction, the knee gives way, they may hear a pop, they fall to the ground, the knee swells up immediately and they are unable to play on. Given this history, an ACL tear should be assumed until proven otherwise.

In the acute examination situation the knee will be markedly swollen with very little movement and will be painful. The initial treatment is to ice the knee, rest the knee, elevate it, and to take anti-inflammatories and painkillers, together with physiotherapy. The aim is to reduce swelling and start to increase the range of motion. If there is any concern regarding the mechanism of injury, x-rays should be arranged to exclude fractures. Within a couple of weeks of this type of treatment, the knee can be re-examined once it has settled somewhat. The classical findings on examining an individual with an ACL tear are laxity of the anterior cruciate ligament, which can be diagnosed on a Lachmann's test or an anterior drawer (both these tests assess how much anterior movement there is of the Tibia on the Femur as the Tibia is pulled forward. The injured side should be compared to the normal knee). If there is any concern or suspicion of a torn ACL, they can be referred to the Orthopaedic Surgeons for further assessment and treatment. An MRI scan can be arranged to show the tear (Figure 10).

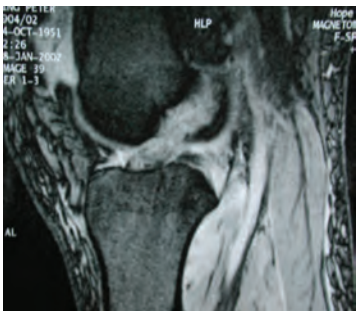


Figure 10: MRI scan of the knee showing a tear of the ACL. The normal ACL is a black structure on an MRI scan, whereas on this scan it is all white (bleeding) with no fibres seen running through it. (ortho pics - ACLuk)

The function of the anterior cruciate ligament is to provide rotational stability of the knee. Once the ACL is completely torn, it does not have the ability to heal in general terms. Individuals with ACL tears lack rotational control of their knee, such that if they plant their leg to change direction the knee will usually give way. This may occur if they return back to sports, or can occur in their everyday activities. The majority of individuals with ACL tears usually will require an ACL reconstruction.

Posterior Cruciate Ligament (PCL) Injuries

The PCL is much less commonly injured, compared to the ACL. The diagnosis is not uncommonly missed. The PCL can and usually does heal. A PCL tear is not as disabling as an ACL tear. Most individuals with isolated PCL tears do not require surgical treatment.

The examination can confirm the diagnosis of PCL injuries, although the examination findings can be subtle, compared to an ACL tear. The test that is classically used is a posterior drawer. In this test, posterior translation of the tibia on the femur is assessed at 90 degrees of knee flexion.

Comparison should always be made to the normal knee to get an idea of what is normal for the individual.

The common mechanism for tearing the PCL is a fall on to the front of a bent knee (Figure 11), or the bent knee being struck by an object such as a dashboard in a road traffic accident. They can occasionally occur in a sporting environment with rotational mechanisms. Typically, individuals with PCL tears will complain of posterior knee pain with only minor degrees of swelling.

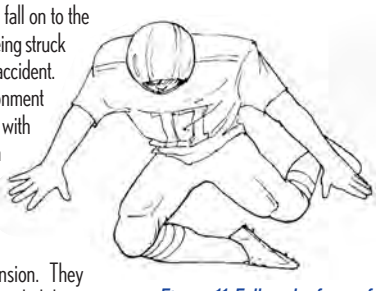


Figure 11: Fall on the front of the bent right knee with the foot plantar flexed can tear the PCL.

The treatment for an acute PCL injury, of lower moderate grade, would be to splint the knee in extension for 4-6 weeks and prevent hyperextension. They would then be put through a physiotherapy rehabilitation programme. Individuals with healed PCL tears will commonly return back to all their normal activities, including high level sports.

Collateral Ligament Injuries

Collateral ligament injuries usually occur from direct blows to the medial or lateral aspect of the knee or lower leg. They are usually isolated injuries but can occur in combination with the ACL or PCL, such as in sporting environments.

The usual examination findings are localised tenderness over the medial or lateral structures with laxity of the ligaments on varus or valgus stressing. The laxity should be compared to the normal knee, as some degree of laxity of the collaterals is present in everyone's knees normally. In low grade injuries, laxity is present at about 30° of knee flexion. In high grade injuries (grade 3), laxity is present with the knee in full extension which would indicate that the knee capsule is also injured. In this latter situation, the individual would be best served being referred to the Orthopaedic Services.

Medial collateral ligament injuries will usually heal in all grade of injury. The treatment would be to initially immobilise the knee in a cast or brace, followed by a graduated physiotherapy-based programme. Surgical treatment to acute medial collateral ligament injuries is very uncommon in isolation.

Lateral side injuries, however, are different and commonly may not heal. The lateral side of the knee has 6 major anatomical structures which contribute to the stability of the knee. While individual injuries to one or two of these structures can be tolerated by the knee, if three of more are injured usually surgical treatment will be required.

With lateral side injuries, these commonly occur in conjunction with ACL or PCL tears. With acute lateral side ligament injuries, the ideal option is surgical repair within the first 10-14 days. Therefore, if lateral sided injuries are suspected, it is recommended to refer the individuals acutely to the Orthopaedic Services.

Osteoarthritis of the knee (localised and generalised)

Osteoarthritis is a very common and increasing common condition seen by GPs and Orthopaedic Surgeons. It is estimated that 1% of the population will develop arthritis in the knee requiring treatment. As the population ages, the number of individuals with symptomatic arthritis will also increase.

Arthritic symptoms have a progressive onset usually, although acute flare-ups can occur. Arthritis can occur constitutionally, but more and more post-traumatic arthritis is now being encountered in younger individuals. This can occur following intra-articular fractures, major ligament injuries to the knee, such as the anterior cruciate ligament, or following sub-total menisectomies in meniscal tears.

The patient will complain of pain, which may be localised to one compartment, but more often is generalised. They will commonly complain of swelling, reduced walking distance, and as the disease progresses, may also develop rest pain and night pain. They can also develop deformities such as the more common, varus (bow legged) (Figure 12) and less common valgus (knock kneed) deformities. They will also lose the ability to fully straighten their knee and develop fixed flexion contractures, which also can affect an individual's ability to walk.

Figure 12: Varus (bow legged) deformity of advanced knee arthritis Right knee. (Kneedoc pics - Bowlegged)

These features are also picked up on when examined, with reduced range of motion and fixed deformities, together with swelling. This diagnosis can be confirmed radiologically on plain x-rays (Figure 13). In the earlier stages, or in individual's with localised areas of arthritis (chondral or osteochondral defects), MRI scanning can be diagnostic.

The treatment is dependent upon the distribution and severity of the arthritis. In young individuals who sustain significant traumatic injuries to their knee, such as patella dislocations or ligament injuries, they can develop localised areas of traumatic arthritis (chondral defects) (Figure 14). This can also occur in developmental conditions such as osteochondritis desiccant. In these conditions, in young individuals, the goal of treatment is to try and preserve or regenerate articular cartilage.

The treatment options will include procedures such as microfracture (Figure 15), cartilage regeneration techniques, or articular cartilage transplantation (Figure 16). These techniques are undertaken by specialist knee surgeons and have good success rates, having been undertaken for over 15 years now. Individuals with these pathologies require specialist referral.

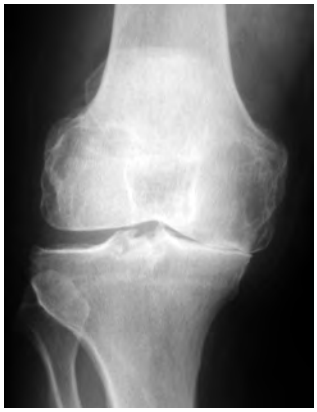


Figure 13: Advanced medial compartment knee OA Right knee. (Ortho pics - TKR - Case 4A)

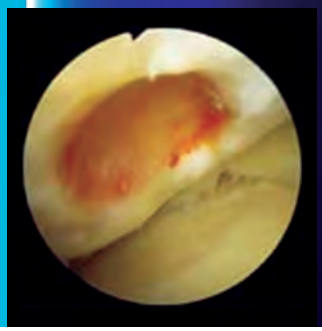


Figure 14: Traumatic chondral defect of medial femoral condyle. (ortho pics - osteochondral - acd big)

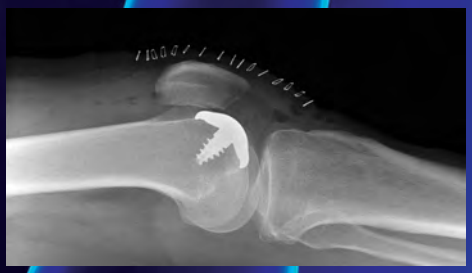


Figure 18: Xray of patellofemoral replacement (isolated trochlear replacement).

In more advanced localised arthritis, which may for example involve both sides of the joint such as the femur and tibia or the patella and the femur, cartilage transplantation options are not as effective and may be contraindicated. In these scenarios one would consider localised arthroplasty options, such as unicompartmental knee replacements (figure 17) or isolated patellofemoral replacement (figure 18).

The role of arthroscopic procedures in arthritis is more controversial. There is a place for it, but generally the days of lavage and washout of arthritic joints has gone, as there is good evidence to show that this does not work and may in fact make the individual worse. Patients, however, with localised mechanical findings, such as joint line tenderness with locking, catching and giving way, may benefit from arthroscopic procedures to debride their meniscal tears and remove any loose fragments or loose bodies. Arthroscopies in this group of individuals can be quite successful.

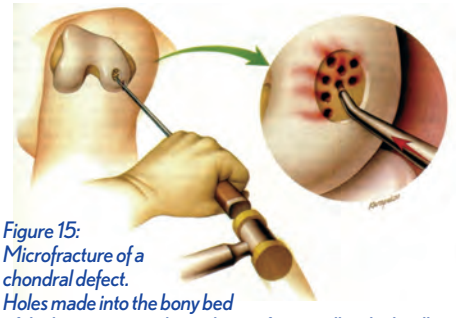


Figure 15: Microfracture of a chondral defect. Holes made into the bony bed of the lesion, to stimulate release of stem cells, which will transform into cartilage. (ortho pics - osteochon leis)

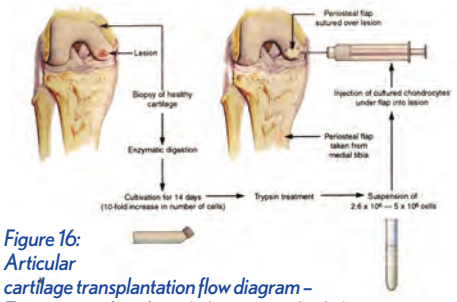


Figure 16: Articular cartilage transplantation flow diagram – Two stage undertaking. (ortho pics - osteochon leis)

In more widespread generalised arthritis, ultimate treatment is total knee replacement surgery. However, in general terms, the aim would be to try and delay arthroplasty surgery for as long as possible, as the average knee replacement has a survivorship of about 10-15 years and would therefore mean in younger individuals, it would require revising. This requirement to aim to delay arthroplasty surgery has, however, to be weighed-up against an individual's quality of life. For example, a 50 year old patient with an arthritic knee and significant intrusion of their quality of life may warrant consideration of total knee replacement to improve their quality of life if it is accepted that they are likely to require 1 or more revision knee replacements during their life time.

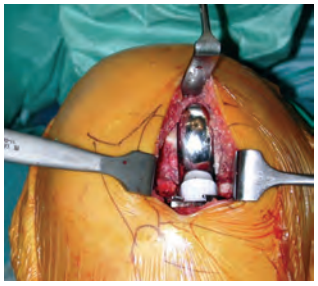


Figure 17: Unicompartmental knee replacement of medial compartment through minimally invasive technique. (ortho pics - uni - case 2b op)

Other options to treat knee arthritis include osteotomies. All of the options such as arthroscopic procedures, cartilage regeneration, osteotomies and even, potentially, unicompartmental replacements, are designed to try and delay the potential requirement of a total knee replacement.

Technology has advanced with total knee replacements surgery, as in other aspects of medicine. The aim of knee replacement surgery is to try to provide a pain-free, functional knee replacement with as long a survivorship as possible, so as to minimise the risk of revision. One of the key factors in delaying revision is having a well aligned knee replacement. Technology can be used to aid with this. Personalised knee replacements are now available and frequently used in my practice.

Personalised knee replacement involves the individual having a preoperative MRI or CT scan in order to 3-dimensionally model their knee anatomy. From this model, computer generated cutting blocks are designed to reproduce anatomical alignments (Figure 19). These blocks are then sent to the surgeon in order to undertake the knee replacement, with the blocks being personalised to the patient's anatomy and corrections required.

Personalised knee replacements also have additional benefits, such as allowing minimal incision surgery, which reduces pain, swelling, and improves recovery rates, thus reducing length of stay. It also does not require penetration of the intramedullary canal of the knee, therefore, reducing fat emboli and its consequence risks to the patient (Figure 20).

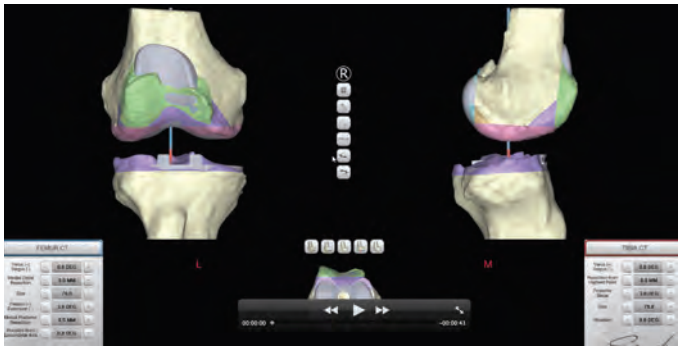


Figure 19: Personalised Knee replacement computerised model generated from MRI or CT scan of patient's knee. The computerised model can be adjusted by the surgeon to meet appropriate criteria. (clinical - signature)

Personalised knee replacements are more expensive than standardised knee replacements and are commonly not funded by the NHS. However, private insurance companies do fund this surgical technique.

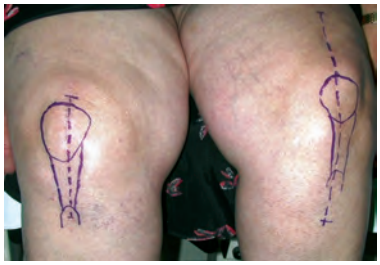


Figure 20: Minimally invasive incision in patient's right TKR performed by author, compared to routine incision in a standard TKR in patient's left knee. The minimally invasive procedure has a number of benefits of which cosmesis is actually the least important, but not for the patients.

Technopathies

Tendinopathies are an overuse injury. Their frequency is increasing as the population is becoming more active and undertaking more sports. Around the knee tendinopathies can occur in the patellar tendon, the quadriceps tendon, the hamstring tendons and the iliotibial band.

