



University Hospitals Birmingham 
NHS Foundation Trust

CENTRAL QUALITY ASSURANCE SCHEME

**Haematology Update Meeting Birmingham
Wednesday 11th May 2011**

10.05-10.50

**Dr. David Bareford
Russells Hall Hospital, Dudley**

Morphology Session - Shape Matters

Modern FBC analysers can give red cell size down to a tenth of a femtolitre and can indicate the range of cell sizes present but give no indication of cell shape. For this reason there is still a valuable place for the examination of blood films by haematocytologists that helps give information towards establishing a diagnosis. This presentation will explore the various red cell shape changes that can be seen and how they can help in diagnosis.

10.50-11.35

**Mr. Phillip Hurley
Birmingham Heartlands Hospital**

Point of Care testing in Pathology-where are we now and the impact of new CPA standards

Point of Care testing is undergoing the biggest changes in its history of existence, with drivers at both National and Local level. This presentation will look at these drivers for change and examine what they actual mean to delivery of service.

The Heart of England NHS Foundation Trust is exploring some novel ways of moving forward with our POCT service and identifying new ways of funding these. This presentation will hopefully make you think of the ways that POCT can be improved within you own Trust.

No Trust as yet opted for a CPA assessment under the new standards, why is this? Should we fear or embrace the new standards? This presentation will give my views on what you should achieve with a POCT service as a minimum before applying for an inspection.

11.35-12.15

Dr. Joost van Veen
Royal Hallamshire Hospital, Sheffield

Heparin Induced Thrombocytopenia

Heparin induced thrombocytopenia (HIT) is a clinicopathological syndrome caused by the formation of IgG antibodies against platelet factor 4 and heparin. The antigens form multimolecular clusters binding IgG molecules, forming large immune complexes. Crosslinking with platelet FcγIIa receptors on platelets causes coagulation activation, thrombin generation and thrombocytopenia due to intravascular activation and release of procoagulant platelet derived microparticles. The antibodies also cause endothelial changes and activation of monocytes and neutrophils. Clinically HIT presents with falling platelet counts with or without venous/arterial thrombosis or skin changes typically within 5 – 15 days after heparin exposure.

Testing to demonstrate the presence of HIT antibodies is most commonly performed using immunoassays. Although these assays have a good sensitivity their usefulness is limited by a poor positive predictive value of only 30 – 50% commonly causing false positive results. It is therefore recommended not to perform antibody testing at low clinical pre-test probability and only to use the assays at intermediate or high clinical probability for HIT. Specificity is improved by using IgG specific assays but false positive results remain common. In the interpretation the optical density (OD) of the test should be taken into account whereby at OD less than 1.0 the antibodies are not likely to be able to activate platelets. Although the

relationship between optical density and the presence of platelet activating antibodies appears to be established, the OD when HIT becomes more likely is dependent on the assay used and should ideally be established locally. The value of confirmatory procedures using high heparin concentrations in these tests remains to be established although there is some evidence suggesting that they may be of value in positive tests with low OD.

HIT can be excluded with a low clinical pre-test probability or negative immunoassay. IgG specific assays improve the specificity but still have a low positive predictive value and significant risk of over diagnosis. HIT is unlikely at positive tests with a low OD and whilst there may be a role for confirmatory steps in this group, it does not exclude the diagnosis. Whereas HIT is likely at high OD the diagnosis remains uncertain in those with intermediate OD and ideally needs confirmation with a functional assay.

12.15-12.30

Karen Morris
University Hospital Birmingham

Can we measure VWF Activity?

In recognition of the importance of von Willebrand factor (vWF) testing in the diagnosis of von Willebrand disease (vWD), the Central Quality Assurance Scheme regularly distributes samples for determination of vWF:activity.

Data from 12 separate surveys (three samples within each) performed between 2008 and 2010 have been reviewed (36 samples total). The average number of results returned for each sample was 18.

During the period of the surveys four methods have been continuously used. The most popular being platelet aggregation, using a combination of 'in house' methods, kits & reference materials, followed by an automated latex immunoturbidometric assay.

In 2008 these two methodologies made up 73% of results returned which has increased to 88% in 2010. Other methods employed include ELISA and slide agglutination.

Poor agreement is seen across all methods as indicated by the coefficient of variations produced (CVs). The Overall CV of results for each sample ranged from 13.92 to 39.21. The CV ranges for the two major method groups were 10.38- 43.04 (Plt Ag) and 2.01- 14.00 (Automated latex immunoturbidometric assays).

Whilst platelet aggregation remains the 'Gold Standard' the calculation of the results can be subjective.

The automated assay gives more precise but higher results.

14.00-14.40

**Dr. Mark Cook
University Hospital Birmingham**

Plasma Cell Disorders and the Kidney

Myeloma is a devastating haematological malignancy affecting approximately 5/100,000 people annually. Life expectancy is between 3 and 7 years from diagnosis. Approximately 10% of patients present with severe renal impairment often requiring dialysis. These patients have a life expectancy of less than 1 year. Recently, changes in anti-myeloma therapy have improved the outlook for patients with myeloma. Some of the newer agents and novel approaches to dialysis are changing the outlook for myeloma patients with renal impairment. In this presentation, I will outline the nature of renal impairment in plasma cell disorders- focusing on myeloma. The consequences and modern approaches to treatment, including current trial data will also be discussed.

14.40-15.25

Dr. Will Lester
University Hospital Birmingham

Antiphospholipid Syndrome

Despite over 30 years of research and international guidance on diagnosis, Antiphospholipid syndrome remains enigmatic. However recent research has given us some fascinating clues into the underlying pathophysiology. The clinical and laboratory diagnosis of Antiphospholipid syndrome will be explored using real clinical cases. Laboratory testing in particular remains a controversial area with a poor rate of concordance between laboratories, highlighted by the recent CQAS returns.

References

Wilson WA et al. International consensus statement on preliminary classification criteria for definite antiphospholipid syndrome: report of an international workshop. *Arthritis Rheum.* 1999 Jul;42(7):1309-11

Miyakis S, et al. International consensus statement on an update of the classification criteria for definite antiphospholipid syndrome (APS). *J Thromb Haemost.* 2006 Feb;4(2):295-306

Pengo V, et al. Subcommittee on Lupus Anticoagulant/Antiphospholipid Antibody of the Scientific and Standardisation Committee of the International Society on Thrombosis and Haemostasis. Update of the guidelines for lupus anticoagulant detection.

Subcommittee on Lupus Anticoagulant/Antiphospholipid Antibody of the Scientific and Standardisation Committee of the International Society on Thrombosis and Haemostasis. *J Thromb Haemost.* 2009 Oct;7(10):1737-40

