## Contents

BIA Corporate Sponsors ................................................................. 4

British Infection Association Council Members ............................. 5

Membership Enquiries and Administration Details ............................ 6

Meeting Programme ........................................................................ 7

Keynote Lectures ........................................................................... 11

Free Scientific Papers 1-15 ............................................................. 12

Clinical Papers A-F ....................................................................... 29

List of Delegates ............................................................................ 35

Notes Pages ...................................................................................... 38
CORPORATE SPONSORS

ViiV Healthcare is a global specialist HIV company established in November 2009 by GlaxoSmithKline and Pfizer dedicated to delivering advances in treatment and care for people living with HIV. The company’s aim is to take a deeper and broader interest in HIV/AIDS than any company has done before and take a new approach to deliver effective and new HIV medicines as well as support communities affected by HIV.

RSTMH promotes and advances the study, control and prevention of disease in humans and other animals in the tropics and plays a leading role in increasing awareness throughout the world of tropical medicine and international health issues.

Throughout our international network of Fellows we facilitate training, education and exchange of information between clinicians, health-related scientists, to non-governmental development organisations and students across all disciplines in the fields of tropical medicine and international health.
BRITISH INFECTION ASSOCIATION COUNCIL

President: Dr Peter Moss (Hull & East Yorkshire)
Department of Infectious Diseases, Hull and East Yorkshire Acute Hospitals NHS Trust, Castle Hill Hospital, Castle Road, Cottingham, East Yorkshire, HU16 5JQ

Vice President: Dr Martin Wiselka (Leicester)
Infection & Tropical Medicine, Leicester Royal Infirmary, Infirmary Square, Leicester, LE1 5WW

Honorary Secretary: Dr Albert Mifsud (London)
Department of Microbiology, Whippers Cross University Hospital, Barts Health, London, E11 1NR

Honorary Treasurer: Dr Stephen Barrett (Essex)
Department of Microbiology, Southend University Hospital, Prittlewell Chase, Westcliff on Sea, Essex, SS0 0RY

Meeting Secretary: Professor Steve Green (Sheffield)
Department of Infectious Diseases & Tropical Medicine, Royal Hallamshire Hospital, Glossop Road, Sheffield, S10 2JE

Membership Secretary: Dr David Partridge (Sheffield)
Department of Microbiology, Northern General Hospital, North Lane, Sheffield, S5 7AU

Clinical Services Secretary (Infectious Diseases): Dr Bridget Atkins (Oxford)
Microbiology & Infectious Diseases, Level 6, John Radcliffe Hospital, Headley Way, Headington, Oxford, OX9 9DU

Clinical Services Secretary (Microbiology): Dr Tony Elston (Colchester)
Department of Microbiology, Colchester General Hospital, Turner Road, Colchester, Essex, CO4 5JR

Guidelines Secretary: Dr Peter Cowling (Scunthorpe)
Department of Microbiology, Northern Lincolnshire & Goole Hospitals NHS Trust, Scunthorpe General Hospital, Cliff Gardens, Scunthorpe, South Humberside, DN15 7BH

Communications Secretary: Dr Khumara Dharmasena (Walsall)
Department of Microbiology, Walsall Hospitals NHS Trust, Manor Hospital, Moat Road, Walsall, WS2 9PS

Manpower & Training Secretary: Dr Albert Mifsud (London)
Department of Microbiology, Whippers Cross University Hospital, Barts Health, London, E11 1NR

Scientific & Research Secretary: Dr Martin Llewelyn & Prof Melanie Newport (Brighton)
Medical School Research Building, Rm 108, Brighton & Sussex Medical School, BN1 9PS

Training Grade Member (Professional Affairs): Dr Thushan De Silva (Sheffield)
Department of Infection & Tropical Medicine, Royal Hallamshire Hospital, Glossop Road, Sheffield, S10 2JF

Training Grade Member (Meetings): Dr Fiona McGill (Liverpool)
Brain Infections UK, Liverpool Brain Infections Group, Institute of Infection & Global Health, University of Liverpool, L69 3BX

Editor Journal of Infection: Professor Robert Read (Sheffield)
Academic Unit of Infection and Immunity, Division of Genomic Medicine, University of Sheffield Medical School, Beech Hill Road, Sheffield, S10 2RX

Newsletter Editor: Dr Paul Collini (Sheffield)
Department of Infection & Immunity, University of Sheffield, Western Bank, Sheffield, S10 2TN

Associate Members Secretary: To be announced
Enquiries for Journal subscriptions, payments and change of membership details

Please contact Anne Taylor, BIA Administrator at:

Hartley Taylor Medical Communications Ltd
Suite GC, Caledonian House
Tatton Street
Knutsford
Cheshire,
WA16 6AG

Mobile: +44 (0) 7578 599902
Tel: +44 (0) 1565 621967
Email: BIA@hartleytaylor.co.uk
Website: www.britishinfection.org

Data Protection

All membership details (as supplied by you) are stored on an electronic database. This database is used for legitimate BIA business only. We occasionally co-operate with other societies and organisations whose objectives are consistent with those of the Society by including material in BIA mailings. If you do not wish to receive such mailings please notify BIA Administration at the above address.
08-45 to 09-10  Registration, coffee/tea & poster viewing

09-10 to 09-15  Welcome
Dr Peter Moss (Hull), President of the BA

09-15 to 10-30  Free Scientific Papers (12 minutes each)

Chairs & discussants -  Professor Richard Bellamy (Middlesborough) & Dr Tristan Clarke (Southampton)

1. Mitochondria damage during herpes simplex virus encephalitis. Malgorzata Wnek et al. University of Liverpool


3. HIV-1 reduces macrophage apoptosis-associated killing of Streptococcus pneumonia. Paul Collini et al. University of Sheffield

4. Arsenic, antimony and Leishmania - has arsenic contamination of drinking water in India led to treatment resistant kala-azar? Meghan Perry et al. University of Dundee

5. Elucidating hospital norovirus transmission using whole genome sequencing. Tse Hua Nicholas Wong et al. NIHR BRC, Oxford


10-30 to 10-50  Coffee/tea & poster viewing

10-50 to 11-50  Free Scientific Papers (12 minutes each)

Chairs & discussants -  Dr Bridget Atkins (Oxford) & Professor Goura Kudesia (Sheffield)

7. Reduced LPS-induced TNFo release by THP-1 cells depleted of mitochondrial DNA. John Widdrington et al. Newcastle upon Tyne Hospitals NHS Foundation Trust

8. In treatment of Pseudomonas aeruginosa with piperacillin tazobactam, addition of low dose gentamicin markedly reduces emergence of resistance and increases antibacterial effect. Alexandra Cochrane et al. University of Bristol