The patient's history is usually one of gradual onset of pain in the particular tendon. Initially, it will tend to occur after the individual has played their sport or exercise. As the tendinopathy worsens, it tends to intrude into the individual's sport and so may stop them playing or exercising. As it becomes more severe, the pain can also occur at rest.

Tendinopathic tendons are at risk of rupture.

Tendinopathies tend to occur in individual's who undertake regular impact-loading sport that involves running and jumping.

The typical examination finding is usually no swelling in the knee with an essentially full range of motion. The individual usually, however, has fairly localised point tenderness over the particular involved tendon.

The treatment for tendinopathies is usually non-surgical. Initially, the individual will have to stop their triggering sport. They may be able to continue non-impacting loading exercise such as swimming and riding a bike, as long as it does not cause pain in the tendons. Physiotherapy modalities can be used, including shock wave treatment to try and break down the tendinopathic process within the tendon and increase the healing rate. Certain types of strengthening processes can also be used, including eccentric strengthening.

Further non-interventional treatments are commonly used, which include dry needling, platelet rich plasma (PRP) injection, and autologous blood injection. The aim of these three treatments is to increase blood flow and growth hormones into the tendinopathic tissues so as to increase the healing rates. Ultimately, if the tendonitis does not respond, then the final treatment would be surgical excision of the tendinopathic segment.

Conclusion

The majority of knee problems can be diagnosed with a detailed history and a good examination. Specialised imaging, such as MRI scanning, is also commonly used as an adjunct to diagnosis.

Common knee conditions are discussed in this article, including management options.

For further information on treatment options, please visit **www.thekneedoc.co.uk**. For further information on knee sports injuries, please visit **www.football4football.com/injuries.**

The complexity of Low Back Pain

Introduction

Low back pain is neither a disease nor a diagnostic entity. It refers to pain of variable duration in the lower back anatomical region. It is the commonest MSK problem seen in the primary care. It will affect 80% of adults in industrialised countries at some time during their lifetime. By the third decade, 50% of adults would have experienced back pain which requires alteration in their activity1. It is the second most com-mon cause for physician visits and the third leading cause for surgery and costs approximately £100 million annually. Even with optimal management 5% of patients with back pain progress to chronicity2.

Causes

Only a minority of back pain is caused by specific physical causes. Specific causes of back pain account for less than 20% of cases: the probability that a particular cause of back pain has a specific cause is only 0.2%3.

Non-specific back pain is thus a major problem for diagnosis and treatment. Studies in the United Kingdom identified back pain as the most common cause of disability in young adults4.

Causes

- Vertebral body - Misalignment and fractures
- Intervertebral Disc - Annular tear, disc prolapse and spondylodiscitis
- Nerves - Referred pain, sciatic pain due to nerve root compression or irritation

Beware of visceral causes of back pain. 2% of all back pain has a non-spinal cause e.g Aortic aneurysm, pan-creatitis, peptic ulcer, renal causes and pelvic pathology in women.

Assessment

Thorough history and detailed information of type, character and duration of pain will help define the diag-nosis. Age can determine the most likely cause. In patients older than 50 years degenerative conditions, osteoporosis and malignancy may be the cause of the back pain.

Beware of presence of red flags

- Age <20 and >55
- Thoracic back pain
- Previous history of cancer
- Immunosuppression
- Significant neurological deficit
- Unintentional weight loss
- Systemically unwell patient
- Structural deformity which is of recent onset

If the patient does not have any reflags then it is important to consider presence of Yellow flags as it is important to address this early to prevent chronicity.

Yellow Flags

- Belief that pain and activity are harmful
- Sickness behaviour
- Social withdrawal
- Emotional - depression and low mood
- Time off work
- Unrealistic expectations of treatment

Examination Tips

L4-5 and L5-S1 are the commonest affected discs. A prolapse of the L4-5 disc will usually cause compression of the L5 nerve root and L5-S1 prolapse causes S1 nerve root compromise. This will result in a positive Straight Leg Raise (Fig 1) test - positive SLR is only if the sciatic pain is re-produced on doing the test. A quick screening test to see if there is any motor weakness is to ask the patient to stand and walk on their tip-toes and to walk on their heels. If there is weakness of the ankle plantar flexors then this is due to S1 root com-pression. Inability to stand and walk on the heels results from a weakness of the ankle dorsi-flexors which may represent either L4 or L5 root pathology.

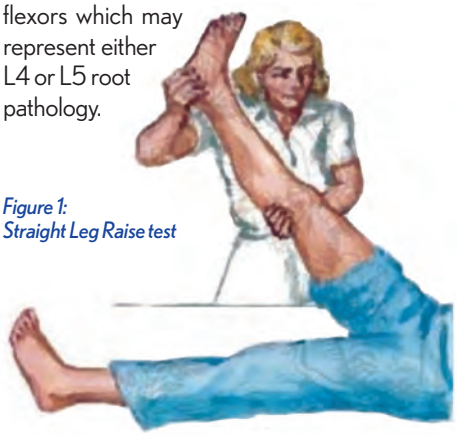


Figure 1: Straight Leg Raise test

Upper lumbar disc prolapse is rare. However when it does occur it causes severe pain and potential significant disability if motor power is affected particularly to the quadriceps. Femoral nerve stretch test (Fig 2) can identify compression of upper lumbar nerve roots.

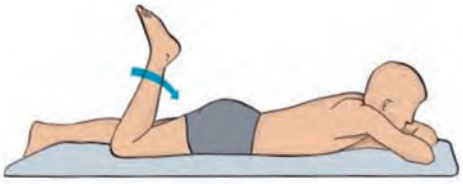


Figure 2: Femoral Nerve Stretch Test

Management

In patients with acute back pain with no red flags signs, the aim of treatment is to prevent it from becoming a chronic problem. This can be facilitated by early identification of those patients at risk of long-term dis-ability. It is important to address psychosocial factors, depressive mood and somatisation to prevent chronicity.

The NICE clinical guideline 88 published in May 20095 lays out principles of management. It is patient centered and encourages clinicians to keep diagnosis under constant review. It encourages self-management of the back pain with simple painkillers and continuing to keep active. Opioid use in patients with back pain should not be encouraged due to the potential addictive nature of the opioid medication.

In a patient who presents with back pain and after ruling out serious pathology, consider offering one of the following modalities-

- Exercise therapy for up to 12 weeks
- A course of manual therapy
- Acupuncture

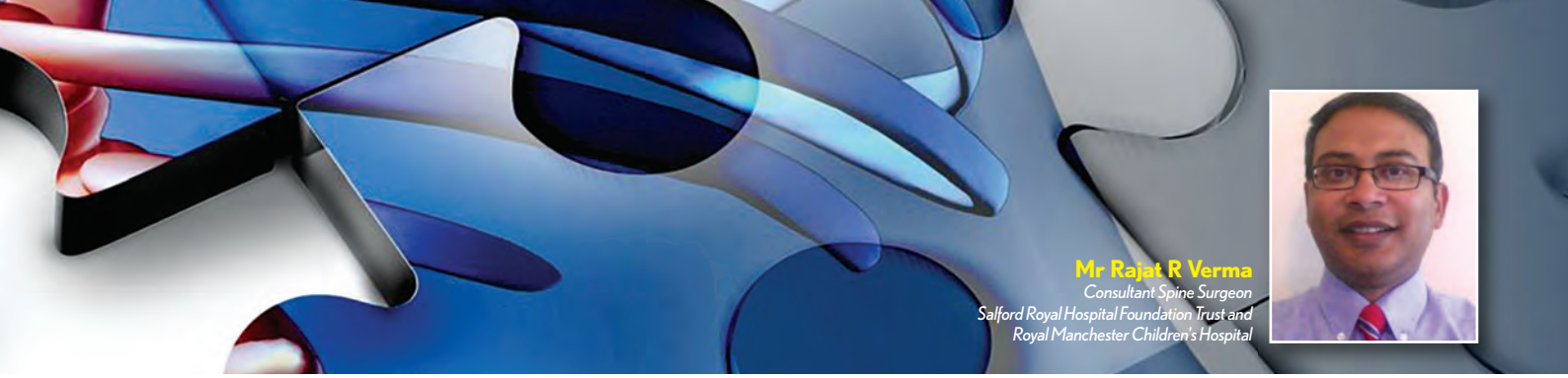
Injection therapy for back pain alone may not give long lasting pain relief. Epidural steroid injections are widely used however, they are not indicated for patients who present with back pain alone. Epidural steroid injections work well in patients who have radicular pain due to nerve root compression or irritation. When carried out it is best to do it under fluoroscopy guidance as studies have shown a 35%-40% miss rate if done blind6. Sacro-iliac joint injection and facet joint injections are done in patients who are clinically di-agnosed as having pain originating from these anatomical structures. These injections on their own are very unlikely to be curative and need to be followed up by a course of physical therapy. There is weak evidence for short term relief with intra-articular steroid injections.

Consider referral for combined physical and psychological treatment in somebody who has received at least one less intensive treatment and has a high disability and/or psychological distress.

Surgery for low back pain: Less than 10% of patients with non-specific back pain undergo surgery. There is a fundamental lack of evidence for imaging to identify the source of back pain. Robust evidence for surgery over best non-operative care is lacking.

Case studies of Specific causes of back pain

1. Prolapsed Intervertebral Disc (Fig 3A & B)



Mr Rajat R Verma
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It commonly involves L4-5 and L5-S1 disc, resulting in compression of L5 or S1 nerve root. L4 root can be involved if a foraminal disc.

Refer patients for imaging and/or specialist opinion if they have:

- Unresolved root pain of more than 6 weeks duration and have not resumed normal activity in 3 months
- Cauda equina compression features - bilateral sciatica, saddle anaesthesia or sensory disturbance in the perineal region, abnormal bladder/bowel function
- Progressive neurological deficit e.g foot drop.

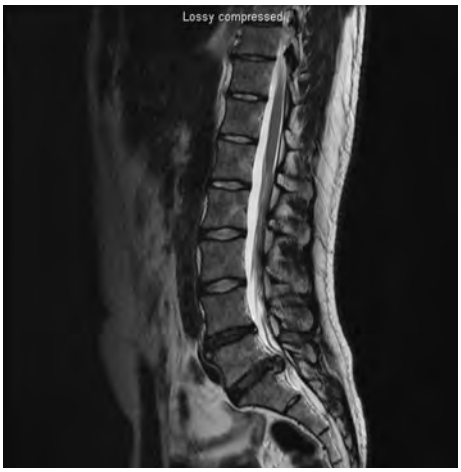


Figure 3A

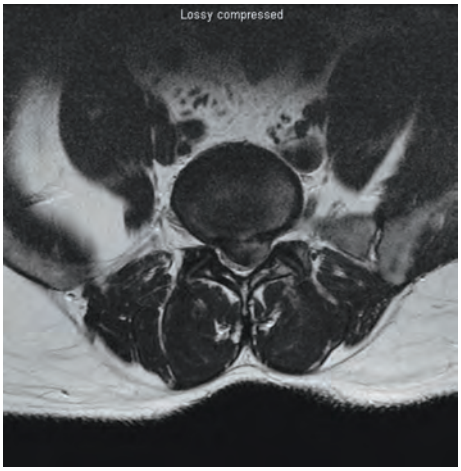


Figure 3B

Surgery for a prolapsed intervertebral disc is indicated only to relieve radicular pain and is not indicated for back pain alone. Microdiscectomy has a 75%-80% success in curing leg pain. However there is a 5%-10% chance of recurrence after a primary lumbar miscrodiscectomy. The risk of paralysis is low and probably in the region of 1 in 300. Most patients will be fit to be discharged after an overnight stay in the hospital. Re-turn to office based work would be in the region of 4-6 weeks.

2. Degenerative lumbar canal stenosis (Fig 4A and Fig4B)

This is a common condition and results in symptoms of neurogenic claudication. This is neuropathic symp-toms in the legs brought on by walking. Natural history studies have established that it is not inevitable that deterioration occurs in all patients. As a rule of thumb a third of patients get worse, a third remain the same and the rest may improve spontaneously over time. Treatment is usually sought to improve quality of life.

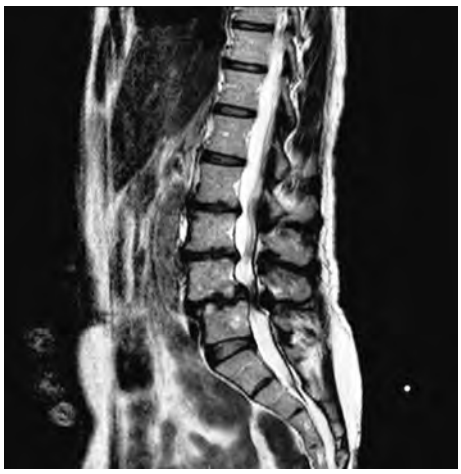


Figure 4A

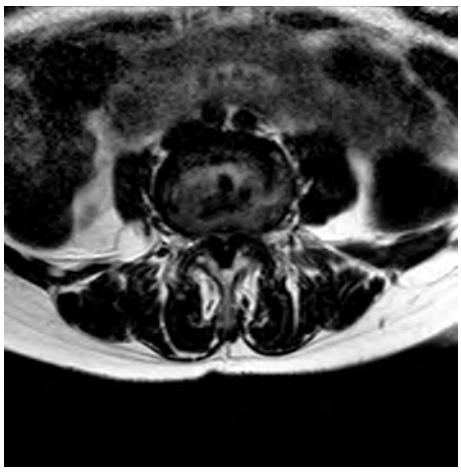


Figure 4B

Patients should be referred for surgical opinion if:

- They have failed conservative therapy
- Patient is willing to consider surgery
- Patient has chronic cauda equina compression type symptoms
- Neurological deficit
- They feel quality of life is poor due to limited mobility.

3. Spondylolytic spondylolysthesis (Figure 5A and 5B)

In adolescent patients with back pain this is a cause to look out for, particularly in athletes, ballet dancers, fast bowlers etc. it commonly affects the L5 level resulting in a defect of the pars interarticularis. Forward slippage of one vertebra over the other is called spondylolysthesis and may occasionally result in nerve root compression resulting in sciatica.

