Spring
2006

Clinipath Pathology Newsletter

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- Haematology of Pregnancy
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CLINIPATH PATHOLOGY

"we take it personally"

From the CEO

Welcome to the spring edition of the Clinipath Pathology newsletter. The style and format has been modified and this newsletter includes two scientific articles, general news about Clinipath Pathology and a questionnaire.

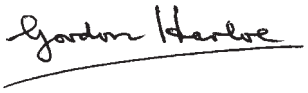
Part of our commitment to you and your patients is to ensure customer service and provide you with access to specialist pathologists. I am delighted to announce that Dr Tiffany Hughes and Dr Michael Watson have recently joined Clinipath Pathology and they are profiled in this newsletter. Tiffany has extensive expertise in immunology and has been appointed head of this department. Michael has specialized in a number of disciplines including being dually qualified as an Infectious Disease Physician and Clinical Microbiologist. Tiffany and Michael would welcome your calls and clinical queries.

As part of our continuous improvement philosophy we have recently installed a new telephone exchange system, added extra staff in our results and enquiries department and modified our internal communications process to facilitate receiving your calls. Mr Ian McPhan, our Chief Operating Officer, has written an article on our communications team and the service they provide.

Our new collection centres are listed, and in particular please note our expanded service with the opening of a collection centre at Halls Head, Mandurah. The Mandurah collection centre establishes a diagnostic service corridor in the South West, linking our Clinipath Pathology and Bunbury Pathology laboratories.

To ensure that we provide you with clinically relevant information, we have included a questionnaire with suggested future topics. We realize that you are pressed for time but we would value your input so that we can provide articles that are of interest to you.

Please fax your response to Louise Campbell on 08 9322 9338 by Monday 14th November to be in the draw to win a fantastic weekend for two at the Bunker Bay Resort.



Gordon Harloe
Chief Executive Officer

Microbiology

Dr Michael Watson MBBS, FRACP, FRCPA, MPH&TM

Dr Michael Watson graduated in medicine from the University of Western Australia in 1988. He completed his fellowship in Paediatrics and Infectious Diseases at British Columbia's Children's hospital in Vancouver, Canada in 1994 and was admitted to the Fellowship of the Royal Australasian College of Physicians in 1995. He then returned to Western Australia and trained in clinical microbiology at Royal Perth, King Edward and Princess Margaret Hospitals. He completed his Fellowship of the Royal College of Pathologists of Australasia (FRCPA) in 1997.

He has subsequently completed a Masters in Public Health and Tropical Medicine from James Cook University in 2001 whilst working as an infectious disease physician and clinical microbiologist at the Children's Hospital at Westmead in Sydney. His main area of research interest has been in the epidemiology of infectious diseases and in particular pneumococcal disease. In 1996, he established the Vaccine Impact Surveillance Network in Western Australia and, in 1999, the NSW Pneumococcal Reference Laboratory.

He has most recently been working at St John of God Pathology as an infectious disease physician and clinical microbiologist and currently has a clinical private practice in general infectious diseases at Hollywood Private Hospital. His areas of special interest in clinical medicine include recurrent staphylococcal infections in all age groups (including community acquired MRSA), orthopaedic infections and paediatric infectious diseases. Dr Watson lectures medical students in clinical microbiology at Notre Dame University.

Immunology

Dr Tiffany Hughes
MBBS, FRACP, FRCPA
Immunology

Dr Tiffany Hughes graduated from University of Adelaide in 1993. Her early post graduate years were spent in Ascot, Berkshire, and at the John Radcliffe and Churchill Hospitals, Oxford, followed by immunology training at Flinders Medical Centre (Adelaide), Royal Perth Hospital and Sir Charles Gairdner Hospital.

Her practice now incorporates all aspects of adult and paediatric clinical immunology and immunopathology. She has particular interest in allergy and autoimmune disease.

Dr Hughes is heavily involved in undergraduate and postgraduate teaching and is the FRCPA representative on the Joint Specialist Advisory Committee (JSAC) for immunology.

She is currently the WA representative for the Australian Society of Clinical Immunology and Allergy, and has been a board member of the Asthma and Allergy Research Institute since 2002.

Escape to Bunker Bay

For your chance to win a fantastic weekend for two at the beautiful Bunker Bay Resort simply complete our enclosed questionnaire.

Gorgeous Bunker Bay lies facing north and this stunning five star resort features 35 acres of landscaped grounds with 150 Luxury Villas. Stroll to the glorious white sands and azure waters of Bunker and Eagle Bay, situated on the dazzling Indian Ocean.

Non melanoma skin cancer – the basics

Skin tumours are frequently seen and removed in general practice. This article highlights variants of common skin tumours that may be associated with an increased risk of local recurrence and / or metastasis following removal, as well as less-common aggressive tumours that may be primarily excised.

Basal cell carcinoma

Basal cell carcinoma is the most common malignant skin tumour. There are several variants that are associated with an increased risk of local recurrence owing to their relatively less-well circumscribed outlines. The risk of local recurrence of these variants depends on the clearance as measured histologically. The reported risk of local recurrence related to the subtype and distance from the surgical margin is shown in Table 1.

Basal cell carcinoma rarely metastasises and then usually in large, neglected tumours.

Table 1

BCC type	< 0.38mm	0.38-0.75mm	> 0.75mm
Nodular (Solid)	40%	10%	4%
Superficial multifocal, Sclerosing, Infiltrative (Micronodular)	80%	45%	20%

- The significance of a close margin varies depending on the subtype of BCC.