<table>
<thead>
<tr>
<th>Time</th>
<th>Event</th>
<th>Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>11-50 to 12-20</td>
<td><strong>UK State of the Art Lecture 1</strong></td>
<td><strong>Professor Sarah Rowland-Jones</strong>, Professor of Immunology, Honorary Consultant in Infectious Diseases, Nuffield Department of Medicine, NDM Research Building, University of Oxford, UK</td>
</tr>
<tr>
<td></td>
<td>Emerging issues in paediatric HIV: adolescent survivors of vertical infection and exposed uninfected children in Africa</td>
<td>Chair &amp; discussant - <strong>Professor David Dockrell (Sheffield)</strong></td>
</tr>
<tr>
<td>12-20 to 12-45</td>
<td><strong>British Infection Association AGM</strong></td>
<td>Dr Peter Moss (Hull), Dr Martin Wiselka (Leicester), Dr Stephen Barrett (Southend), Professor Steve Green (Sheffield)</td>
</tr>
<tr>
<td>12-45 to 13-30</td>
<td>Lunch &amp; poster viewing</td>
<td></td>
</tr>
<tr>
<td>13-30 to 14-10</td>
<td><strong>Free Scientific Papers</strong> (12 minutes each)</td>
<td>Chairs &amp; discussants - <strong>Professor Tom Evans (Glasgow) &amp; Dr David Partridge (Sheffield)</strong></td>
</tr>
<tr>
<td></td>
<td>12. Prediction of outcome from adult bacterial meningitis in a high HIV seroprevalence, resource-poor setting using a new severity scoring tool, the Malawi Adult Meningitis Score (MAMS). Emma Wall et al. Malawi-Liverpool-Wellcome Trust clinical research programme, Blantyre, Malawi</td>
<td></td>
</tr>
<tr>
<td>14-10 to 15-05</td>
<td><strong>International Keynote Lecture</strong></td>
<td><strong>Professor Salim S. Abdool Karim</strong>, Pro Vice-Chancellor (Research), University of KwaZulu-Natal, Republic of South Africa</td>
</tr>
<tr>
<td></td>
<td>Integrating HIV and TB treatment: challenges and opportunities</td>
<td>Chair &amp; discussant - <strong>Professor David Goldberg (Glasgow)</strong></td>
</tr>
<tr>
<td>15-05 to 15-24</td>
<td>Coffee/tea &amp; poster viewing</td>
<td></td>
</tr>
<tr>
<td>15-24 to 16-00</td>
<td><strong>Free Scientific Papers</strong> (12 minutes each)</td>
<td>Chairs &amp; discussants - <strong>Dr David Partridge (Sheffield) &amp; Professor Tom Evans (Glasgow)</strong></td>
</tr>
<tr>
<td></td>
<td>15. Abdominal tuberculosis and vitamin D status: effect of age, sex &amp; ethnicity. Catherine Cosgrove et al. St George’s Hospital, London</td>
<td></td>
</tr>
<tr>
<td></td>
<td>16. TLDA assay saves the day: a baffling case of relapsing myopathy. Suzanne English et al. Addenbrooke’s Hospital, Cambridge</td>
<td></td>
</tr>
</tbody>
</table>
16-00 to 16-30  **UK State of the Art Lecture 2**  
**Ms Karen Wilson**, Regulatory Policy Manager, Care Quality Commission (CQC), UK  
CQC's new regulatory and inspection model: how we will assess infection prevention and control and infectious disease management

Chair & discussant - **Professor Steve Green (Sheffield)**

16-30 to 16-35  **Comfort break & poster viewing**

16-35 to 17-35  **Clinical Papers (10 minutes each)**

Chairs & discussants - **Dr James Dunbar (Northallerton, Teeside) & Dr Claire Donnelly (Belfast)**

A. Butterflies in the tummy? **Helen Winslow** et al. Royal Liverpool University Hospital

B. A tale of two Darlington heliophiles. **Thomas Lavender** et al. Newcastle upon Tyne Hospitals

C. A furry tale: fever in a child with short gut syndrome. **Alison Burgess** et al. Bristol Microbiology Laboratory

D. Oops, not again! **Dami Collier** et al. University College London Hospital

E. Children are better than doctors when screening for macroscopic haematuria in a high prevalence area for *Schistosoma haematobium*. **Anand Odedra** et al.

F. All that is gold does not glitter. **Firas Maghrabi** et al. Royal Victoria Infirmary, Newcastle

17-30 to 17-35  **Close of proceedings**

BIA Meetings Secretary

BIA Scientific Secretary

Dr Peter Moss, President of the BIA

---

**Selected Poster Presentations:**

01  Development of an assay to detect mutations in the IL-28B and ITPA genes associated with response to hepatitis C virus treatment. **Gemma Quinney**. Empath, Leicester

02  *Elizabethkingia meningoseptica*: The role of MALDI-TOF analysis in strain typing in an outbreak situation. **Luke Bedford** et al. Addenbrookes Hospital, Cambridge

03  Microbiological characteristics of acute osteoarticular infections in children. **Clark Russell** et al. Royal Hospital for Sick Children, Edinburgh

04  Penicillin allergy documentation; making rash decisions. **Alasdair Munro** et al. Poole Hospital NHS Foundation Trust

05  The diagnostic challenges of malaria: current perceptions and practices in the Tanga region of Tanzania. **Celia Jackson** et al. London School of Hygiene and Tropical Medicine

06  Efficacy and safety profile of daptomycin in treatment of skin and soft tissue infections (SSTIs) in clinical practice: retrospective analysis from the European Cubicin® Outcomes Registry and Experience (EU-CORESM). **Mike Allen** et al. Novartis Pharmaceuticals Ltd
07 Adjunctive rifampicin may improve outcomes in *Staphylococcus aureus* bacteraemia: a systematic review. Aaron Lawson McLean et al. Queen Mary University of London

08 Outpatient antibiotic treatment in primary care: a survey of primary care doctors. James Meiring et al. Sheffield

09 Abdominal tuberculosis in HIV positive patients: demographic and diagnostic data from a London tertiary referral centre. Catherine Cosgrove et al. St George’s Hospital, London

10 An assessment of the impact of education, strengthening of referral pathways and multidisciplinary team working on delays in diagnosis and treatment commencement in abdominal tuberculosis. Aula Abbara et al. Ealing Hospital, London

11 United Kingdom and Republic of Ireland renal physicians’ experiences of patients undergoing renal transplants abroad: a questionnaire-based cross-sectional survey. Anand Odedra et al. Sheffield Teaching Hospitals, Sheffield

12 Correlating microscopic and clinical findings in patients with pulmonary aspergillosis. Clark Russell et al. Royal Hospital for Sick Children, Edinburgh

13 An unusual case of brain abscess. Hoi Ping Mok et al. Addenbrookes Hospital, Cambridge

14 The trouble with *Treponema*: an unusual intracerebral pathogen diagnosed by 16s PCR. Luke Bedford et al. Addenbrookes Hospital, Cambridge