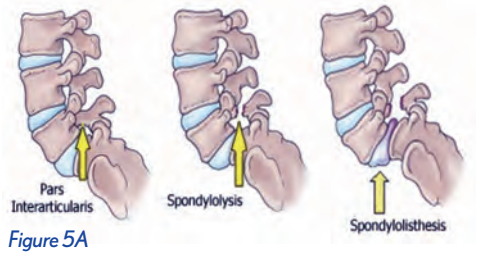


Figure 5A

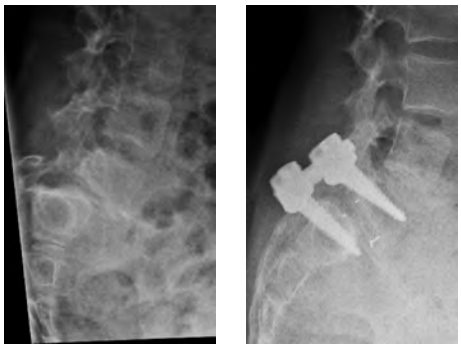


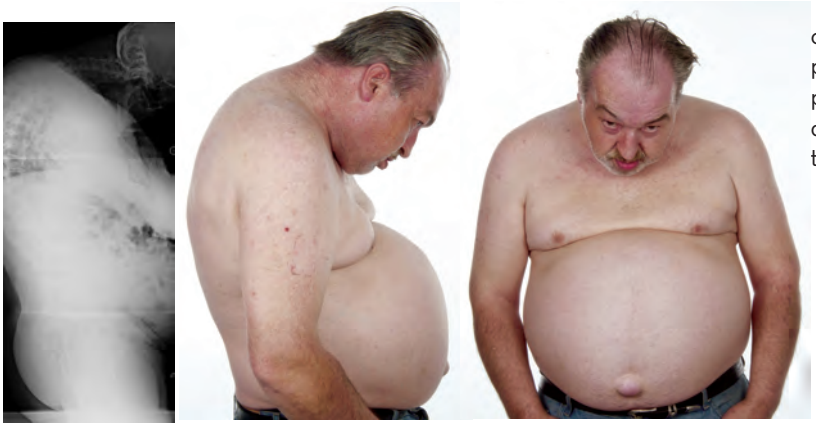
Figure 5B

Initial treatment is in the form of activity modification and avoiding actions that exacerbate pain. Surgery is required in a small minority of patients where either there is a progression of the slip or there is failure of conservative treatment.

4. Ankylosing Spondylitis (Figure 6 A,B and C)

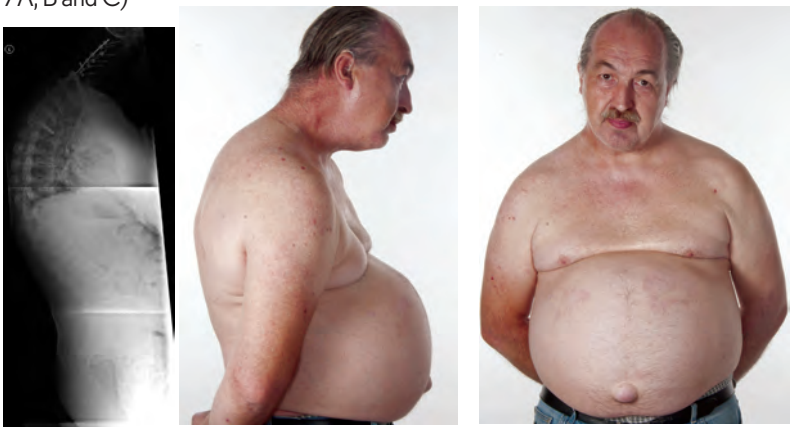
This is a chronic inflammatory spondyloarthropathy which occurs 3 times more commonly in males. There is an association with HLA-B27 but not always positive. There is no known cure and modern treatment is directed at reducing the effects of the disease. It presents in the 15-25 year age group. Uveitis must be rec-ognised and treated promptly. Back pain is the main symptom. Majority of patients remain fully independent. Due to ankylosis of the spine there is a significant risk of fractures even with relatively minor falls. Beware of fracture at 2 non-contiguous levels and frequently ankylosing spondylitis patients will have fracture of 2 areas of the spine and both may require operative intervention.

The complexity of Low Back Pain *(continued)*



Figures 6A (left), 6B (centre) and 6C (right)

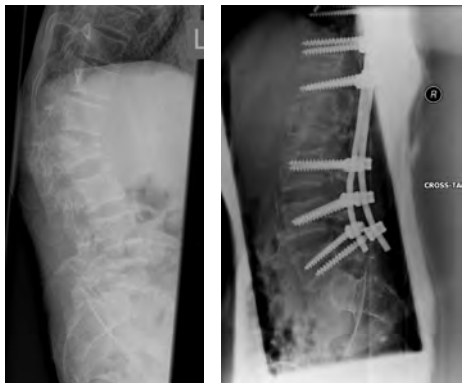
Kyphosis deformity can result in loss of horizontal gaze and some patients need a spinal realignment os-teotomy to correct the horizontal gaze (Figs 7A, B and C)



Figures 7A (left), 7B (centre) and 7C (right)

5. Infective spondylodiscitis (Figure 8A & B)

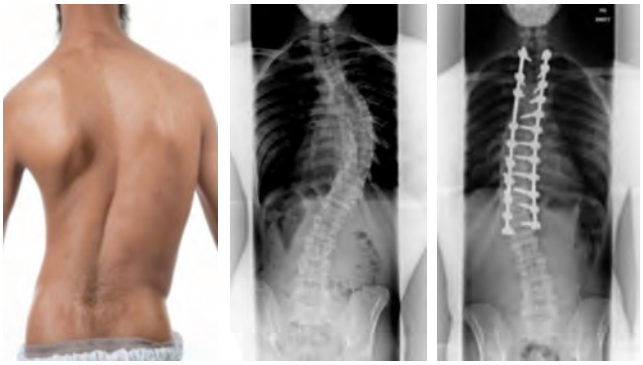
This is usually caused by haematogenous spread of bacteria. It is commoner in adults of more than 50 years of age. Presenting symptom is usually back pain refractory to conservative measures. Patients may feel sys-temically unwell and the inflammatory markers like CRP may be raised. If not treated promptly it may lead to bony destruction and osteomyelitis resulting in deformity of the spine or rarely neurological deficit. Majority resolve with 6-12 weeks of antibiotic therapy.



Figures 8A (left) and 8B (right)

6. Adolescent idiopathic scoliosis (Figure 9a, b and c)

This is commoner in girls and the exact cause is unknown. Positive family history is present in approximately 30% of patients. Although back pain may be present in some patients it is usually not caused by the scoliosis. AIS on its own is a painless condition and does not lead to 'pressure on internal organs' or short-ness of breath etc. Risk of curve progression is highest in patients who present with a curve greater than 30 degrees and are pre-menarchal at presentation. This is due to the fact the curves progress during the pe-riod of maximum growth velocity which corresponds to the 12 months before and after attaining men-arche.



Figures 9A (left), 9B (centre) and 9C (right)

7. Spine metastasis

Spine is the commonest location of skeletal metastasis due to the large volume of bone mass. Skeletal spine metastasis can be seen in up to 40% of patients with cancer. Common primaries that metastasise to the spine are breast, lung, prostate, thyroid and renal tumours. Sixty percent will be located in the thoracic spine. Progressive and unrelenting pain is the commonest symptom and this is due to infiltration of the bone by the tumour or by an acute pathological fracture. If left untreated all patients with a spinal metastasis will end up developing neurological compromise.

Twenty percent of patients with metastatic spinal cord compression do not have a diagnosis of primary cancer and their first presentation is either severe back pain or neurological deficit secondary to neural compression.

The NICE clinical guidance on metastatic spinal cord compression CG75 issued in November 2008 covers management of patients who develop MSCC because of spread of cancer from elsewhere in the body. Patients with MSCC who have a life expectancy of more than 3 months may benefit from surgical stabilisation and oncology treatment (Figures 8A and 8B).

Conclusion

Majority of acute back pain resolves spontaneously in 6-8 weeks with simple measures. Encourage self management however keep diagnosis under constant review to avoid missing serious pathology. Surgical intervention for non-specific back pain is rarely rewarding in the long term, however specific causes can be amenable to timely and well thought out surgery.

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A lady presenting with Chest Pain – case report

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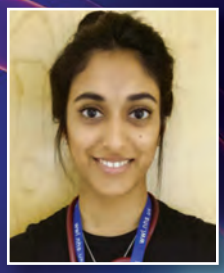


Case History

A 77 year old female with known hypertension presented with sudden onset chest pain. There was radiation to the back and left arm with associated nausea and dizziness. No relief was found with GTN spray given in the A&E, however, the pain resolved with analgesia approximately one hour later. Examination was relatively unremarkable, with the exception of the patient looking pale and feeling clammy. Observations showed a blood pressure of 100/60mmHg and a regular heart rate of 58bpm. Initial investigations were all negative, including no acute changes on chest x-ray and ECG, with negative troponin levels. At this stage impression was of musculoskeletal pain. Patient was kept in for observation at the insistence of her daughter.

Over 24 hours observations found an increase in blood pressure results and repeat bilateral blood pressures found a significant discrepancy with left arm measuring 210/100mmHg and right arm 180/90mmHg. An urgent CT was arranged which showed an extensive aortic dissection, originating from the lateral aspect of the aortic arch immediately distal to the left subclavian artery and extending to the left iliac artery (Figure 1). CT showed a decompensated left kidney. During her stay she developed a mild degree of renal impairment, however overall she recovered well and was discharged after a few days with good control of her hypertension. The decision was for conservative antihypertensive management and regular follow up to ensure a blood pressure below 130/80mmHg is maintained and at 10 year follow up this was the case.

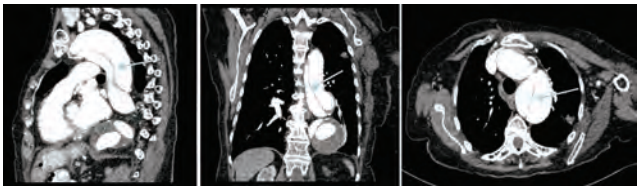


Figure 1 – blue arrow shows the dissection flap separating true and false lumen of Stanford type B aortic dissection. Aneurysmal dilatation is also seen.

Aortic Dissection

Aortic dissection refers to a rupture of the intimal layer of the aortic. The result is a vessel with a true and false lumen as demonstrated in figure 1. In the UK and USA it is estimated that 3-4 people per 100,000 are diagnosed with aortic dissection per year (1). The consequences, depending on location and extent of dissection, can be managed either with medical or surgical intervention or may result in organ failure/deficiency or death.

Classifications

The two most prominently used classification systems are The Stanford Classification and the De Bakey system (Table 1). These systems are related to anatomy which is crucial when considering management and treatment options available. Acute dissection refers to those occurring within two weeks of symptom onset, as this is the time with the highest rates of morbidity and mortality (2).

Stanford Type A dissections have a high rate of mortality and in the acute stage is a surgical emergency (3). They are, however, more common, occurring in about two thirds of cases (4). Stanford Type B dissections have better prognosis. If uncomplicated the treatment choice is medical management only, however, in complicated cases surgery may be indicated.

Classifications	Site Involved
Stanford Type A	Involving the ascending aorta
Stanford Type B	Not involving the ascending aorta
De Bakey Type I	Originating in the ascending aorta and extending to at least the aortic arch
De Bakey Type II	Originating in the ascending aorta and confined
De Bakey Type III	Originating in the descending aorta

Table 1: Classification of Aortic Dissection

Aetiology

Any mechanism causing weakening of the aortic wall can be considered as a risk factor in the predisposition to aortic dissection. The range stems from congenital genetic disorders such as Marfan's disease, or acquired disease such as HTN and drug use. The most common risk factor documented in the IRAD registry is hypertension, found in 72% of patients (2).

Presentation

Initial presentation may be broad and non-specific with a list of differential diagnoses ranging from acute coronary syndrome to cardiovascular event. The most common symptom in the acute stage is pain - classically described as "tearing" and "sharp" and usually severe with sudden onset (2). Migratory pain has been described as the dissection progresses and the absence of pain is reported in as high as 15% of cases (5).

5 to 10% of cases present with syncope, in some circumstances found to be the only presenting complaint (2,6). It more commonly occurs due to carotid artery involvement causing reduced cerebral blood flow or rupture and the resulting hypovolaemia. Less common causes are direct or indirect (e.g response to pain) vasovagal responses (6).

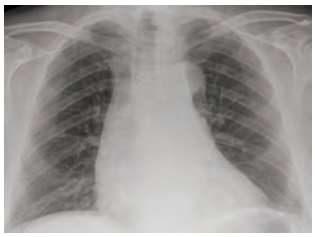
Examination findings vary depending on location of dissection. Blood pressure can differ with hypertension commonly found in distal dissections and hypotension in proximal. The classic presentation of pulse deficit and aortic regurgitation murmur is more commonly found in Stanford Type A dissections (4). Neurological deficit can be attributed to hypotension or occlusion of supplying vessels, and have been found in one third of patients without pain (5).

Investigations

As mentioned, differential diagnoses include acute coronary syndrome and cardiovascular events. Treatment of these diseases often involves antiplatelet therapy which would be lethal in the event of an aortic dissection, therefore rapid, accurate diagnosis important.

ECG findings are often normal in aortic dissection. Evidence of acute MI can be seen if the dissection progresses into a coronary artery - treatment with antiplatelet therapy will often result in death (7). CXR is abnormal in 60-90% of patients (7). It can show a widening mediastinum, aorta with abnormal cardiac outline and pleural effusion, however, in unstable patients a chest x-ray will delay treatment and should therefore be omitted.

Once aortic dissection is suspected, prompt diagnosis is essential. Imaging methods available include echocardiogram (TTE plus TOE), contrast CT, aortography and MRI. All methods have high levels of accuracy in diagnosis, however, in the initial stages consideration must be given to availability, technical experience and speed of scan (4). IRAD study found that CT was most commonly used (61%), followed by echocardiogram (33%), angiography (4%) and MRI (2%)(2). Further imaging following the acute stage is often required to assess associated complications and to further define anatomy (4).



Chest X-ray showing mediastinal widening

Management

Initial management involves analgesia, antihypertensive therapy and maintaining a haemodynamically stable patient. Systolic pressure should be maintained low enough for sufficient perfusion, between 110-120mmHg. IV beta-blockers such as labetalol and esmolol are recommended as they reduce blood pressure, are short acting and reduce the force of LV contraction (therefore, reducing aortic wall stress) (7).

Definitive management is dependent upon type and complications relating to dissection. With Stanford type A dissections, surgical management is considered the preferable option. Medical management alone has a mortality rate of 20% within the first 24 hours and increases to 50% within 1 month (3). Surgical repair, however, also has a high mortality rate of 20% in the first 30 days (3). Methods of surgical repair involve resection of intimal tear with graft replacement or partial/total arch replacement with aortic valve surgery if necessary (8,9). Consideration must be given to size of the aortic root and state of the aortic valve when deciding on surgical repair method (3). More recent developments have included percutaneous stenting and balloon fenestration (8).

Stanford Type B dissections are better managed with medical treatment in uncomplicated cases. Hypertensive therapy, maintaining systolic pressure between 100-120mmHg, with regular follow up can result in a 78% three year survival rate (1). In patients where this not possible or if there is evidence of ischaemia, rupture or refractory pain, surgery may be necessary.

Chronic Type B dissections often develop complications such as aneurysms, suggested to occur in 15% (2). Regular follow up and imaging is recommended with the European Society of Cardiology recommending appointments at 1, 3, 6 and 12 months with annual follow up thereafter (8).

Summary

Aortic dissection is considered a medical emergency, often with poor outcome, however, prognosis varies. Type of dissection, as well as speed of diagnosis and management, are variables that can alter the survival rates. As in the case of our patient uncomplicated type B dissections can be managed well with hypertensive therapy.