15 When indolent becomes interesting - a case of abdominal actinomycosis. Judith Johnston et al. Southend University Hospital
Professor Sarah Rowland-Jones  
University of Oxford, UK

Emerging issues in paediatric HIV: adolescent survivors of vertical infection and exposed uninfected children in Africa  
ABSTRACT HERE

Professor Salim S. Abdool Karim  
University of KwaZulu-Natal, Republic of South Africa

Integrating HIV and TB treatment: challenges and opportunities  
ABSTRACT HERE

Ms Karen Wilson  
Care Quality Commission (CRC), UK

CQCs new regulatory and inspection control model: how we will assess infection prevention and control and infectious disease management  
ABSTRACT HERE
Title: Mitochondria damage during herpes simplex virus encephalitis

Authors: Malgorzata Wnek1, Zarini Ismail1, Emanuele Ricci1, Lorenzo Ressel1, Patrick F. Chinnery2, Anja Kipar1,4, Tom Solomon1,2, Michael J. Griffiths1,3

Addresses: 1University of Liverpool, UK, 2The Walton Centre NHS Foundation Trust, Liverpool, UK, 3Alder-Hey Children’s NHS Foundation Trust, Liverpool, UK, 4University of Zurich, Switzerland, 5Newcastle University, Newcastle upon Tyne, UK

Abstract

Introduction:
Herpes simplex virus (HSV) encephalitis is a devastating disease with high morbidity and mortality despite available antiviral treatment. Better understanding of HSV encephalitis pathogenesis may guide development of more effective treatments.

Methods:
Post-mortem brain tissues from HSV encephalitis and control (road traffic accident) cases, together with an in vitro human astrocyte model for HSV infection, were studied to identify key pathogenic mechanisms. Directed by the results, minocycline, a mitochondria-protective drug, was tested in vitro.

Results:
RNA microarray analysis of brain tissue demonstrated 287 host transcripts with significantly lower abundance in encephalitis compared to control cases, amongst which mitochondrial DNA-encoded transcripts were significantly over-represented. Immuno-staining of the encephalitic brain tissue revealed a reduced staining for cytochrome c oxidase subunit 1 (CO1), a mitochondrial-encoded protein, in areas of high staining for HSV. Supporting the post-mortem findings, abundance for CO1 transcript decreased as HSV DNA abundance increased during in vitro infection, and mitochondrial dysfunction and damage occurred prior to nuclear damage. Minocycline-treated astrocytes showed significantly higher CO1 transcript abundance, lower HSV DNA abundance and sustained mitochondrial enzyme activity compared to non-treated.

Discussion:
We observed a highly preferential loss of mitochondrial DNA-encoded transcripts and protein in brain tissue of HSV encephalitis patients. Mitochondrial dysfunction and structural damage occur before nuclear damage and cell death, suggesting it is a critical and early event during HSV infection. Minocycline preserves mitochondrial function and impairs HSV replication. Routinely used as an antibiotic, minocycline may offer a valuable new adjunctive treatment for HSV encephalitis.
Title PARV4 in South Africa: evidence for vertical transmission of a novel parvovirus

Authors Philippa Matthews1, Colin Sharp1, Amna Malik1, Philip Goulder1,2, Pieter Jooste4, Peter Simmonds3, Paul Klenerman1,2

Addresses 1University of Oxford, UK, 2Oxford University Hospitals NHS Trust, UK, 3University of Edinburgh, UK, 4Kimberley Hospital, South Africa

Abstract

Introduction:
PARV4 is a parvovirus, first identified in 2005. In Western cohorts, it is found almost exclusively in association with other blood-borne viruses. In African cohorts, 30-50% of individuals are seroprevalent, irrespective of risk factors for, or the presence of, other blood-borne viruses. Clinical impact remains uncertain; one study shows an association with increased rate of progression to AIDS, and others have suggested respiratory, gastrointestinal and CNS infections in children. In this study, we set out to investigate the epidemiology of PARV4 in South Africa, in the presence and absence of HIV.

Methods:
We tested plasma for PARV4 IgG by ELISA in 96 individuals recruited from Kimberley hospital, South Africa, as follows: 40 HIV-positive children, 32 of their mothers (also HIV-positive), and 24 HIV-negative siblings.

Results:
In total, 45/96 of individuals were PARV4 IgG positive (46.9%). There was a strong concordance between mother and child serostatus (p=0.006); strikingly, 100% of IgG-positive children had an IgG-positive mother. However, there was also an increase in PARV4 seroprevalence with age in children. PARV4 IgG positivity was not enriched among HIV-positive children, nor was it associated with any alteration in CD4+ T cell percentage.

Discussion:
These are the first data to suggest mother/child concordance in PARV4 serostatus, suggesting possible mother-to-child transmission, in combination with other infection events throughout childhood. In this small cohort, we did not identify a link between PARV4 status and HIV disease progression. This study paves the way for future investigations into transmission routes and clinical significance of this virus.
BRITISH INFECTION ASSOCIATION

Free Scientific Paper 3

Title HIV-1 reduces macrophage apoptosis-associated killing of Streptococcus pneumoniae

Authors Paul Collini, David Dockrell

Address University of Sheffield, UK

Abstract

Invasive pneumococcal disease (IPD) incidence is high in HIV-seropositive individuals (HIV+ve) even with antiretroviral therapy (ART). Alveolar macrophages (AM) phagocytose Streptococcus pneumoniae (Spn) in the lung and a programme of macrophage apoptosis-associated killing is essential for bacterial clearance. HIV-1 infection is associated with macrophage resistance to apoptosis. We hypothesised that apoptosis-associated Spn killing is altered during HIV infection.

Human monocyte-derived macrophages (MDM) were infected with HIV-1_BaL (HMDM) or treated with gp120 (gpMDM) and compared with sham infected/medium alone controls after challenge with opsonised Spn serotype 2 or mock infection. Apoptosis (increases in caspase 3/7 and nuclear condensation/fragmentation), Mcl-1 expression, mitochondrial superoxide and bacterial killing were measured. Additionally, healthy, non-smoker, HIV+ve on ART (HAM) and matched, HIV-ve control volunteers underwent bronchoalveolar-lavage (BAL). AM and lymphocytes were harvested and AM challenged with Spn.

4h after Spn challenge, there was no difference in numbers of internalised viable bacteria in HMDM, HAM or gpMDM vs. controls. At 16-20h caspase 3/7 and nuclear condensation rates were significantly lower for HMDM, gpMDM and HAM vs. controls. Bacterial killing was significantly lower in HMDM and gpMDM vs. controls. Mcl-1 fell and mitochondrial superoxide increased post Spn in control but not HMDM. HIV+ve ART had detectable HIV-1 p24 in AM, a BAL fluid CD8 lymphocytosis and reversed CD4:CD8 ratio which correlated with AM apoptosis.

HIV-1 is associated with reduced macrophage apoptosis-associated killing of pneumococci. A CD8 lymphocytosis is present in the lungs of HIV+ve despite suppressive ART. These may contribute to IPD risk in HIV+ve.
Title: Arsenic, antimony and Leishmania - has arsenic contamination of drinking water in India led to treatment resistant kala-azar?

Authors: Meghan Perry¹, Susan Wyllie¹, Vijay Prajapati², Joris Menten³, Andrea Raab², Joerg Feldmann², Dipankar Chakraborti³, Shyam Sundar³, Albert Picado⁶, Marleen Boelaert⁴, Alan Fairlamb¹

Addresses: ¹University of Dundee, UK, ²University of Aberdeen, UK, ³Benares Hindu University, Uttar Pradesh, India, ⁴Institute of Tropical Medicine, Antwerp, Belgium, ⁵Jadavpur University, Calcutta, India, ⁶Center for International Health Research, Barcelona, Spain

Abstract

In Bihar state, India, the cure rate of antimonial compounds (Pentostam) in the treatment of visceral leishmaniasis (VL) has declined from over 85% to less than 50%. This has been attributed to prolonged, widespread misuse of antimonials within the Indian private healthcare system. An alternative resistance hypothesis is that exposure to arsenic in drinking water in this region has resulted in antimony-resistant Leishmania parasites. Leishmania donovani were serially passaged in mice exposed to environmentally-relevant levels of arsenic in drinking water. Arsenic accumulation in organs of these mice was proportional to exposure. After five passages, isolated parasites were refractory to Pentostam in drug sensitivity assays. Treatment of infected mice with Pentostam confirmed that these parasites retained resistance in vivo, supporting this hypothesis. A retrospective field study on a cohort of antimony treated VL patients was performed in an arsenic contaminated area of Bihar to evaluate the presence of an increased risk of treatment failure and death in those exposed to arsenic. It demonstrated a significant increased risk of death from VL in arsenic exposed patients but did not indicate a significant relationship between arsenic exposure and antimonial treatment failure. Collectively these data suggest that it is biochemically possible that arsenic contamination may have contributed to the development of antimonial resistance in Bihar although issues of underpower and the retrospective nature of our epidemiological study made it difficult to conclusively demonstrate this. Further research into the relationships between arsenic exposure and antimonial treatment failure and death in the leishmaniases is warranted.
Elucidating hospital norovirus transmission using whole genome sequencing

Tse Hua Nicholas Wong, Kate E. Dingle, Derrick W. Crook, David H. Wyllie, Tim E.A. Peto


Abstract

Background: Noroviruses are the causative agents of the majority of gastroenteritis outbreaks worldwide, representing a considerable public health burden. Whole genome sequencing (WGS) and bioinformatics have already become powerful microbiological tools in understanding the transmission of many bacterial organisms. We adapt this technology to elucidate the transmission and evolutionary dynamics of Norovirus in hospital outbreaks.

Methods: Full length norovirus genomes were generated from a total of 337 faecal samples collected over a four year period from 11 independent geographical UK locations. Samples were sequenced using the Illumina MiSeq and HiSeq sequencers. Epidemiological information was also collected to compliment these samples. Replicates were re-sequenced to confirm reliability of both the sequencing and analytical processes.

Results: The whole genome sequences show that outbreaks within wards and hospitals may share identical genomes. Re-introduction of divergent genomes (>100 SNVs) are seen within wards which may prolong the duration of outbreaks. Sequence diversity is large between samples over time and geographical locations; indicating local reservoirs as possible sources of evolution. Over the last season, the emergence of a new recombinant strain is seen rapidly replacing the older variant.

Discussion: Whole genome sequencing can be a realistic platform for detecting noroviruses during hospital outbreaks. The approach facilitates the rapid determination of complete norovirus genomes. This can be deployed rapidly within a hospital laboratory to detect the emergence of a new variant within a short space of time, and can provide the healthcare professional with an early warning system for hospital outbreaks.
Title: Multiple pathways of anti-mycobacterial immune dysregulation by HIV-1 revealed in an in vivo human challenge model

Authors: Lucy Bell¹, Gillian Tomlinson¹, Gabriele Pollara¹, Mellissa Pascoe², Keertan Dheda², Mahdad Noursadeghi¹

Addresses: ¹Division of Infection & Immunity, University College London, UK, ²Lung Infection & Immunity Unit, Department of Medicine, University of Cape Town, South Africa

Abstract

Introduction:
We sought to understand changes in anti-mycobacterial immune responses attributable to HIV-1 infection in an in vivo human experimental challenge model, using genome-wide transcriptional profiling of biopsies at the site of tuberculin skin tests (TSTs).

Methods:
We enrolled 52 HIV-1 negative or positive active TB patients within 4 weeks of commencing anti-TB therapy. TSTs were performed and punch biopsies taken at the site at 48 hours. Biopsy RNA was purified and analysed using microarrays. Normalised gene expression data was analysed via comparative, clustering, bioinformatic and module-driven approaches.

Results:
Gene expression profiles of positive TST biopsies from HIV-ve patients showed a complex immune response enriched for the IFNγ/IL-12 Th1 axis. HIV+ve patients were stratified into three groups: those with anergic TSTs, those with responsive TSTs, and those classified as undergoing an unmasking immune reconstitution inflammatory syndrome (IRIS). In each group, comparison of gene expression profiles with HIV-ve patients demonstrated distinct pathways of dysregulation. HIV-1 anergy was associated with relative enrichment of the type I interferon response in the absence of an effective type II interferon or inflammatory response. Positive TSTs in HIV+ve individuals had a specific deficit in the immunomodulatory IL-10 response. TSTs from unmasking IRIS patients had an expanded gene expression profile, including enrichment for the Th2 axis.

Conclusions:
We have used a human experimental challenge model to gain insights into the anti-mycobacterial immune response at a systems level, and have shown with molecular resolution diverse ways in which this immune response is dysregulated in vivo in HIV-1 infection.
Title: Reduced LPS-induced TNFα release by THP-1 cells depleted of mitochondrial DNA

Authors: John Widdrington1,2, Aurora Gomez-Duran2, Angela Pyle2, Marie-Helene Ruchaud-Sparagano2, Patrick Chinnery1,2, John Simpson1,2

Addresses: 1Newcastle upon Tyne Hospitals NHS Foundation Trust, Tyne and Wear, UK, 2Newcastle University, Tyne and Wear, UK

Abstract

Introduction:
In sepsis monocyte immune deactivation is associated with increased mortality and susceptibility to secondary infections. There is increasing evidence of depletion of mitochondrial DNA (mtDNA) and impaired mitochondrial respiration in monocytes from septic patients. This study aimed to assess the links between mtDNA depletion and immunity in human monocytic THP-1 cells.

Method:
To selectively deplete their mtDNA THP-1 cells were incubated with 50ng/ml ethidium bromide (EtBr) for 8 weeks. After confirming the effects of EtBr on mitochondria, immune responses were assessed by measuring lipopolysaccharide (LPS)-induced cytokine release and phagocytosis of fluorescent Staphylococcus aureus (SA).