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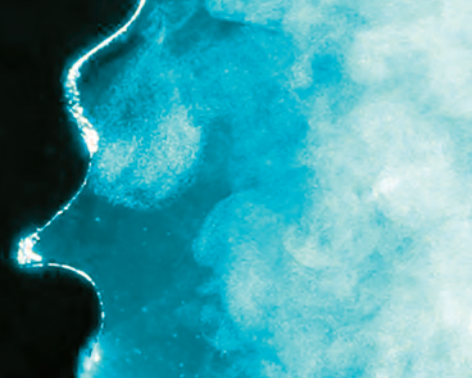
A case of Breathlessness and Chest Pain



Case History

A 60 year old lady presented to the A&E with three weeks history of breathlessness, non- productive cough and chest pain. She had a normal coronary angiogram and a normal echo previously. She has history of hypertension, Hypercholesterolemia, hypothyroidism. She is an ex-smoker. She drank alcohol 30 units per week. Her blood results showed normal U&E, abnormal LFTs, mild anaemia and no features of infection. She was found to be in AF with rapid ventricular rate but spontaneously reverted into sinus rhythm 24 hours later. She subsequently complained of abdominal pain. CXR revealed cardiomegaly and surgical cause was excluded. She became tachycardiac with hypotension. Bedside echo-cardiogram revealed large pericardial effusion, collapse of right atrium and right ventricle. Urgent pericardiocentasis with insertion of pericardial drain was performed. Pericardial fluid analysis suggested exudative effusion. But pericardial fluid cytology examination showed metastatic adenocarcinoma. CT scan of chest revealed hilar lymphadenopathy, left lower lobe (likely) adenocarcinoma, right sided pulmonary nodule and acute portal venous thrombosis. She was referred to oncology for further treatment. A final diagnosis of Malignant pericardial effusion presenting with cardiac tamponade, adenocarcinoma of lungs with metastases and acute portal vein thrombosis was made.

Pericardial Disease

Pericardium surrounds most of the heart. It has two layers - visceral and parietal. The pericardial space in between these two layers contains serous fluid up to 50 ml. Usual abnormalities and diseases of pericardium include the following :-

1. **Absence of pericardium—congenital or after surgical resection**
2. **Pericardial cysts**
3. **Pericarditis and Pericardial effusion**

The absence of pericardium or pericardial cysts does not cause major problem. Pericarditis and pericardial effusion can result in mild to life threatening illnesses. These can be:

Acute pericarditis, chronic constrictive pericarditis, Cardiac Tamponade, Relapsing or recurrent pericarditis.

1. Pericarditis and Pericardial effusion

Pericarditis is inflammation of the pericardium usually resulting in increased fluid accumulation in the pericardial space (pericardial effusion).

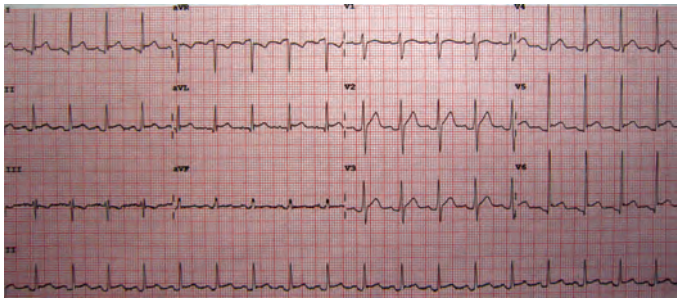
CAUSES OF Pericardial effusion / Pericarditis

1. **Idiopathic** — common
2. **Infective** — Bacterial - pneumococcus, mycoplasma, mycobacteria/Viral –echovirus, adenovirus, coxsackievirus, HIV virus /Fungal –histoplasma, coccidioidmycosis/ Protozoal
3. **Malignancy** — a. Primary –mesotheliomas, fibro sarcoma b. Secondary-cancers from lungs, breasts, lymphomas
4. **Inflammatory** — systemic lupus erythematous, systemic sclerosis, rheumatoid arthritis, inflammatory bowel disease, Arteritis (polyareteritis Nodosa, giant cell arteritis
5. **Drugs** — procainamide, hydralazine, methyldopa, isoniazid, cyclosporine
6. **Post** — Radiation
7. **Post cardiac surgery/post cardiac procedures** — CABG, valve operations, pacemakers, ablation
8. **Metabolic** — uraemia, Hypothyroidism, hyperthyroidism
9. **Trauma**
10. **Post myocardial infarction** early and late (Dressler syndrome)
11. **Miscellaneous** — dissection of aorta, amyloidosis, cholesterol pericarditis (gold paint pericarditis)

Symptoms and signs include dyspnoea, cough, fever, chest pain (pleuritic pain which gets better on leaning forward), raised JVP. Presence of Pulsus paradoxus (10-mm Hg or more drops in systolic pressure during inspiration) raises suspicion of cardiac Tamponade. Pericardial friction rub may be heard (classically tri-phasic).Heart sounds are muffled in large effusions or in tamponade.

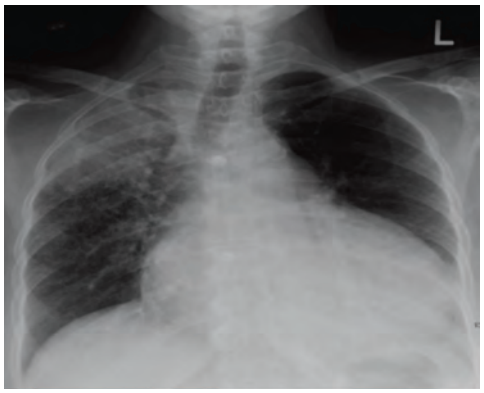
ECG changes in Pericarditis

Stage I	ST-segment elevation (usually in all leads except in aVR) with shape of ST concave upwards and PR-segment depression (diffuse)
Stage II	Normalization of the ST and PR segments
Stage III	T-wave inversions (diffuse)
Stage IV	Normalization of the T wave.
ECG in Pericardial effusion	Low voltage complexes in all leads, Electrical alternans (in Tamponade)



ECG showing diffuse ST elevations in acute pericarditis

CXR—may be normal; may show features of underlying disease e.g. pneumonia, tuberculosis, enlarged hilar lymph nodes (malignancy), pleural effusion, increased cardiothoracic ratio due to pericardial effusion (However small effusions usually do not cause this feature)



Chest X-Ray (CXR) showing globular heart suggestive of pericardial effusion

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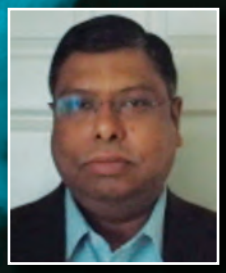


Bloods: General – raised ESR, CRP, WBC, raised troponin and Creatinine Kinase – (associated myocarditis): abnormal results due to underlying disease e.g. raised anti-nuclear factor in SLE.

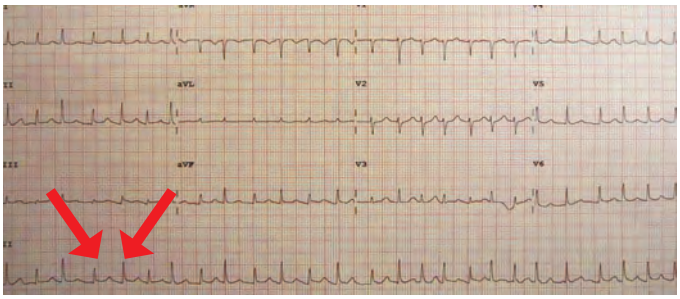
Echocardiography – in acute pericarditis may be normal or it can show presence of small, moderate or large effusion. It can show features of myocardial infarction or myocarditis.

Treatment – acute pericarditis is usually self-limiting without complications or recurrences. Initial treatment is with NSAIDs. Non responders or slow responders can be treated with colchicine for 14 days. Use of corticosteroids should be reserved for exceptional cases and may increase in incidences of recurrence. The following are warning signs which may indicate a complicated course :-

Fever >38°C (bacterial aetiology), patients who are on immuno-suppressive, bleeding into pericardium (e.g. traumatic, those who are receiving anticoagulants), tuberculous pericarditis, large effusions (>2cm), and effusions accumulating rapidly.

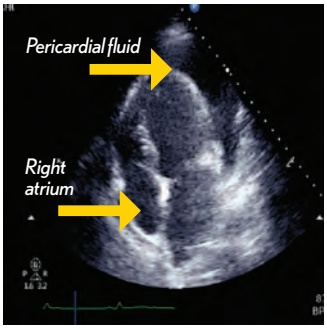
2. *Cardiac Tamponade*

As effusion begins to accumulate it increases the pressure in the pericardial space which has a very small reserve volume. As soon as this volume is reached, it rapidly increases the pressure on the surface of heart which is transmitted to the cardiac chambers restraining cardiac filling. It results in low cardiac output, compensatory tachycardia and increased adrenaline level which augment contractility. It causes haemodynamic instability, prominent X descent with absent or reduced Y descent in JVP, along with Pulsus paradoxus

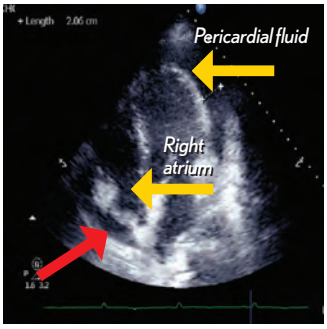


ECG –electrical alternans in cardiac tamponade

Echocardiography– shows large pericardial effusion >2cm, right atrial collapse in diastole (see below), right ventricular collapse lasting more than one third of diastole, distended inferior vena cava with reduced inspiratory collapse, increased variation in mitral and tricuspid flow velocities (reciprocal).



Picture 1 – pericardial effusion (normal right atrium)



Picture 2 – collapsing wall of right atrium (red arrow) (Tamponade)

Treatment -This is a medical emergency and requires pericardial fluid drainage urgently. Fluid can be given to those who are hypovolemic. But otherwise medical treatment is ineffective. Treatment of the underlying aetiology should be sought.

3. *Pericardial constriction*

longstanding pericardial inflammation results in scarring, fibrosis and calcification of pericardium.

Common causes are – idiopathic, post radiation, tuberculous, post–cardiac surgery pericarditis.

Clinical features: It commonly present with features of right heart failure, prominent X and Y descent, inspiratory rise in JVP (Kussmaul's sign) and diastolic Knock due to sudden cessation of rapid ventricular filling may be present. Rarely can it cause anasarca and cardiac cirrhosis. Low cardiac output may cause muscle fatigue, muscle wasting, and cachexia.

Pathophysiology: Because of pericardial fibrosis the pericardium restricts the cardiac filing in diastole. The hallmark is equalisation of pressures in all 4 chambers in diastole. There is abnormal ventricular filling, low cardiac output, systemic and pulmonary venous congestion resulting in hepatic congestion and peripheral oedema.

Investigation: CXR – can show pericardial calcification. Further imaging studies are performed to rule out restrictive cardiomyopathy. **Echo-cardiography** may show thickening of pericardium (>2mm), exaggerated inspiratory variation in mitral inflow velocity (25% or more). **CT** or **MRI** scan shows thickening and calcification of pericardium. **Cardiac catheterisation** reveals early diastolic dip followed by plateau in both ventricular pressure tracings (square root sign) curve.

Treatment: Medical treatment with diuretics and salt restriction can be tried. As sinus tachycardia in these patients can occur as compensatory mechanism beta blockers or calcium channel blockers should be avoided and in case of AF digoxin should be used initially to control ventricular rate. Surgical treatment is pericardiectomy.

4. *Others:* **Transient constriction** can happen after cardiac surgery with spontaneous improvement after few months. These patients need careful monitoring for few months to watch for deterioration. **Effusive – constrictive pericarditis** may be present in a group of patients who present initially with tamponade but the hemodynamic features are not normalised even after pericardiocentasis. Echocardiographic findings are a mixture of both effusion and constriction. Often they require pericardiectomy. **Relapsing pericarditis** is painful and debilitating disease. NSAIDs and prophylactic colchicine can be tried. While no published data exists steroids and immunosuppressives are used by some. Rarely pericardiectomy may be needed.

In conclusion, pericardial disease encompasses a variety of illnesses varying from asymptomatic effusions to life threatening tamponade and may develop pericardial constriction requiring surgery.

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A Practical Approach to a patient with Pleural Effusion

Epidemiology and Pathogenesis

Pleural effusions are common with around 1.5 million cases in the United States each year. In health, the pleural cavity contains a very small amount of pleural fluid which lubricates the surfaces and reduces friction between the parietal and visceral pleura allowing smooth expansion and contraction of the lungs during inspiration and expiration. Pleural fluid accumulates and forms an effusion when there is either increased production of fluid or reduced reabsorption of fluid or both processes happening simultaneously. (1)

Classification of Pleural Effusion

The classical way of classifying pleural effusions is into transudates and exudates. Transudates are due to increased hydrostatic pressure, reduced oncotic pressure, movement of fluid across the diaphragm or more negative intrapleural pressure. Exudates are caused by increased capillary permeability, and reduced lymphatic drainage. (1) As there are as many as 50 different causes of pleural effusion it is important to differentiate them into transudates and exudates as this narrows the differential diagnoses and directs further investigations. Traditionally an exudate contains >30g/L of protein in contrast to a transudate which is said to contain <30g/L. However this is not reliable when serum protein is outside the normal range and causes difficulty when the pleural fluid is very close to containing 30g/L of protein.