Results:
Incubation with EtBr successfully generated THP-1 cells lacking mtDNA (termed ρ0 cells). There was a selective depletion of mtDNA-encoded RNA transcription and protein expression in these cells. In addition ρ0 THP-1 cells had complete loss of activity in mitochondrial respiratory chain complex IV, which contains key mtDNA-encoded subunits. LPS-induced release of the pro-inflammatory cytokine tumour necrosis factor-α (TNFα) was significantly reduced but phagocytosis of SA was increased in ρ0 cells.

Discussion:
THP-1 cells depleted of mtDNA by treatment with EtBr produce significantly less TNF-α in response to LPS, a typical feature of deactivated monocytes in sepsis. However, mitochondrial depletion did not produce a global down-regulation of immune responses and phagocytosis was actually enhanced in ρ0 THP-1 cells. Further investigation into the complex effects of mitochondrial depletion on immunity may provide important insights into immune suppression in sepsis.
In treatment of *Pseudomonas aeruginosa* with piperacillin tazobactam, addition of low dose gentamicin markedly reduces emergence of resistance and increases antibacterial effect

**Authors**
Alexandra Cochrane¹,³, Karen Bowker², Alan Noel², Alasdair MacGowan²,³

**Addresses**
¹University of Bristol, UK, ²Bristol Centre for Antimicrobial Research and Evaluation, UK, ³North Bristol NHS Trust, UK

**Abstract**

**Introduction:**
Treatment of *Pseudomonas aeruginosa* with combination therapy to reduce the emergence of resistance has been proposed, but assessing benefit has been hampered by lack of defined pharmacokinetic/pharmacodynamics (PK/PD) targets for the combinations tested. Here PK/PD targets for combination therapy of piperacillin/tazobactam (P/T) plus gentamicin against *P. aeruginosa* were assessed *in vitro*.

**Method:**
Kill curve methodology and an *in vitro* dynamic pharmacokinetic model were used to assess the antibacterial effect (ABE) of combinations of P/T and gentamicin against a wild type clinical isolate of *P. aeruginosa*. Emergence of resistance EoR to both antimicrobials was assessed.

**Results:**
Time kill demonstrated that where P/T (196mg/l) was used in combination with gentamicin (0.5mg/l), ABE at 24 hours was increased by >2 log(10) compared to the most potent agent alone. In the dynamic models the addition of gentamicin markedly decreased the percentage of time above the MIC over 24 hours (P/T %T>MIC) required to achieve static effect. In models of P/T alone resistance emerged rapidly with 16xMIC resistance present at 24 hours. In the presence of gentamicin no resistance to P/T at 4xMIC or 16xMIC emerged by 72 hours.

**Discussion:**
In treatment of *P. aeruginosa*, it can be difficult to achieve the %T>MIC targets for B-lactams. We show that *in vitro*, addition of gentamicin reduces the target %T>MIC for P/T, and protects against emergence of resistance against P/T. These effects are robust to changes in the gentamicin dosing and occur at levels of gentamicin exposure below that usually considered to be nephrotoxic in humans.
Free Scientific Paper 9

Title A case of triclabendazole resistant *Fasciola hepatica* infection in humans

Authors Charlotte Zheng¹, Charles Fry¹, Laura. E Nabarro¹, Peter. L Chiodini².¹

Addresses ¹University College Hospital, UK, ²The Hospital for Tropical Diseases, London, UK

Abstract

Case description:
A 71 year old man presented with colicky right upper quadrant pain and weight loss. Blood tests revealed an eosinophilia of $3.4 \times 10^9$ /litre and an elevated alkaline phosphatase of 347u/l. Computed tomography of the abdomen showed patchy nodular abnormalities in the liver, initially thought to be metastases. Subsequent liver biopsy revealed a marked eosinophilic infiltrate in the portal tracts.

On further questioning, the patient described eating watercross picked on his local golf course in a sheep rearing area. He denied previous travel history. Stool microscopy revealed ova of *Fasciola* species. Serology was also positive. He was treated with 2 doses of triclabendazole one month apart.

Unfortunately his symptoms continued. He denied any further consumption of wild watercress or travel history. Repeat stool microscopy showed ova of *Fasciola* species. Magnetic resonance imaging of the liver demonstrated small filling defects in the bile duct and duct wall thickening suggestive of persistent fluke infection. He was treated again with two further doses of triclabendazole one month apart. Repeat stool microscopy is pending.

Discussion:
Both ovine and human fascioliasis is thought to be increasing in the UK. In 2012, triclabendazole resistant *Fasciola hepatica* was confirmed in sheep in the UK. This case describes the first case of UK acquired human infection with triclabendazole resistant *Fasciola hepatica*. The case demonstrates the importance of a one health policy promoting collaboration between physicians, veterinarians and environmental agencies in attaining optimal health for all.
Title: Detecting latent TB, HIV, and hepatitis B/C in new migrants in primary care

Authors: Sally Hargreaves¹, Farah Seedat¹, Josip Car², Samia Hasan², Joseph Eliahou¹, Jon Friedland¹

Addresses: ¹Imperial College London, UK, ²Hammersmith and Fulham Centres for Health, Hammersmith Hospital, London, UK

Abstract

Introduction:
Rising rates of IDs has reignited the debate on migrant screening, amid calls to strengthen primary-care-based programmes, focusing on latent TB. We did a cross-sectional study of new migrants (≤ 10 years in the UK) to test a one-stop blood test approach to detect HIV, latent tuberculosis, and hepatitis B/C on registration with a GP, and performed a Systematic Review of screening effectiveness in Europe.

Method:
The cross-sectional study was done across two GPs attached to two hospital A&Es in a high migrant area/West London for 6 months. All new migrants who attended a New Patient Health Check were screened for eligibility and offered the blood test. The systematic review was reported according to PRISMA.

Results:
Foreign-born patients were under-represented in relation to 2011 Census data (p<0.001). Of 1235 new registrations, 453 attended checks, of which 47 (10.4%) were identified as new migrants (mean 32.11 years/time in UK 2.28 years; 22 nationalities). 36 (76.6%) participated. The intervention only raised latent tuberculosis prevalence (18.18% [95% CI 6.98-35.46]; 181.8 cases per 1000). 0 (0%) of 6 went on to receive treatment (3 did not attend referral). The Review highlights multiple approaches, varying in effectiveness (80% uptake; 42% failed to access treatment on diagnosis; 18% failed to complete).