British Thoracic Society recommends applying Light's criteria (see Box 1). The one condition where Light's criteria are known to be consistently inaccurate is in congestive cardiac failure (CCF) treated with diuretics. This is because the diuretics increase the amount of protein and lactate dehydrogenase (LDH) in pleural fluid hence causing misclassification of the effusion as an exudate when in truth it is a transudate. A useful method of compensating for this is by measuring serum N-terminal pro-brain natriuretic peptide (cleaved from brain natriuretic peptide which is released by the ventricles on excessive stretch) which is raised in CCF thus helping to clarify the diagnosis.(2)

Pleural fluid is an exudate if one or more of the following criteria are met:
Pleural fluid protein divided by serum protein is >0.5
Pleural fluid LDH divided by serum LDH is >0.6
Pleural fluid LDH is >2/3 the upper limits of laboratory normal value for serum LDH

Box 1: Light's Criteria 2

Causes of Pleural Effusion

Transudates have fewer potential causes than exudates and are more likely to be bilateral. The commonest transudate cause by far is heart failure (80%) followed by liver cirrhosis (13%). Rarer causes include atalectasis, nephrotic syndrome, peritoneal dialysis, hypo-albuminaemia, superior vena cava obstruction and urinothorax.(1)

Exudates have a much broader differential but commonly are due to malignancy, pneumonia, or tuberculosis. There are many other less common causes, for example, pulmonary embolism, pancreatitis and rheumatoid arthritis as well as some drugs.(2) Around 15% of patients diagnosed with cancer will develop a pleural effusion (3) and around 50% of patients with metastatic cancer will develop a pleural effusion (4). The commonest cause of malignant pleural effusion in men is lung cancer and in women, breast cancer though many other cancers can be responsible including: lymphomas, ovarian cancer, gastric cancer and mesothelioma. (5)

Transudate	Exudate
Left ventricular failure	*Infection (empyema / parapneumonic effusion / TB)*
Renal failure	*Malignancy (primary and secondary)*
Liver failure	*Inflammation (vasculitis, autoimmune disease)*
Low protein states	*Pulmonary embolus* *Oesophageal perforation* *Hypothyroidism* *Chylothorax / Pseudochylothorax* *Post-cardiac surgery* *Drugs*

Table 1: Potential causes of pleural effusions 6

Diagnosis and Investigation of Pleural Effusion

History and Examination

A detailed history and examination is essential as they will often reveal possible causes and can guide choice of further investigations. The collection of fluid constricts the underlying lung causing dyspnoea (3) thus patients usually complain of shortness of breath and sometimes cough. However small effusions can be asymptomatic. Pain associated with a pleural effusion can be sharp, suggestive of pneumonia or pulmonary embolism, or constant aching which is more suggestive of malignancy. (1) On examination dullness to percussion is the most reliable sign of pleural effusion though there may also be reduced or absent breath sounds, reduced tactile fremitus, asymmetrical expansion and possibly a friction rub. (7)

Chest X-ray

If a pleural effusion is suspected the, initial investigation is a plain posteroanterior chest x-ray, which can indicate an effusion of >200mls, though a lateral chest x-ray can show an abnormality with a collection of just 50mls of fluid. (2)

If there are bilateral symmetrical pleural effusions with a clinical scenario highly indicative of a transudate no more investigations need to be done, instead the cause of the transudate should be treated. However often in CCF the effusions may be unilateral or asymmetric but for these further investigation should be carried out. (6)

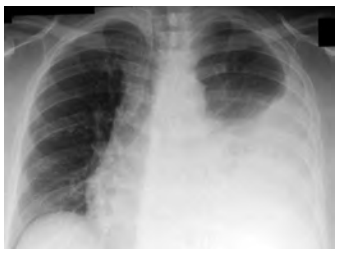
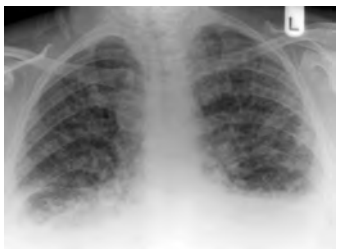


Image1: Plain CXR of left pleural effusion



Plain CXR of bilateral pleural effusion

Ultrasound and Thoracentesis

The next line of investigation is thoracic ultrasound which is more sensitive at detecting fluid and is also able to differentiate between fluid, consolidation, pleural thickening and potentially indicate a malignant cause.(1) Ultrasound is able to safely guide thoracentesis with reduced incidence of iatrogenic pneumothorax.(2) Analysis of the collected pleural fluid should be carried out firstly to differentiate a transudate from an exudate by using Light's criteria but also further tests can be done to provide clues as to the cause.

Computerised Tomography

Computerised tomography (CT) is useful in distinguishing benign from malignant pleural effusions, and also in diagnosing empyema and differentiating it from a lung abscess. Therefore CT should be carried out in all patients with an exudative pleural effusion of unknown cause or patients with a complicated pleural infection. (2)

Invasive Investigations

Invasive investigations may be required if the investigations discussed above have not produced a diagnosis. This usually involves obtaining a biopsy of the pleura and this can be image-guided (usually CT) or during thoracoscopy. The advantage of thoracoscopy is that it allows the pleural cavity to be examined as well as taking biopsies. Consequently thoracoscopy facilitates identification of patients at risk of trapped lung, where fibrosis or a multi-loculated effusion prevents the full re-expansion of the lung once the effusion has been drained. Thoracoscopy also enables treatments such as chest drain insertion or pleurodesis. (8) Furthermore as thoracoscopy allows visualisation of the pleural cavity it means biopsies can

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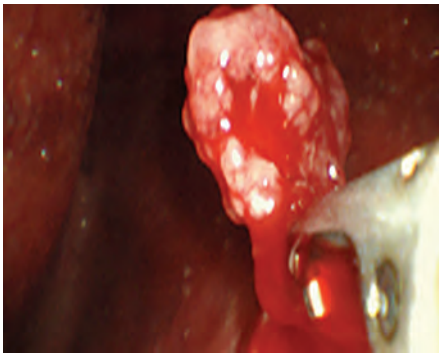


Image 2: Suspicious lesion during thoracoscopy, being biopsied

be taken from visually abnormal areas thus maximising the diagnostic yield giving a 92.6% diagnostic sensitivity for malignant pleural disease. Its diagnostic yield is as high as video assisted thoracopic surgery (VATS). (9) VATS is a much more invasive investigation requiring GA and single lung ventilation, to obtain biopsies but allows more extensive surgical intervention. (10)

Treatment of Pleural Effusion

Transudates

In general the treatment for transudates is to treat the cause. As discussed previously the two commonest causes are CCF and liver cirrhosis. For both these the treatment is diuretics though in patients with large effusions refractory to diuretics a chest drain may be needed.1 In a few cases indwelling pleural catheters have been used for refractory transudative effusions but there is limited evidence to support their use and there is an increased risk of empyema. (11)

Exudates

1. Infection

A complication of pneumonia is a parapneumonic effusion which may resolve with antibiotic therapy, however the risk is it will progress to empyema, virtually all of which require chest tube drainage. Thus the challenge is to decide which parapneumonic effusions require drainage as delay can increase hospital stay, morbidity and mortality.(1)

2. Malignant Pleural Effusion

A malignant pleural effusion suggests advanced disease and these patients have a reduced life expectancy. Survival is dependent on the underlying cancer but generally ranges from three to twelve months. Treatment depends on symptoms, patient performance status and primary site of the cancer. (5) If systemic treatment of the malignancy is possible this may help the effusion. If it is not, palliative control of the effusion is required. (12)

Observation and monitoring may be all that is needed if the effusion is an incidental finding, the patient is asymptomatic, the malignancy is already known, and is being actively treated. These patients are likely to become symptomatic at which point further treatment can be initiated. (5)

Initial treatment is usually therapeutic thoracentesis to improve symptoms although in nearly all cases the effusion will reaccumulate. Repeated thoracentesis should be avoided as it can create adhesions and loculations which will make further treatment of the effusion more complex. This should therefore be restricted to frail patients who have a very limited life expectancy. (13) No more than 1.5 litres should be drained on a single occasion as this can precipitate pulmonary oedema due to rapid re-expansion of the collapsed lung. (5)

Pleurodesis

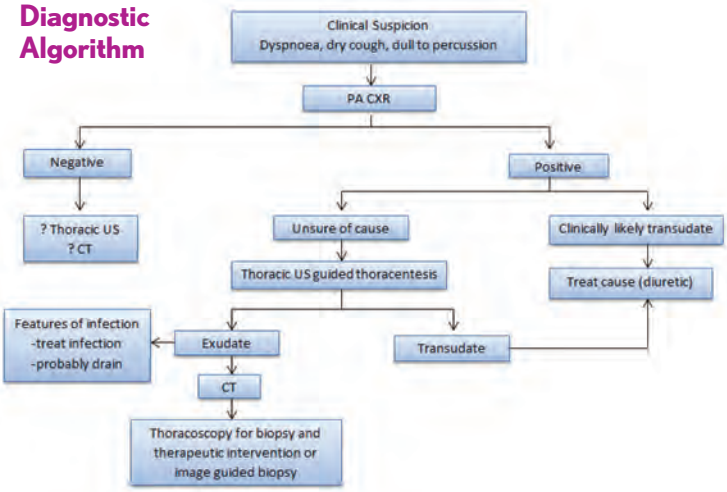
Prevention of recurrence is an important consideration and so pleurodesis should be attempted in patients without trapped lung. Pleurodesis aims to obliterate the pleural space by irritating the pleura thereby causing fibrosis and 'sticking' the visceral and parietal pleura together. This can be done by either chest tube insertion, drainage of the effusion and instillation of a sclerosant, or during thoracoscopy. A recent Cochrane review concluded that pleurodesis is more likely if a sclerosant is used and that talc (an inert trilayered magnesium silicate sheet) is the most effective sclerosant. It also established that pleurodesis attempted during thoracoscopy (talc poudrage) was more likely to be successful than if done at the bedside through a chest drain (talc slurry) because thoracoscopy is better able to fully drain the effusion and equally distribute the sclerosant. However bedside pleurodesis may be preferable in patients with a poor performance status where thoracoscopy and sedation may be difficult. (4)

Pleurodesis may not be possible or successful in some patients where there is incomplete lung expansion due to trapped lung or many loculations. (14) In this situation, an indwelling pleural catheter (IPC) should be considered, which can be drained periodically in the comfort of the patient's home. IPCs are effective in managing recurrent, symptomatic malignant pleural effusions and through repeated drainage spontaneous pleurodesis may often be achieved. (11) Moreover placement of an IPC may be associated with a shorter hospital stay than pleurodesis so could be a preferred management option in patients with short life expectancies, although it must be remembered that there needs to be community expertise to be able to drain the IPC with vacuum bottles.

Conclusion

It is essential to adopt a systematic approach (suggested algorithm given below) when managing pleural effusions so as to accurately diagnose and promptly treat the effusion, and thus ensure that the patients' quality of life is optimised.

Diagnostic Algorithm



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Febrile Convulsions

Introduction

Febrile convulsions are the most common seizure disorder in young children. They are defined as seizures associated with fever in the absence of any other underlying cause, such as CNS infection or acute electrolyte imbalance(1).

The generally accepted criteria or consensus guidelines for a febrile convulsion agree that(2):

1) axillary temperature is > 37.8°C OR

2) presence of clinical history or examination indicative of febrile seizure

Occurring most frequently between 6 months and 6 years of age, the median age of onset is 18 months and in fact 50% of children present between 12-30 months(1). The prevalence of these seizures is between 3-8% in children up to 7 years of age but the incidence is as high as 15% in some populations(3).

Aetiology

The exact mechanism behind febrile seizures remains unknown. There is uncertainty surrounding whether it is the degree of the fever or the rate of rise in temperature which triggers the seizure which may even occur before the seizure is apparent(1). However the combination of environmental factors in individuals who are susceptible based upon their genetic make up and stage of brain development, may explain why seizures are generated in response to fever(3). Family history of febrile seizures occurs in 24% of cases and is has been found that 4% have a family history of epilepsy(1). In addition to this, monozygotic twins have a much higher concordance rate compared with dizygotic twins(3). Polygenic inheritance patterns are typical although a small number of cases have ben identified as having an autosomal dominant inheritance. Certain mutations in the genes coding Na+ ion channels and -aminobutyric acid A (GABA) receptors have been found in children with febrile seizures(1). They are both thought to be temperature sensitive proteins generating fever associated synchronized neuronal activity.

More importantly the specific pathology causing the febrile illness in the child should be identified as accurately as possible in order to determine subsequent investigation and management as well to exclude serious illness (see Table 1).

Causes of fever in children with febrile convulsions:

- Viral infections (influenza, adenovirus, parainfluenza, RSV, rotavirus, HHV6)
- Otitis Media
- Tonsillitis
- Gastroenteritis
- Post immunization (DTP, MMR)

Serious illnesses which need to be excluded:

- Meningitis
- Encephalitis
- Urinary Tract Infection
- Lower Respiratory Infection
- Cerebral Malaria

Table 1: Common causes of fever in febrile convulsions and serious illnesses to exclude. RSV: respiratory syncytial virus, HHV6: human herpes virus 6, DTP: diphtheria tetanus polio, MMR: measles mumps rubella

Classification

Simple febrile convulsions are the most common type encountered. They are predominantly tonic-clonic seizures (see Figure 1) lasting <15 minutes that do not recur within 24 hours or within the same illness(4).

Complex febrile convulsions include those that last >15 minutes, recur within 24 hours or within the same illness, have focal neurological features at onset/during the seizure or when the child does not recover within 1 hour of the initial episode(4).

Febrile status epilepticus is a febrile convulsion lasting >30 minutes either continuously or intermittently without neurological recovery(3).

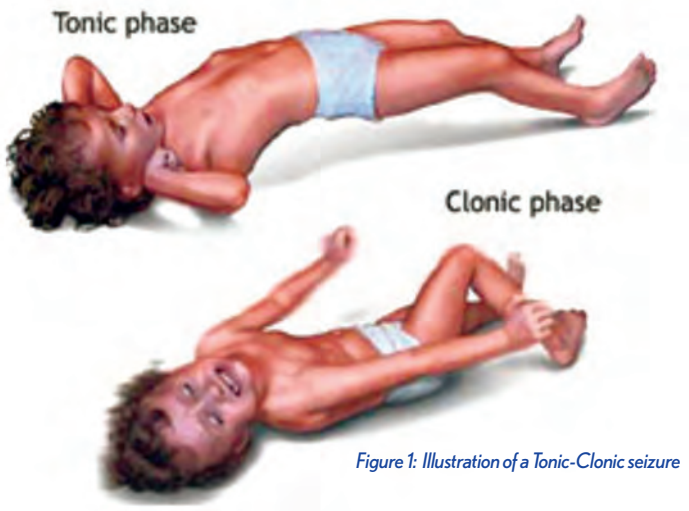

Figure 1: Illustration of a Tonic-Clonic seizure

In 87% of children the duration of the seizure is <10 minutes(1). Complex seizures occur in about 20% and are more likely to be due to intracranial infection compared to simple seizures. Febrile status epilepticus is the least common with an incidence of approximately 5%(4). The majority of children have convulsions on the first day of their illness and this may be the first presenting feature of the underlying cause.

Assessment

A detailed history should include an eyewitness account of the seizure from the parent/guardian as to the sequence of events. The description of the seizure, conscious level prior to the seizure, duration, time taken for the child to recover and condition after the episode should be ascertained. Symptoms to rule out serious illness should be determined e.g. rapid onset of illness, abnormal behavior or cry, stiffness or floppiness, leg pains, cold peripheries, pallor or mottled skin could be early symptoms of meningitis or septicemia. Past medical and family history of previous febrile convulsions or epilepsy should also be sought.

Clinical examination should include the measurement of vital signs (temperature, heart rate, capillary refill time, respiratory rate, oxygen saturations) and consciousness level, a head to toe examination for a petechial rash, palpation of the fontenelle dependent on the age of the child and special tests for signs of meningeal irritation (neck stiffness, Kernig's and Brudzinski's sign).

For babies and infants, examination is often more important and informative than the history. The NICE traffic light system(5) should be used as a guide to assess the severity of the febrile illness (see Figure 2).

A number of differential diagnoses exist for seizures in children (see Table 2) and therefore thorough clinical assessment must be used to distinguish febrile convulsions in order for the child to be investigated and managed appropriately.

Differential Diagnosis for febrile convulsions

- Rigors
- Syncope
- Breath-holding spells (tantrums causing episodic apnoea)
- Reflex anoxic seizures (vagally mediated cardiac asystole)
- Post-ictal fever (seizures lasting >10 minutes, temperature >38°C)
- Afebrile seizures with gastroenteritis
- Neurological disorders – epilepsy, encephalitis, head injury
- Metabolic disorders – hypoglycaemia, hypo/hypercalcaemia, hypo/hypernatraemia

Table 2: Differential diagnoses for febrile convulsions [4]

Investigation

Diagnostic testing is unnecessary in most children with simple febrile convulsions (see Table 3). Initial investigations including blood tests (full blood count, electrolytes, C-reactive protein, coagulation

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	Green – low risk	Amber – intermediate risk	Red – high risk
Colour (of skin, lips or tongue)	• Normal colour	• Pallor reported by parent/carer	• Pale/mottled/ashen/ blue
Activity	• Responds normally to social cues • Content/smiles • Stays awake or awakens quickly • Strong normal cry/not crying	• Not responding normally to social cues • No smile • Wakes only with prolonged stimulation • Decreased activity	• No response to social cues • Appears ill to a healthcare professional • Does not wake or if roused does not stay awake • Weak, high-pitched or continuous cry
Respiratory		• Nasal flaring • Tachypnoea: – RR >50 breaths/ minute, age 6–12 months – RR >40 breaths/ minute, age >12 months • Oxygen saturation ≤95% in air • Crackles in the chest	• Grunting • Tachypnoea: RR >60 breaths/minute • Moderate or severe chest indrawing
Circulation and hydration	• Normal skin and eyes • Moist mucous membranes	• Tachycardia: – >160 beats/minute, age <12 months – >150 beats/minute, age 12–24 months – >140 beats/minute, age 2–5 years • CRT ≥3 seconds • Dry mucous membranes • Poor feeding in infants • Reduced urine output	• Reduced skin turgor
Other	• None of the amber or red symptoms or signs	• Age 3–6 months, temperature ≥39°C • Fever for ≥5 days • Rigors • Swelling of a limb or joint • Non-weight bearing limb/not using an extremity	• Age <3 months, temperature ≥38°C • Non-blanching rash • Bulging fontanelle • Neck stiffness • Status epilepticus • Focal neurological signs • Focal seizures

Figure 2: Traffic light system used to identify serious illness. Any red amber features = urgent paediatric assessment within 2 hours. Any amber features = urgent paediatric assessment or safety net and arrange follow up. Green features = child can be managed at home

screen), blood cultures and urine culture/microscopy will be determined by the febrile illness rather than the seizure itself as per the NICE traffic light system. In regards to specific investigations for a febrile convulsion, lumbar puncture (LP) should be considered if the child is <18months or the history/ examination is suggestive of meningeal signs and symptoms. In febrile status epilepticus, bacterial meningitis has been found in up to 18% of cases therefore administration of early intravenous antibiotics is recommended followed by a LP when safe. No rationale exists for electroencephalography (EEG) investigation of febrile convulsions as there is no evidence to show that epileptiform discharges that occur have any diagnostic or prognostic implications(1). Similarly, neuroimaging with computerised tomography (CT) or magnetic resonance imaging (MRI) is not necessary unless children present with recurrent complex seizures or other persistent neurological features e.g. abnormal head circumference, signs of intracranial pressure or significant developmental delay(3).

Features	LP	Neuroimaging	EEG
Simple febrile seizure	✗	✗	✗
Complex febrile seizure	consider	✗	✗
Febrile status epilepticus	✓	✗	✗
Age <18 months	consider	✗	✗
Impression of meningitis	✓	✗	✗

Table 3: Investigations for febrile convulsions[1]

Management

1. Immediate:

Seizures which last for >5 minutes should be treated. The child should be placed in the recovery position and Airway, Breathing, Circulation should be assessed including a baseline blood glucose, Acute medical treatment includes rectal diazepam (0.5mg/kg) which can be repeated after 5 minutes if the seizure has not terminated with no increased risk of respiratory depression. A single dose of buccal (0.4-0.5mg/kg) or intranasal (0.2mg/kg) midazolam is also effective and all treatments can be administered by parents at home(4). Emergency ambulance should be called immediately if seizures last >10 minutes (including ongoing twitches) or if further seizures occur before the child fully recovers normal consciousness. There is no evidence that antipyretics reduce the number of febrile seizures or affect the rate of reoccurrence(3,4). However paracetamol and ibuprofen are most useful to relieve the discomfort of a febrile child but rigorous use of these drugs is also not recommended.

2. Further Management:

Children presenting in the community should be referred for urgent paediatric assessment when(1):

a) serious illness cannot be excluded by history and examination
b) diagnostic uncertainties exist about the cause of the seizure or a focus of infection cannot be found
c) the seizure is complex or recovery from the seizure is prolonged
d) home circumstances are unsuitable/poor psychosocial setting
e) no fever is present (non urgent referral to investigate possible epilepsy)

Subsequent seizures can be managed at home, as long as the child is well and the parents have a good understanding about how to treat the febrile illness or future seizures with an action plan.

3. Advice to Parents:

Parental anxiety can be extremely high following a febrile convulsion as many think their child is dying leading to actions that can interfere with daily family life e.g. the child sleeping in the parental bed(1). Therefore reassurance with appropriate emotional and educational support via explanation of the benign nature of the disorder (verbally and using leaflets) can alleviate fears and allow any specific questions to be answered (see Figure 3).

- ✓ What febrile seizures are...
- ✓ How to treat a fever at home - remove excessive clothing, give fluids, give antipyretics if the child is uncomfortable, tepid sponging or excessive cooling is not recommended, check the child for rash/dehydration, stay with the child at night...
- ✓ First aid if the child has a fit - recovery position, do not put anything in their mouth...
- ✓ When to call 999 - when a seizure lasts >5 minutes, non blanching rash, lack of normal alertness, dehydration...

Figure 3: Advice for parents and the important points to cover(4)

Prognosis and Prevention

The prognosis of children with febrile convulsions is favourable as there is no evidence that there is increased risk of death or that intellect, academic progression or behaviour is affected(4). Approximately 30-35% of children have recurrent seizures(1) which increases to 76% if the child has certain risk factors including younger age < 18 months, a family history of febrile seizures, a short duration <1 hour of fever before the seizure occurs and a lower temperature (close to 38°C) during the seizure (see Table 4).

Risk Factor	Recurrent Febrile Seizure	Epilepsy
Age <18 months	✓	✗
FH of febrile seizures	✓	✗
Lower temperature close to 38°C during seizure	✓	✗
Short duration < 1 hour of fever before the seizure	✓	✓
Prolonged seizure > 15 mins	✗	✓
Multiple seizures in 24 hours	✗	✓
Focal features	✗	✓

Table 4: Risk factors for recurrent seizures or epilepsy[1]

Febrile Convulsions

(continued)

Neurological sequelae are rare following febrile convulsions and the vast majority do not develop epilepsy. The risk of epilepsy increases with complex seizures, a family history of epilepsy and a short duration <1 hour of fever before the seizure occurs. Children with these features have a 10% risk by 7 years of age. However children without these features have a 2.4% chance of developing epilepsy by the age of 25 compared with 1.4% which is the risk of the general population(1). Prolonged febrile convulsions (>15 minutes) increases the risk of epilepsy to 21%. Studies have shown that prolonged seizures with fever are associated with sclerosis of the hippocampus, which is hypothesised to be abnormal before the seizure, therefore predisposing the child to this type of seizure(1).

No compelling evidence exists that continuous anticonvulsant treatment reduces the risk of epilepsy and the side effects of these drugs outweigh the potential benefits for most patients therefore prophylactic treatment is not recommended(3). Diazepam (oral and rectal) at high doses, given at the onset of the illness may prevent febrile convulsions but its adverse effects including ataxia, lethargy and irritability can mask an underlying serious illness.

Ultimately febrile convulsions can be common benign cause of seizures in children with an excellent outcome. As clinicians we need to accurately assess these children to ensure we exclude serious causes of the febrile illness, particularly CNS infections and be able to recognise when children need to be referred for urgent specialist review. The mainstay of management however is centered around effective communication with parents; acknowledging concerns, providing support and educating them about the disorder to ensure they are empowered to manage subsequent febrile convulsions without fear and anxiety.

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CHEST PAIN

following a recent aortic valve replacement

Presentation

A 64 year old man presented to the emergency department complaining of a 48 hour history of cardiac-sounding chest pain. His past medical history included a bioprosthetic aortic valve replacement (AVR) for severe calcific aortic stenosis 2 months previously, COPD and hypertension. A pre-operative coronary angiogram had demonstrated normal epicardial coronary arteries.

During initial assessment, medical staff monitored a bradycardic episode with a ventricular rate of 20bpm and a 10 second asystolic pause. Subsequent 12 lead ECGs demonstrated bradycardic atrial fibrillation with pauses interspersed with transient ST-segment elevation in the anterior chest leads V1-V6. Due to his marked clinical instability, external pacing was initiated and he was subsequently transferred directly to the catheter laboratory and a single chamber ventricular permanent pacemaker implanted as an emergency procedure. An initial high-sensitivity Troponin T was measured at 128ng/l (reference range 0-14ng/l), and had risen to 1271ng/l at 6 hours. The preliminary diagnosis was of an acute coronary syndrome STEMI leading to conduction system disturbance and a haemodynamically compromising bradycardia.

Investigations

Blood results showed marked leucocytosis, anaemia, elevated CRP, acute kidney injury and deranged liver function tests.

A transthoracic echocardiogram performed 24 hours after admission demonstrated moderately severe left ventricular (LV) dysfunction, with akinetic mid-apical regions and bilateral atrial dilatation. His tissue AVR was opening well with no significant regurgitation. This was a marked change from his pre-operative echo which was reported as mildly impaired LV dysfunction with an AV that had reduced opening and a peak gradient of 58mmHg, mean gradient 30mmHg.

Management

Following pacemaker insertion the patient was rhythmically and haemodynamically stable but remained clinically unwell. The cause of a significant myocardial infarction in a gentleman in a low risk group due to a recently normal coronary angiogram remained unclear but the possibility of endocarditis with a septic coronary embolus was considered early by the team. Whilst not obviously septic, his recent tissue AVR with a raised CRP, eucocytosis and anaemia, meant the clinical suspicion was high and blood cultures were sent in the absence of a significant fever. A transoesophageal echocardiogram was arranged and after discussion with the microbiology team antimicrobial treatment was started. ECG showed resolution of ST segment within a few days. Blood cultures subsequently grew a gram positive streptococcus (later identified as an enterococcus).

The Trans Oesophageal Echo (TOE fig 1) showed impaired LV systolic function with LA and LV spontaneous contrast. The AVR was seated well and opened with mild restriction and had mild AR. A mobile mass was seen on the aortic side of the valve which was suspicious of vegetation and confirmed our diagnosis.

After 6 weeks of intravenous antibiotics in the hospital, the patient was discharged home with cardiology follow up. A repeat transthoracic echocardiogram before discharge demonstrated that the aortic prosthetic valve was opening well with mild aortic regurgitation. Leucocytosis, CRP, renal and liver function had returned to normal.

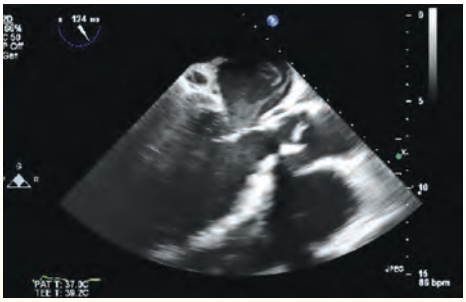

Figure 1: TOE showing suspected vegetation on aortic valve

Discussion

Despite our understanding and progress in the diagnosis and treatment of infective endocarditis (IE) (1,2,3), mortality rates over the past 25 years have not changed significantly (4) and it remains an important cause of in-hospital mortality, with figures ranging between 10 and 26%.

Endocarditis can present in many different ways; varying from a chronic, subacute condition to an acute presentation such as the case we describe here. The Duke criteria; first proposed in 1994 (5), are still the cornerstone of diagnosis and calculating this score is recommended to assist in confirmation. In recent years many modifications have been suggested to increase its sensitivity. (6) However, it should be remembered that this system is still not infallible due to the highly diverse clinical presentations of IE, and therefore diagnosis and treatment cannot rest solely on these criteria.

The ICS-PCS trial (4) reviewed the presentation and characteristics of 2781 patients presenting with endocarditis. Most importantly, this has found that the presentation of IE is changing. The classical subacute or chronic onset of clinical symptoms and signs is becoming rare. For example; the rare presence of Osler's nodes (3% of patients), Janeway lesions (5%), splinter haemorrhages (8%) or splenomegaly (11%). Infective endocarditis has become a more acute disease with varying characteristics, more commonly presenting in those with prosthetic valves or in intravenous drug users (IVDUs). In relation to the case we report above, 68% were male, 17% presenting as a vascular event and 8% as a new conduction abnormality. Other studies have reported the incidence of acute coronary syndromes as low as 2.9% (7).

The ICE-PCS trial also found that the most common bacterial causes of prosthetic valve IE to be staph aureus (23%), coagulase negative staph (17%), strep viridans (12%) and enterococcus species in 12%. Negative cultures were found in 12% of cases. In native valve IE, staph aureus and strep viridans are the most common causative organisms, particularly in IV drug users. Our local antimicrobial guidelines are guided by this and similar evidence, so that when a specific bacterial cause is suspected, an appropriate antibiotic therapy can be administered. This should be then altered depending on sensitivities found on blood cultures, of which a minimum of 3 sets should be obtained prior to starting antibiotics. Our current local guidelines follow the BSAC recommendations (8) (see table), however an individual's local trust guidelines should be followed.