Discussion:
The one-stop approach was feasible in this context. Uptake of screening, when accessible, is high in migrants. Patient numbers were surprisingly low, perhaps a consequence of the recent debate around immigration checks/charging at GP services. A better place to test this approach may be in A&Es.
A 59 year old woman presented to a local hospital with fever and abdominal pain. She had raised inflammatory markers and an eosinophilia. CT scan of the abdomen, performed to exclude cholecystitis, showed a large hepatic cyst extending through the diaphragm and into the chest in keeping with a WHO type 3A cyst. The presentation with sepsis and eosinophilia suggested that the cyst had ruptured and developed superadded bacterial infection. She was initially treated with antibiotics and albendazole and, once stabilised, had a hemi hepatectomy with resection of the diaphragm and partial right lower lobectomy. Histology subsequently confirmed cystic echinococcosis (CE). Hydatid serology was positive.

She improved clinically and received one year of albendazole therapy. She has been followed up for 18 months and has no evidence of recurrence. She will be followed up for 10 years in total to ensure there is no late recurrence.

Of note, this patient was born and bred in the UK. She had not previously travelled to highly endemic areas for CE which suggests that her disease was acquired in the UK.

This case is an example of hepatic CE which had eroded through the diaphragm and into the lung. It highlights that severe extensive CE can be acquired and successfully treated in the UK with the use of a multidisciplinary team.
Title Prediction of outcome from adult bacterial meningitis in a high HIV seroprevalence, resource-poor setting using a new severity scoring tool, the Malawi Adult Meningitis Score (MAMS)

Authors Emma Wall¹², Mavuto Mukaka¹, Matthew Scarborough¹, Katherine Adukievicz⁴, Katherine Cartwright⁵, Brian Faragher², David Laloo², Robert Heyderman¹

Addresses ¹Malawi-Liverpool-Wellcome Trust clinical research programme, Blantyre, Malawi, ²Liverpool School of Tropical Medicine, UK, ³John Radcliffe Hospital, Oxford, UK, ⁴North Manchester General Hospital, UK, ⁵University Hospitals of Leicestershire NHS Trust, UK

Abstract

Bacterial meningitis in sub-Saharan African adults is associated with mortality rates in excess of 50%, is predominately caused by S. pneumoniae, is strongly HIV-associated and occurs primarily in young adults. To improve this poor outcome, a severity prediction tool is required to inform both interventional trials and clinical management, aiming to optimise inter-hospital referrals, duration of antibiotics and hospital stay. However, a previously derived European severity score for bacterial meningitis was poorly predictive when applied to Malawian data.

We utilised individual predictors of death derived from a Malawian clinical trial dataset (n 400) to develop a severity prediction tool that can be applied in a high HIV seroprevalence, resource-poor setting (Malawi Adult Meningitis Score (MAMS)). Five of fifteen variables tested (CSF culture, CSF WCC, Haemoglobin, GCS and pulse rate) were shown to be strongly associated with poor outcome on multivariate analysis, and were converted into a predictive tool using a nomogram. The nomogram was tested against a separate clinical trial data-set for validation (n 193). Concordance of the nomogram in the validation dataset between predicted and actual outcome was 0.74 (95% CI 0.65 : 0.82), agreement 62.5%, Kappa 0.6, with an estimated sensitivity of 75% and specificity of 55%.

MAMS has equivalent power to predict outcome when applied in Malawi as the European meningitis score when applied in Europe. Comparison of the two scores may help us understand the pathophysiological differences and the difference in efficacy of adjunctive therapies such as corticosteroids in these two populations.
BRITISH INFECTION ASSOCIATION
Free Scientific Paper 13

Title
Epidemiology, diagnosis and management of strongyloidiasis in an NHS tertiary referral hospital

Authors
Daniel Greaves, Suzanne English, Elinor Moore

Address
Department of Infectious Diseases, Addenbrooke’s Hospital, Cambridge, UK

Abstract

Introduction:
The prevalence of strongyloidiasis in the UK among returning travellers and migrants is unknown. We sought to investigate the population diagnosed with strongyloidiasis over a 7 year period in a large tertiary referral centre.

Methods:
Retrospective case note review of patients diagnosed with strongyloidiasis from 2007-2013. Data were collected on ethnicity, travel history, method of diagnosis, treatment and outcomes.

Results:
47 patients were diagnosed with strongyloidiasis over the study period. White British returning travellers were the largest single group (49%), followed by migrants from the Indian subcontinent (25%) and Africans (17%). Of note, 18 of the 47 patients (38%) were either immunocompromised or had immunosuppression planned for the future. Strongyloidiasis was diagnosed by positive serology in 45 cases (98%) and microscopy for the remaining 2. Stool microscopy was performed for 30 of the seropositive patients and was negative in all cases. Eosinophilia was the most common reason for investigation (28 patients - 60%) and 33% of patients had symptoms attributable to strongyloidiasis, including one case of hyperinfection. 40 patients (85%) received anti-helminthic treatment: 38 with ivermectin and 2 with albendazole. 23 patients were followed up after treatment, of which 20 showed improvement of symptoms and/or resolution of eosinophilia.

Conclusions:
In areas with low migrant numbers, returning travellers may constitute a large percentage of patients with strongyloidiasis. Clinicians should consider investigations for strongyloidiasis in at-risk patients for whom immunosuppression is planned. Sensitivity of stool microscopy for S. stercoralis larvae outside the reference laboratory setting may be very low.
Title  Pyogenic liver abscess in adults: recent trends in a UK hospital

Authors  Sarah Mycroft, Elizabeth Sheridan, Guduru Gopal Rao

Address  North West London Hospitals NHS Trust, UK

Abstract

Objectives:  To assess epidemiology, aetiology, diagnosis, management and outcomes of patients presenting with pyogenic liver abscess (PLA) at a UK hospital and make recommendations for improving the care pathway.

Methods:  PLA was considered retrospectively over five years (July 2007 to July 2012) at a two-site London DGH. Patient demographics, past medical history, time from admission until the diagnostic scan for the liver abscess and the time to drainage (if performed) were recorded. Laboratory results were reviewed to establish causative organism frequencies.

Results:  56 patients were identified, 64.3% male and 35.7% female. Median age was 65.5 years. The most common ethnicities were white (41.0%) and Indian (25.0%), and comorbidities were biliary pathology (25.0%), diverticular disease (17.8%) and diabetes (17.8%). Median time from admission to diagnostic scan was 3 days, and to drainage 6 days. An organism was found in 60.7% patients. Bacteria from the Streptococcus anginosus group were most prevalent (26.5%), followed by Escherichia coli (17.6%). Klebsiella pneumoniae was found in 11.8%. Mortality was 8.9% during admission, 12.5% at 6 months.