Antibiotic Prophylaxis

A common misperception is that antibiotic prophylaxis against IE is needed for at-risk patients undergoing dental or surgical procedures. Both the British Society for Antimicrobial chemotherapy and NICE currently do not recommend this; however when there is

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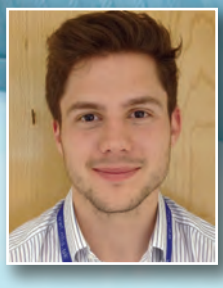

Table 1. Definition of IE According to the Modified Duke Criteria[a]

Definite IE
Pathologic criteria
Microorganisms demonstrated by results of cultures or histologic examination of a vegetation, a vegetation that has embolized, or an intracardiac abscess specimen
Pathologic lesions: vegetation, or intracardiac abscess confirmed by results of histologic examination showing active endocarditis
Clinical criteria
2 Major criteria
1 Major criterion and 3 minor criteria
5 Minor criteria
Possible IE
1 Major criterion and 1 minor criterion
3 Minor criteria
Rejected
Firm alternate diagnosis explaining evidence of IE
Resolution of IE syndrome with antibiotic therapy for ≤4 d
No pathologic evidence of IE at surgery or autopsy, with antibiotic therapy for ≤4 d
Does not meet criteria for possible IE
Definition of Terms Used in the Modified Duke Criteria for IE Diagnosis
Major criteria
Blood culture findings positive for IE
Typical microorganisms consistent with IE from 2 separate blood cultures
Viridans streptococci, *Streptococcus bovis*, HACEK group, or *Staphylococcus aureus*
Community-acquired enterococci, in the absence of a primary focus
Microorganisms consistent with IE from persistently positive blood culture findings, defined as:
≥2 positive culture findings of blood samples drawn >12 h apart
3 or most of ≥4 separate culture findings of blood (with first and last sample drawn ≥1 h apart)
Single positive blood culture for *Coxiella burnetii* or antiphase I IgG antibody titer >1:800
Evidence of endocardial involvement
Echocardiographic findings positive for IE (TEE recommended in patients with prosthetic valves, rated at least possible IE by clinical criteria or complicated IE [paravalvular abscess]; TTE as first test in other patients), defined as follows:
Oscillating intracardiac mass on valve or supporting structures, in the path of regurgitant jets, or on implanted material in the absence of an alternative anatomic explanation
Abscess
New partial dehiscence of prosthetic valve (including new valvular regurgitation, worsening or changing of preexisting murmur not sufficient)
Minor criteria
Predisposition, predisposing heart condition, or intravenous drug use
Fever, temperature >38°C
Vascular phenomena, major arterial emboli, septic pulmonary infarcts, mycotic aneurysm, intracranial hemorrhage, conjunctival hemorrhages, and Janeway lesions
Immunologic phenomena: glomerulonephritis, Osler nodes, Roth spots, and rheumatoid factor
Microbiological evidence: positive blood culture finding but does not meet a major criterion[b] or serologic evidence of active infection with organism consistent with IE
Echocardiographic minor criteria eliminated

Acute presentation	Flucloxacillin (8-12g daily IV in 4-6 divided doses) ***Plus*** Gentamicin (1mg/kg body weight IV 8 hourly, modified to renal function)
Indolent presentation	Penicillin (7.2g IV daily in 6 divided doses) or ampicillin/amoxicillin (2g IV 6 hourly) ***Plus*** Gentamicin (1mg/kg body weight 8 hourly IV, modified according to renal function)
Penicillin allergy	Vancomycin (1g 12 hourly IV, modified to renal function)
Intra-cardiac prothesis / suspected MRSA	Plus Rifampicin (300 - 600mg 12 hourly by mouth) ***Plus*** Gentamicin (1mg/kg body weight 8 hourly IV, modified according to renal function)

When to offer prophylaxis

- **Do not offer** antibiotic prophylaxis against infective endocarditis:
  - to people undergoing dental procedures
  - to people undergoing non-dental procedures at the following sites:
    - upper and lower gastrointestinal tract
    - genitourinary tract; this includes urological, gynaecological and obstetric procedures, and childbirth
    - upper and lower respiratory tract; this includes ear, nose and throat procedures and bronchoscopy.
- **Do not offer** chlorhexidine mouthwash as prophylaxis against infective endocarditis to people at risk undergoing dental procedures.

Top:
The Modified Duke Criteria 4,6

Above:
Table 2: BSAC recommendations for the empirical therapy of endocarditis 8

Left:
Figure 2: NHS guidance on antibiotic prophylaxis10

a suspicion of infection following these procedures, prescribing an antibiotic that covers common IE-causing organisms is recommended (10). They do recommend keeping a high-level or oral and dental hygiene to help prevent IE.

The management of ACS secondary to IE is debated. Coronary angiography is useful in that it can establish the presence of septic emboli in the coronary artery. However, there is a risk of contact with any vegetation releasing systemic emboli. Therefore angiography has only reported to be safe if no vegetation is observed on the aortic valve (11). Prompt antimicrobial treatment is vital as it has been shown to dramatically decrease the risk of embolic events (12).

Surgery in Infective Endocarditis

Indications for early surgery in the treatment of IE include congestive cardiac failure, large vegetations seen on echocardiography (>10mm), cerebrovascular complications and persistent sepsis. Congestive cardiac failure is the clearest indication for surgery as it substantially reduces mortality rates when compared to medical therapy alone. Virtually all prosthetic valves affected will need surgical intervention at some point (13).

Learning Points

1. Infective endocarditis remains an important cause of inpatient mortality despite advances in diagnosis and treatment
2. Infective endocarditis is a rare but important differential diagnosis in patients presenting with ACS or symptomatic bradycardia following valve replacement surgery
3. The classical history and clinical signs are now rare and it's onset is far more acute, commonly on a background of IV drug use or prosthetic valve implantation
4. Prompt antimicrobial treatment reduces the risk of further embolic events
5. Antibiotic prophylaxis is not needed in patients undergoing dental or surgical procedures.

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The management of Infertility in a Primary Care setting

Infertility is defined as failure to conceive after 1 year of unprotected intercourse. Over 80% of couple in the general population will conceive within 1 year with regular unprotected sexual intercourse (if the woman is under 40years of age). Of those who do not conceive in the first year about half will do so in the second year (cumulative pregnancy rate of over 90%).

In the UK, approximately 1 in 7 couples (14%) will experience difficulty in conceiving with an annual incidence estimated at 1.2 couples per 1000 total general population. Infertile couples frequently approach their General Practitioners to seek advice and referral to the specialists. Therefore, a basic understanding of the overall management will aid in implementation of care, avoid unnecessary investigations and will facilitate appropriate escalation from primary care to secondary and tertiary settings.

When to investigate?

NICE recommends investigating the couple if they have not conceived in 1 year following unprotected intercourse. However in the presence of risk factors such as amenorrhea, oligomenorrhea, and pelvic inflammatory disease, or where a woman is aged 36 years or over, and any known male factor (history of undescended testis), investigation should be offered at first contact.

What are the causes?

- Male factor: Oligo/azoospermia secondary to genetic, iatrogenic causes, or secondary to medical /surgical treatments
- Female factors: Ovulatory or Tubal factors or unexplained infertility.

In the majority (40%) of couples, disorders are found in both the man and woman.

The main causes of infertility in UK are (approximate figures):

- Factors in the male causing infertility (30%)
- Unexplained infertility (25%)
- Ovulatory disorders (25%)
- Tubal damage (20%)
- Uterine or peritoneal disorders (10%)

How to investigate?

A detailed history from both partners aids in appropriate assessment and general advice should be given to the couples *(Table 1)*

Initial Assessment

Preliminary screening for infections such as Chlamydia and susceptibility to Rubella form the main components of the initial assessment.

- Where the woman is positive to chlamydia it is important to treat both the partners and do the contact tracing. Depending upon the local arrangements, couples may need to be referred to local GUM services.
- If the woman is seronegative to rubella, she should be offered immunization with MMR and be advised to use barrier methods of contraception till her follow up blood test a month later has confirmed to be sero-positive.

Baseline Investigations:

Given the wide range of causes, it is important to do appropriate investigations. These are directed primarily at three factors:

- Semen analysis
- Assessment of ovulation and
- Evaluation of tubal patency and to rule out uterine abnormalities.

Semen analysis: Depending upon the local arrangements, semen sample must be sent to the relevant laboratory. It is good practice to provide them with written information about the actual procedure. The couple should ideally abstain intercourse for three days before producing a sample and should not use a condom either. This consultation should be sensitive to cultural and religious beliefs.

- Recent WHO Classification (2013) categorizes a fertile semen sample as following:
  - Volume: 1.5ml or more
  - pH: 7.2 or more
  - Sperm Concentration: 15million spermatozoa/ml
  - Total motility: 40% or more motile or 32% or more with progressive motility
  - Vitality: 58% or more live spermatozoa
  - Sperm morphology (% of normal forms): 4% or more

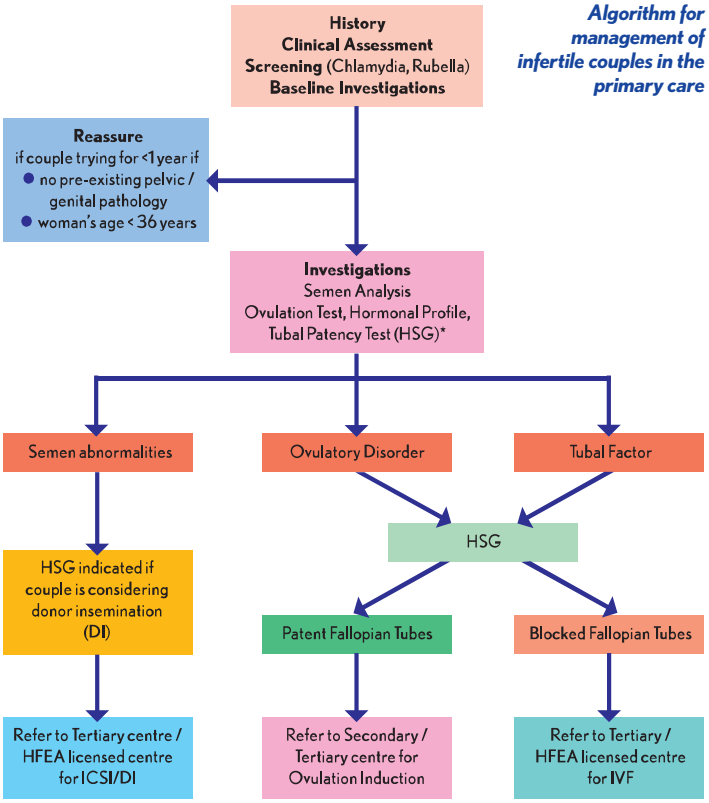
If semen analysis is abnormal, repeat testing is requested in 3 months to allow time for the cycle of spermatozoa formation.

- **Biochemical evidence of ovulation:**
  - This is done by estimation of midluteal phase serum progesterone (day 21 in women with regular cycles i.e. 28 days). In women with irregular cycles (a maximum cycle length of 42 days), twice weekly serum progesterone levels can be checked from day 21 until the next period.
  - Further assessment of basal hormonal profile (Prolactin, FSH, LH and estradiol) is done in the early follicular phase (day 2 to day 5) for women who are over 36 years or those with irregular cycles.
  - In women with irregular cycles and the hormonal profile suggestive of polycystic ovaries, should be referred for a trans vaginal scan to assess ovarian volume (> 10 ml) and the morphology of ovaries (> 12 follicles around the peripheries- pearl necklace appearance as per the Rotterdam criteria - 2003)
- **Establishing tubal patency:** There are two methods for assessing tubal patency:
  - Hystersalpingography and
  - Diagnostic laparoscopy and trans- cervical injection of methylene blue dye.

Hystero-salpingography: It is reliable in ruling out tubal occlusion and is less invasive. This is an outpatient based procedure to check tubal patency for women who do not have a history of endometriosis, pelvic inflammatory disease, previous ectopic pregnancy, suspected genital organ developmental abnormality or have had a reconstructive surgery of the uterus. An open access hystersalpingography (HSG) to assess the tubal status, as the first line investigation was introduced by NICE in 2013 in England. This should be considered once chlamydia screening test has been reported to be negative.

Diagnostic laparoscopy and dye test is now considered as a second line investigation for women where HSG has been unsuccessful, or results of HSG are inconclusive or she is in the above high-risk group.

Management *(See Algorithm)*



Algorithm for management of infertile couples in the primary care

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* Accessibility to HSG may not be available to all primary care centers

Male factor Infertility:

An abnormal semen analysis would indicate referral of the couple to a tertiary unit or a HFEA, (Human Fertilization and Embryology Authority) licensed unit for Intracytoplasmic Sperm Injection (ICSI) and / or donor insemination (DI). These women require HSG if they are considering donor insemination (rules governing DI were modified in 2005 lifting the anonymity of donors).

Ovulatory disorder:

Abnormalities in hormonal profile are an indicator for referral to a secondary unit for further investigation and management such as ovulation induction. HSG may be deferred until the underlying cause in this scenario has been established.

Unexplained Infertility:

A normal semen analysis, ovulatory progesterone, and a normal HSG lead to a working diagnosis of unexplained infertility and can be managed expectantly, taking into consideration the age of the female partner. Around 46% of these couple conceive in the next two years. However women over 35 year old and a history of primary unexplained infertility for more than 12 months should be referred to the secondary care unit.

General advice: *(Table 1)*

Table 1: General Advice & Education:

- Stop smoking
- Restrict alcohol intake to 1-2 units/week (women) and 3-4 units / week (men)
- Stop recreational drug intake
- Adopt healthy life style
- Optimization of BMI (if >30 or <19)
- Men to avoid working in hot conditions or wearing tight underclothing
- Regular intercourse - 2-3 times a week (Do not use basal body temperature charts or LH kits which increase stress)
- Pre-conceptual Folic acid – prophylaxis against Neural Tube Defects

Knowledge of cumulative conception rate (as alluded to earlier) helps reassure the couples that approach for help in the community.

- High (BMI) body mass index (>30) of the woman: Couples should be counselled as regards weight management strategies. A high BMI is associated with infrequent ovulation, reduced fecundity, increased risk of spontaneous miscarriages and a higher failure rate with ovulation induction or in vitro fertilization (IVF). Some of these women with a very high BMI may not be suitable for onward referral for HSG due to practical logistics involved in this procedure.

Equally important is to evaluate the women with low BMI (below 19) for the underlying problems such as eating disorders and psychological issues.

- General advice regarding folic acid supplementation (400mcg per day) for prevention of neural tube defects should be given. Women with high BMI (> 40), diabetes, epilepsy and those with previous history of neural tube defects should receive higher prophylactic dose (5mg) in the peri-conceptual period.

Referral:

It is important to be aware of the local protocols for referrals and policy for funding which varies across the country.

When to refer? *(Table 2)*

- Any couple trying to conceive for 1 year after unprotected intercourse, who have undergone preliminary investigations with an identifiable cause can be referred to the specialist unit.
- Age of the female partner is an important factor that needs to be taken into consideration in fast tracking the referral of couples. Natural conception rate falls significantly when the woman's age is 35 years or over as the ovarian reserve generally declines. Pregnancy rate with Artificial Reproductive Technology (ART) reflects this. In 2013 as per NICE recommendations, upper age limit at the time of ART has been increased up to 42 years.

Table 2: Indications for Early Referral (NICE Recommendations 2013):

Female factors:

- Age >36 years
- Previous Pelvic Inflammatory Disease/ Sexually Transmitted Infection (STI)
- Amenorrhea/ Oligomenorrhea
- Previous abdominal or pelvic surgery

Male factors:

- History of Genital Pathology (Undescended Testes)
- Previous Urogenital Surgery
- Previous STI
- Significant Systemic Illness

Cancer treatment: Where treatment may lead to infertility

Chronic viral infections: Hepatitis B, Hepatitis C or HIV for safe risk reduction investigation and treatment

- Women with unexplained infertility can be offered IVF for who have not conceived after 2 years of regular unprotected intercourse; hence an appropriate referral could be made.

Where to refer?

Couples could be referred either to

1. Secondary units which are not HFEA-licensed, where ovulation induction protocols (with Clomiphene or Gonadotrophins) are implemented or
2. Tertiary units that are HFEA-licensed, where IVF and other artificial reproductive technologies are carried out.