Conclusions:  PLA epidemiology reflected previous UK results in terms of age, gender and comorbidities. Streptococcus anginosus group bacteria were the most frequently identified pathogen with Klebsiella pneumoniae a notable but not remarkable cause as has been seen in Asia, and to a lesser extent the USA. Delays in diagnosis and drainage of PLA were significant which might contribute to the comparatively high mortality of this infectious disease. We advocate raising clinician awareness to identify patients at high risk of PLA, and expediting ultrasound in these cases.
Title: Abdominal tuberculosis and vitamin D status: effect of age, sex & ethnicity

Authors: Jeremy Nayagam, Claire Mullender, Andrew Poullis, Catherine Cosgrove

Address: St George’s Hospital, London, UK

Abstract

Introduction:
The emergence of vitamin D beyond its role in bone health, as an immune regulator in inflammatory bowel disease (IBD) has become of great interest. The interest in the role of vitamin D in other chronic inflammatory conditions is growing, and supplementation of vitamin D is now routine practice in the treatment of pulmonary tuberculosis (TB). We sought to identify the vitamin D levels of patients with abdominal TB (ATB).

Methods:
A retrospective review of patients treated at St George’s Hospital, London, for ATB from June 2003 to August 2013 was conducted. Information was gained from electronic patient records and the hospital’s tuberculosis database. Vitamin D levels were classified as deficient (25-50nmol/L), or severe deficiency (<25nmol/L).

Results:
65 cases of ATB were identified. Mean age was 42 years (range 18-97). 49.2% were females. Vitamin D levels were measured in 52 patients (80%), with an average of 23.1nmol/L (range undetectable-102). 35 (67.3%) had severe deficiency, 15 (28.8%) had moderate deficiency. There was no difference between vitamin D levels between ages (≤50:>50 mean 22.3nmol/L v 25.6nmol/L, p=0.59), sexes (M:F mean 19.8nmol/L v 26nmol/L, p=0.25) or ethnicities (BME:Caucasian mean 22.0nmol/L:41.7nmol/L, p=0.08).

Conclusions:
Vitamin D deficiency is common and often severe in patients treated for ATB in London, across a range of demographics and phenotypes of disease. Further work needs to be carried out to identify if vitamin D deficiency plays a pathological role in ATB and if supplementation improves treatment outcomes, however in the interim, testing and treatment appears to be advisable.
Title  TLDA assay saves the day: a baffling case of relapsing myopathy

Authors  Suzanne English¹, Dominic O’Donovan², Martin Curran¹, Ruchi Arora¹, Robert Fincham², Hamid Jalal¹, Andrew Dean², Hongyi Zhang¹

Addresses  ¹Department of Virology, Addenbrooke’s Hospital, Cambridge, UK, ²Department of Histopathology, Addenbrooke’s Hospital, Cambridge, UK, ³Department of Paediatrics, Jenny Lind Children’s Hospital, Norwich, UK

Abstract

Persistent parvovirus B19 infection has been reported as an uncommon cause of fatigue and arthralgia in adults, and acute B19 infection has also been associated with rare cases of rhabdomyolysis or vasculitis in both children and adults. Here, we present the first case of an otherwise healthy nine-year-old child with evidence of persistent parvovirus B19 infection associated with a relapsing myopathy and recurrent urinary symptoms over five years, as well as an acute episode of severe rhabdomyolysis at age eight. On admission for acute rhabdomyolysis (creatine kinase 520 064 U/L, plasma lactate 2.9mmol/L), serology was positive for parvovirus B19 IgG, but negative for IgM. However, electron microscopy (EM) of a skeletal (thigh) muscle biopsy taken 8 months after admission demonstrated small, intracellular virions, approximately 20 to 30nm in diameter, contained within endosomes, 88 to 92nm in diameter, in the capillary endothelium. Parvovirus B19 was positively identified by customized Taqman low-density array (TLDA) assay. Furthermore, preservation of surrounding myocyte architecture on EM, in association with localization of intracellular B19 virions within the capillary endothelium, was consistent with virus-mediated small vessel injury.
Title: Association between cytomegalovirus seropositivity and cardiovascular disease among immunocompetent adults without previous history of cardiovascular disease

Authors: Mohsin Ali¹, Eddy Malouf², Effrossyni Gkrania-Klotsas³,⁴

Addresses: ¹Institute of Public Health, University of Cambridge, UK, ²School of Medicine and Medical Science, University College Dublin, Ireland, ³Infectious Diseases Unit, Addenbrooke’s Hospital, Cambridge, UK, ⁴School of Clinical Medicine, University of Cambridge, UK

Abstract

Introduction: Established risk factors for cardiovascular disease (CVD) do not fully explain CVD risk. Cytomegalovirus (CMV) has been linked to development of atherosclerotic CVD among transplant recipients, but its association with CVD in immunocompetent adults without previous history of CVD is unknown.

Methods: Four databases (Embase®, Medline®, Scopus®, and Web of Science®) were systematically searched in March 2014. Titles, abstracts and full-text articles were screened using a priori criteria. Included studies were critically appraised and synthesised, emphasising findings of methodologically robust studies.

Results: Of 1,163 titles screened, eight prospective studies met criteria for synthesis. All included studies defined CMV exposure using serological tests, but varied substantially regarding potential bias, confounding, and chance. Among them, one study was considered most robust, in part due to its high degree of adjustment for potential confounders and markedly large study sample size (n=12,574). Six studies reported a null association with CMV seropositivity. However, three studies indicated a modest increased risk in CVD among participants with higher titres, with the most robust study reporting an adjusted hazard ratio (95% CI) of 1.21 (1.04-1.41) for participants in the highest tertile of titres versus seronegative subjects.

Conclusions: There appears to be a modest increased risk in CVD associated with higher CMV antibody levels. Given high burden of CVD and high seroprevalence of CMV among adults worldwide, this association is of potential public health relevance. Further research examining this association in other cohorts, and prospective studies correlating CMV antibody levels with direct measurements of active infection are necessary.
Abstract

A 44 year old company director presented in March 2014 with a two week history of central abdominal pain and worsening diarrhoea. At its worst, he was opening his bowels 20 times a day. He didn't report any fever, vomiting or other focal symptoms.

He did not take any regular medication and did not have any known allergies. His past history and family history were unremarkable.

His travel history included a weekend break to Provence, France 4 weeks before presentation. Within the 18 months prior to presentation he had also travelled to Madrid, coastal Morocco and Austin, Texas.

He was initially reviewed by his GP and blood tests showed a normal haemoglobin and fractionally raised white cell count at 11.3 x 10^9/L with raised eosinophils at 2.6 x 10^9/L. CRP was less than 5 mg/L. Renal and liver function tests were normal. Stool culture and microscopy for ova, cysts and parasites were negative.
Title  A tale of two Darlington heliophiles

Authors  Thomas Lavender, Helen Brocklehurst, Uli Schwab

Address  Newcastle upon Tyne Hospitals, UK

Abstract

Patient 1, a 61 year-old male intermittently resident in Spain, with a history of Burkitt’s lymphoma in 2010 and severe idiopathic thrombocytopenia purpura with splenectomy in 2012, presented with severe thrombocytopenia unresponsive to corticosteroids. Blood film confirmed the diagnosis.