Referral of couples with semen problems or tubal disorders should be to dedicated specialist fertility units capable of delivering the treatment necessary, typically Intra-Cytoplasmic Sperm Injection (ICSI) or IVF respectively.

Same sex couples could be referred directly to the tertiary units for their necessary management.

It has been shown that up to 38% of couples referred to non-HFEA licensed secondary units have either tubal or semen abnormalities. Over 60% of IVF cycles in UK are carried out in private sector (Table3). However, only 5% of these are referred from primary care directly to private care while the rest are referred to NHS care, which in turn refers couples for private treatment. These inappropriate and misdirected referrals can be avoided by formulating referral pathways within the local commissioning consortium.

Table 3: Couples who are not eligible for ART under NHS:

Direct referral to Private sector

- Those who have a child already from this relationship or from a previous relationship who is living with the couple
- Woman had been sterilized in the past
- Male partner had vasectomy
- Woman's age over 42 years

Infertility is not life threatening, however, has huge psychosocial consequences. Hence education, reassurance and timely escalation of the individual cases to the relevant area all play a vital role in its management.

References:

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2. Epidemiology and management of infertility: a population-based study in UK primary care - Scott Wilkes, David J Chinn, Alison Murdoch and Greg Rubin. Family Practice. 2009; 26(4): 269-274
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"Aim High and you will succeed": A time-tested dogma

Mr Anthony Victor Babu Bathula
Associate Speialist Surgeon, MBBS; MS (Surgery); DNB (Surgery); FRCSEd; FRCS (Eng); Dip Lab Surg; MBA (Health Executive), Glan Clwyd Hospital, Rhyl.

I emigrated to the United Kingdom in 1999 with high ambitions to succeed in life. I had obtained Fellowship (FRCSEd) in Surgery in the year 2000. My aim was to get a job soon in NHS and start my career as a surgeon. After a year of doing locums at House Officer and Senior House Officer (SHO) grades I managed to get a two year substantive training grade Senior SHO in Scotland. I tried my best to get into Specialty Training, and eventually had to make a choice to settle down with a staff grade job supporting my family in North Wales. Today I am a proud Associate Specialist Surgeon in Surgery at Glan Clwyd Hospital of Betsi Cadwaladr University Local Health Board (BCULB) in North Wales, with a special interest in 'Clinical Leadership and Management'.

In 2006, I went to attend a 'Clinical Management and Leadership' course for SAS doctors in Keele University, just to escape from routine hospital work. By then I had realised I could not achieve more than what I could do as an SAS (Staff and Associate Specialist) doctor. I was just surviving in my professional career, having forgotten how to thrive. All my high ambitions were on hold. I never, ever thought of a career in Health Management, nor believed that I could be a leader. On Friday 29th September 2006, the last day of this course changed my perspective forever. There was this speaker, Dr Umesh Prabhu, the then Medical Director of Bury NHS Trust, who delivered an inspiring talk explaining the various options / opportunities that are available in NHS (National Health Service) for all doctors, especially for the SAS doctors. In his talk he said, "aim high and you will succeed". Since then I have tested this dogma time and time again, only to find it be 100 percent true.

With renewed enthusiasm and ambition I went on to do a master's degree in Health Management (MBA - Health Executive) with Merit from Keele University, truly believing in a bright career in NHS Corporate Leadership. My understanding and knowledge in NHS and Health Management has increased beyond measure ever since I started this degree. I believed that education is the only way forward should I ever want to thrive, and therefore made 'education' paramount in my life. Since 2006, I have gone on to do two more post-graduate degrees in Surgery and two post-graduate degrees in Health Management. Having never given up studies, I sincerely hope to obtain a Masters degree in Corporate Leadership in the NHS from the University of Chester by the end of this year.

I had always resisted discrimination. I felt that I was discriminated when I had to do an exam in English (IELTS) to obtain registration with the GMC (General Medical Council) whilst my colleagues from the European Union did not have to. I had difficulties in getting the required score in IELTS for a year. In 2000, even before I obtained my registration with the GMC, I did not hesitate to raise this issue with the President of the then RCSEd, GMC, then the Health Minister, and the Prime Minister. I am in favour of all the frontline clinicians having proficiency in English language. In 2010, I did set up an e-petition to the Prime Minister in this regard urging him to bring in legislation to test all doctors' English equally. I am glad that the legislation is about to change for the good of our patients, addressing issues that I raised 10 years ago.

Over the years I had learned not to be put down by people. I have resisted many of my peers who have tried to restrict me into what they want me to become. It is important for me to strike a balance of pleasing my colleagues to win their favour and to focus on running my own race without any distractions. We all face bouncers in life. One day I was told that I would not have the same opportunity as that of a trainee to gain expertise in one of the commonly done surgical procedures. I was disappointed and had no hesitation in announcing my decision to challenge such guidance. I was requested to look at the Royal College of Surgeons of England (RCSE) website should I require any further clarification in this regard. I did look at the website, and to my surprise found an advert to apply for the SAS doctors' committee membership at RCSE. I was successful in not only becoming a member through an application and interview process but went on to become this committee's Co-Chair just a few months later in 2009. I had the opportunity to represent the interests of the SAS surgeons in the College Council for the subsequent four years in this capacity. From then on I went on to learn how best to score runs using the opponents pace.

I was a university lecturer in Surgery before I came to the United Kingdom. I have always enjoyed teaching and training. I made use of every opportunity available in and outside the hospital to keep up my teaching skills. For the last 5 years, I have been invited to several SAS doctors' study days across the United Kingdom to promote interest in Health Management and Clinical Leadership. To create interest, bring in enthusiasm and to inspire doctors to achieve excellence in their profession is no easy task. I continue to enjoy promoting this Professional Development among SAS doctors and Consultants by teaching Health Management and Leadership. I am delighted at having recently been appointed a 'Visiting Speaker' at Keele University School of Medicine's Keele Clinical Leadership Academy, delivering lectures in Clinical Leadership and Management.

I believe that nothing comes in the way of my life that is too much for me to handle. As long as I believe that I am strong, well qualified, equipped with wisdom, empowered with knowledge and above all filled with that 'I can do' power within me, I would be 'able to handle' any challenge that should come my way. In the year 2010, I faced the challenge of representing the SAS surgeons in SAC (Specialty Advisory Committee) in Trauma Orthopaedics of the UK and Republic of Ireland by being a General Surgeon. I did this job well for the next two years.

Being an SAS doctor in hospital has its limitations in what one wants to do and is allowed to do, so I began to look for available opportunities out in the community beyond working in the hospital. In April 2008, I accepted an opportunity to join the Trustees of a Charity called North Wales Regional Equality Network (NWREN) to serve my fellow citizens in North Wales. Through this charitable work I continue to draw great pleasure and satisfaction in serving my fellow citizens in North Wales, by protecting the human rights and promoting Equality, and by challenging discrimination in all its forms. I am now the current Chair of this charity, fully responsible to achieve this organisation's strategic aims and objectives. Representing NWREN, I had the opportunity to work with the planning department of BCLHB (Betsi Cadwaladr University Local Health Board) to be a part of the team providing equality impact assessment in the ongoing reconfiguration of health services.

In 2010, I was invited to work with the newly constituted 'Stakeholders Reference Group' of BCULHB. This group is appointed by the Welsh Government to provide continuous engagement and feedback to the Local Health Board representing the people of North Wales. Representing NWREN in this group is an honour. In November 2013, I was elected to become the Chair of the Stakeholders Reference Group. In this capacity I am appointed by the Health Minister of the Welsh Government to become an Associate Member of the 'Health Board' of BCULHB, to represent the communities of North Wales in the Health Board. This assignment of Health Board working for an SAS doctor is next to an impossible job opportunity in NHS (National Health Service). This is the greatest height that I have reached so far and I sincerely hope to do a good job in this capacity.

In recognition to my work done inside and outside the hospital, I was very fortunate in getting the BEST doctor (Best Educational Supervisor and Trainer) award for brining innovation in to the education by the Wales Deanery for the year 2012. Her Majesty the Queen extended an invitation to attend the Royal Garden Party at Buckingham Palace in 2012 for protecting Human Rights and for promoting equality by challenging discrimination in all its forms in North Wales. These are humble reminders both to me and my colleagues of the things that can be achieved by an SAS doctor.

Very often we focus on things that we do not have. Most often we fail to see what we have at our disposal, and fail to make the best use of the talents, resources, ideas and opportunities that we already have. In some instances, in deep disappointment, we make ourselves worse by creating obstacles in furthering towards our divine destiny of success, failing to realise our own potential. Considering the number of setbacks one has had, it is easy for one to slip into a survival mode. I came to the United Kingdom to thrive, not to survive. Soon I realised that I was not aiming high enough to achieve all that I could. There are plenty of opportunities available out there for all doctors in the United Kingdom. Our grade in employment and our background should never be barriers in tapping these opportunities. All you need to do is to aim high and you are bound to succeed.

ACCEA

ADVISORY COMMITTEE on CLINICAL EXCELLENCE AWARDS

2015

The ACCEA has now announced that the 2015 Round is now open, and will close at 17:00 hours on Thursday 14th August. If you are intending to make an application you are strongly advised to check the ACCEA Nominal Roll to make sure your details are correct. Applicants who wish to apply for BIDA support MUST submit their application to bida@btconnect.com, and the application must be received by 5 pm on Thursday, 17th July 2014.

Application forms, FAQs etc. will be found on the ACCEA website as soon as it has been updated at https://www.gov.uk/government/organisations/advisory-committee-on-clinical-excellence-awards. Please note that these forms are for BIDA USE ONLY but you will be able to cut and paste from these forms to the online ACCEA forms when they are available. As previously, Bronze and Silver applicants may choose to submit one of the additional options: Research, Management, Teaching/Training

Gold applicants may choose to submit up to two

Divisional News

North Wales Division

At the North Wales division of BIDA meeting On the 11th of June 2014, Consultant Urological Surgeon, Mr Iqbal Shergill and Dr Sanjay Agarwal, Consultant radiologist gave a joint presentation. This provided update on the diagnosis and management of Prostate Cancer as well discussed common urological problems like renal colic and haematuria with specific emphasis on imaging. Prostate cancer remains the main health concern in the male populations'. The advances in imaging mean that it is now possible to biopsy specific areas in the prostate gland and thus achieve a highly accurate diagnosis. A joint approach between radiology and urological surgery allows for accurate diagnosis and provide specific treatment options for this condition. Both speakers stressed the importance of being vigilant about the health issues in men that are often neglected until the disease is advanced

Report submitted by Sanjay Agarwal and Jay Nankani

Scottish Division

BIDA Scottish Division held a meeting on the 5th of June 2014 in Spice restaurant in Hamilton, Lanarkshire. The topic of the meeting was Management of Eczema and Acne in Primary care. The speaker was Dr Girish Gupta, Consultant Dermatologist and senior lecturer at the University of Glasgow. The meeting was very educational and informative. The meeting relevant to general practice. It was attended by members of BIDA Scottish division, GP and hospital doctors. Dr KB Singh MBE, Chairman Scottish division, chaired the meeting and the vote of thanks was given by Dr Chinta Mani, Vice Chairman division.

Since staging these regular educational meetings, BIDA Scottish division has increased its membership as a result.

*Report submitted by **Dr K B Singh***

Congratulations!

Dr Satya Sharma MBE

Dr Satya Sharma MBE has been appointed Deputy Lieutenant (DL) for West Midlands by Lord Lieutenant (LL) Mr Paul Sabapathy CBE. The appointment is until the age of 75 and the title can be retained for life. He has been asked to serve on awards and honours committee. Dr Sharma said that these achievements are reflections of well wishes from friends and families and that this honour has been a humbling experience.

Dr Sharma is a very active BIDA member, Chair Promoting Organ Donation (POD), Chair India UK (INUK) forum and Vice Chair BMA regional council West Midlands.

Obituary

Dr Ishwar Munsadia

Dr Ishwar Munsadia was born in Kenya in October 1934 and came to the UK to study in 1954. He graduated from University of Aberdeen Medical School in July 1963 and worked in various hospitals within the UK including Aberdeen, Newcastle and Rochdale.

He became a Consultant Psychiatrist at Birch Hill Hospital Rochdale in 1976. He was very well respected and a leader in his profession. He was committed to improving psychiatric services in teaching and training staff in his workplace.

He became a member of ODA (BIDA) in the 1970s and served as President and Chairman of the Rochdale Division as well as other National Executive posts. He was awarded the Honorary Fellowship of BIDA in 1989.

He endeavoured to serve the community at large and contributed towards greater understanding with in the multiracial and multi-ethnic society. He was a Rotarian (President of Rotary Club of Heywood). He also served as the JP on the Bench. A very warm hearted man with a good sense of humour was well respected by his peers and colleagues. A proud family man Dr Munsadia is survived by his wife Susheila and children Ranjna, Camilla, Sarita and Hitesh and his grandchildren Daniel and Nathan

Report submitted by Dr Pranab Sarkar, Secretary, Burnley, Pendle & Rossendale Division of BIDA

of the additional options and Platinum applicants may choose to submit all three. The use of these supplementary options is not mandatory and, if you choose to use them, please make sure that the corresponding domain in the application form is left blank.

The Awards remain extremely competitive and BIDA, as a Nominating Body, is only permitted to support a very limited number of applications in each category. If you are unsuccessful in obtaining College support you should not be discouraged from making an application as those with strong Trust support have been successful in previous years. To maximise your chances of success you must ensure that you are able to score in each of the 5 domains.

The design of the form remains that used in previous years but, when available, the 2014 appliction has had a countdown facility introduced on boxes where there is a character limit.

Dr Ahmed Sadiq
Chairman,
BIDA Hospital Doctors' Forum

TOGETHER, WE ARE STRONGER

BRITISH INTERNATIONAL DOCTORS' ASSOCIATION

The British International Doctors Association (BIDA) is a professional doctors' association. Its sole objective is promoting ***Equality*** and ***Fairness*** for all doctors and dentists working throughout the UK.

BIDA's mission is to achieve equal treatment of all doctors and dentists based on their competence and merit, irrespective of their Race, Gender, Sexual orientation, Religion, Country of origin or School of graduation.

If you believe in this mission and would like to be part of this endeavour, join us!

- You will make professional contacts, gaining the opportunity to network with people who can impact your profession, and giving you access to new opportunities, friends and information.
- In addition to being part of a group of like-minded professionals, and having the recognition of your peers, specific member benefits include:
  - Attending BIDA-organised international, national and regional conferences, seminars, meetings and many other educational and social activities
  - Constant access to pastoral support
  - Nominations for excellence awards
  - BIDA Journal, our Scientific journal, complete with news, interviews and much more.

If you are interested in joining BIDA, or would simply like to know more about us, please either write to **BIDA, ODA House, 316A Buxton Road, Great Moor, Stockport, SK2 7DD** or e-mail us at **bida@btconnect.com**, or contact us through our website at the address below.

We look forward to hearing from you!

www.bidaonline.co.uk