Treatment was complicated by pancreatitis on day 5 (amylase 591 unit/L), and was switched to liposomal amphotericin B 3mg/kg for 14 days, then weekly for 6 weeks. Platelet count returned to baseline within 2 weeks of commencing therapy. Bone marrow aspirate at 3 months showed no evidence of recurrence.

Patient 2, also 61 years old, presented with a slowly progressive, ulcerated lesion affecting the right side of the face 3 months after returning from Ibiza, unresponsive to corticosteroids or antibiotics; biopsy confirmed the diagnosis. Intravenous treatment was commenced due to the severity of the lesion and proximity to the eye. At day 17 ECG showed significant QTc prolongation (600ms) with U waves consistent with severe hypokalaemia (2.3mmol/L).

These cases highlight the need to consider this diagnosis in patients returning from the Mediterranean and the complications of treatment; liposomal amphotericin B is as efficacious but less toxic. The blood film is rarely diagnostic; the absence of a spleen in patient 1 caused the parasite to spill over.
Title  A furry tale: fever in a child with short gut syndrome

Authors  Alison Burgess¹, Martin Williams¹, Chris Linton²

Addresses  ¹Public Health England, Bristol Microbiology Laboratory, UK, ²UK National Mycology Reference Laboratory, Bristol, UK

Abstract

A 6-month old boy, born at 25 weeks gestation, was receiving total parenteral nutrition (TPN) via a tunnelled central venous catheter for short gut syndrome following bowel resection for volvulus.

He became pyrexial (38.8 °C) with no clear source of infection. His initial full blood count and CRP were normal. Blood cultures were sent from the central venous catheter. A presumed diagnosis of necrotising enterocolitis (NEC) was made and amoxicillin, gentamicin and metronidazole commenced.

Despite broad-spectrum antibiotic therapy, he remained pyrexial, developed neutropaenia (nadir 0.22x10⁹/L) and thrombocytopaenia (nadir 29x10⁹/L) with increased CRP (29mg/L). Abdominal radiograph and ultrasound scan did not demonstrate acute pathology. Blood cultures remained negative.

The haematology team contacted the ward having noted an unusual appearance in the blood film...
A 49 year old Caucasian homosexual man (MSM) was diagnosed with HIV in 1997. He had a baseline CD4 count of 360 cells/mL and an HIV viral load of 28 000 copies/ml. He was found to be hepatitis B virus (HBV) immune and HCV negative. He commenced antiretroviral therapy (ART) with Atripla (a combination of tenofovir, emtricitabine and efavirenz) in October 2011 with a nadir CD4 of 310 cells/mL and HIV RNA of 98 000 copies/mL and achieved viral suppression within 12 weeks. Three years after commencing ARTs, routine blood tests revealed deranged liver enzymes with an alanine aminotransferrase of 57 IU/L. A detailed social history revealed that he was part of the hardcore, high-risk sex scene in London where he had recently been involved in slamming- a culture of injecting crystal methamphetamine and mephedrone to get a bigger rush. He had also shared needles and used drugs mixed with the blood of other MSMs. Two weeks later his ALT was 32 IU/L and he became lost to follow-up. He presented for a sexual health screen 3 month later with progressive jaundice over a 2 week period with a bilirubin of 350 mmol/L and his ALT of 3 400 IU/L. His clotting was also marginally abnormal. So he was transferred to a tertiary hepatology centre with a plan for a liver biopsy and further investigation.
BRITISH INFECTION ASSOCIATION
Cryptic Clinical Case E

Title
Children are better than doctors when screening for macroscopic haematuria in a high prevalence area for Schistosoma haematobium

Authors
Anand Odedra, Cristina Bocanegra, Anna Chirra, Ho kwong Li, Clair Lindsay, Lairumbe Silangei, Geremy Opoki, Regis Boillet, Jenny Smith, Oliver Smith

Address
London School of Hygiene and Tropical Medicine, London, UK

Abstract

Introduction:
Our study compared the abilities of a group of foreign doctors to detect haematuria (haematuria being 95% sensitive for Schistosoma haematobium amongst children in a high prevalence area) compared to a group of local children in Pemba Island, Tanzania.

Methods:
We visited a community with a high prevalence for schistosomiasis and asked 18 children to complete a questionnaire verbally using an interpreter. Each child was asked to provide a urine sample which was subsequently examined by two clinicians, who were blinded to the results of the questionnaire, using the urine colour chart, a result of 4 or above was said to be positive. Each sample was subjected to urinalysis by a different blinded clinician and a result of trace or above was noted as positive for haematuria. Microscopic haematuria acting as our gold standard.

Results:
13 (72%) children reported macroscopic haematuria using the urine colour chart. 13 (72%) children tested positive on urinalysis, however 2 children who reported haematuria were urinalysis negative and vice versa. Only 2 of the 13 children who had haematuria on urinalysis had macroscopic haematuria visualised by a clinician. All children with observed macroscopic haematuria had confirmed haematuria on urinalysis.

Conclusion:
Our study suggests that a group of children aged 5 to 18 in a high prevalence area for Schistosomiasis haematobium are better at detecting macroscopic haematuria than a group of foreign doctors. Furthermore the urine colour chart appears to be a simple, cheap and useful screening tool for macroscopic haematuria and therefore Schistosomiasis haematobium.
BRITISH INFECTION ASSOCIATION

Cryptic Clinical Case  F

Title All that is gold does not glitter

Authors Firas Maghrabi¹, Uli Schwab¹, Christopher Duncan¹

Addresses ¹Department of Infection and Tropical Medicine, Royal Victoria Infirmary, Newcastle, UK, ²Newcastle University, UK

Abstract

We report a case of a twenty year old man, who was previously fit and well. He was brought into the emergency department after an emergency response call. His mother reported that he was behaving in a strange manner that day. He had left the taps in the kitchen and shower open, and was drinking from a soft drink can that was closed, and later, he had developed slurring of speech and was incontinent of faeces. The patient’s mother gave a three day history of her son feeling generally unwell, with fatigue, muscle aches, reduced oral intake and a sore throat.

On initial assessment he was pyrexial at 40°C, with sinus tachycardia, BP was 127/49 and a respiratory rate of 30. On examination he had neck stiffness, photophobia and a non blanching petechial rash over his chest and lower limbs.

His oropharynx was inflamed with dry mucous membranes. His chest and heart sounds were normal. He had bilateral renal angle tenderness. Bloods showed Hb 157, WCC 9.2, and platelets 39. Na 130, K 3.5, U 7.1, Cr 150. Bilirubin 52, albumin 42, ALT 53, CRP 245. PT 15.3, fibrinogen 5.8. Lactate was 2.8.

A lumber puncture was performed and showed a protein count of 0.68 g/L. Glucose of 3.7. WCC of 175 with 60% polymorphs. No organisms were seen on gram stain.
British Infection Association

Delegates
British Infection Association

Delegates
British Infection Association

Delegates