  Dixon AY et al J Cutan Pathol 1993; 20: 137-42

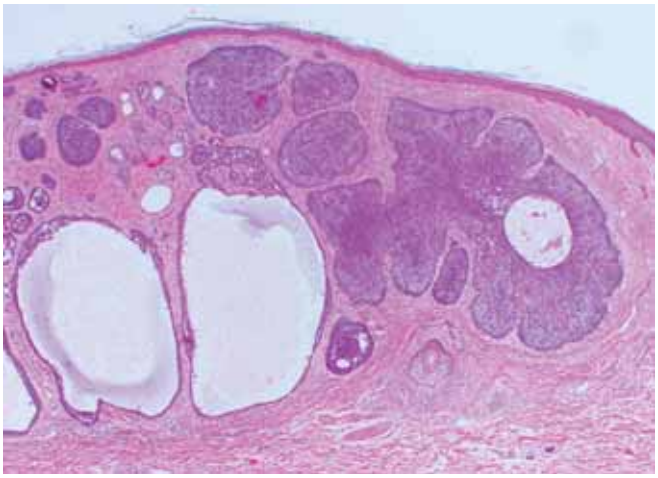


Fig.1 Nodulocystic Basal Cell Carcinoma

continued overleaf

Non melanoma skin cancer – the basics

Squamous cell carcinoma

Squamous cell carcinoma is associated with a risk of metastasis as well as local recurrence. Recurrence is more common in lesions on the lip and ear, in acantholytic, desmoplastic and poorly differentiated tumours, in tumours that show perineural invasion, and those in which the surgical margin is narrow. The risk of metastasis depends on the type of squamous cell carcinoma, the situation in which the tumour arises and the tumour thickness (Tables 2 and 3).

Acantholytic SCC is characterised by discohesion of the malignant cells that results in a pseudoglandular appearance. Tumours with this appearance have a slightly increased risk of local recurrence and metastasis.

Desmoplastic SCC is an aggressive variant characterised by a prominent desmoplastic stromal reaction and an increased risk of metastasis.

Verrucous carcinoma is a very well-differentiated squamous cell carcinoma that most commonly arises on the skin of the feet, the anogenital region and in the upper aerodigestive tract. It is invasive and can be locally destructive but rarely metastasises.

Table 2. Squamous cell carcinoma metastasis

- **Metastasis varies with the subtype and the situation in which the tumour develops**

1. Verrcous carcinoma: rarely metastasizes
2. SCC in sun-damaged skin (0.5%)
3. SCC in non sun damaged skin (2-3%)
4. SCC in Bowen's disease (2-5%)
5. Acantholytic SCC (2-19%)
6. Lip (2-16%)
7. External ear (10%)
8. Desmoplastic SCC (20%)
9. SCC in chronic ulcer (10-30%)
10. Vulva, perineum, penis (30-80%)

- **Usually regional lymph node(s) initially**
- **Systemic metastasis preterminal**

Table 3. Squamous cell carcinoma metastasis

- **The risk of metastasis in cutaneous SCC varies with the tumour thickness. The following has been reported:**

	SCC nos.	SCC desmoplastic
≤2mm	0%	0%
2-5 mm	3%	20%
>5 mm	15%	45%

Breuninger H et al Cancer 1997 79:915-9

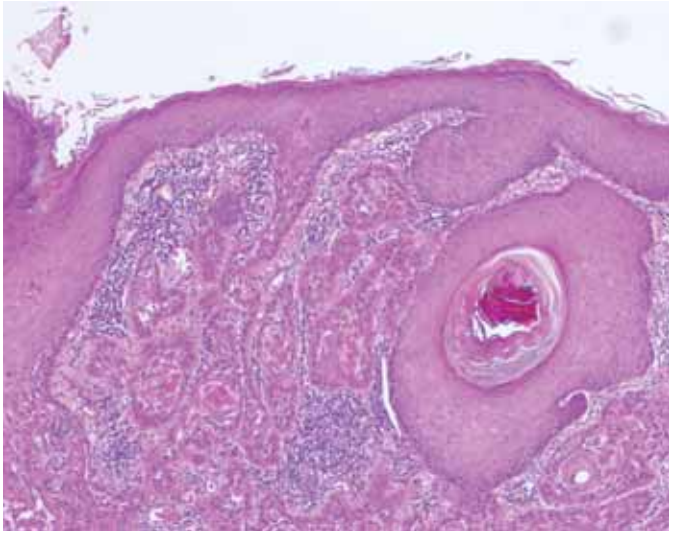


Fig. 2 Squamous Cell Carcinoma

Keratoacanthoma

Keratoacanthoma is a self-limiting well-differentiated squamoproliferative lesion that typically grows rapidly to form a nodule containing a keratin plug. These lesions can be locally destructive before involution necessitating treatment. One variant occurs following local trauma, including tumour excision, and may suggest tumour recurrence clinically. Other variants include keratoacanthoma centrifugum marginatum that presents as an expanding annular lesion with central clearing; multiple keratoacanthomas that can occasionally be a marker for Muir-Torre syndrome; and subungual keratoacanthomas which are frequently more destructive than SCC in this site.

There has been a tendency in the USA to consider these lesions as variants of SCC, but the clinical behaviour is sufficiently distinctive to warrant retention of this diagnosis. Unlike SCC, perineural and lymphovascular invasion are of no prognostic significance in KA.

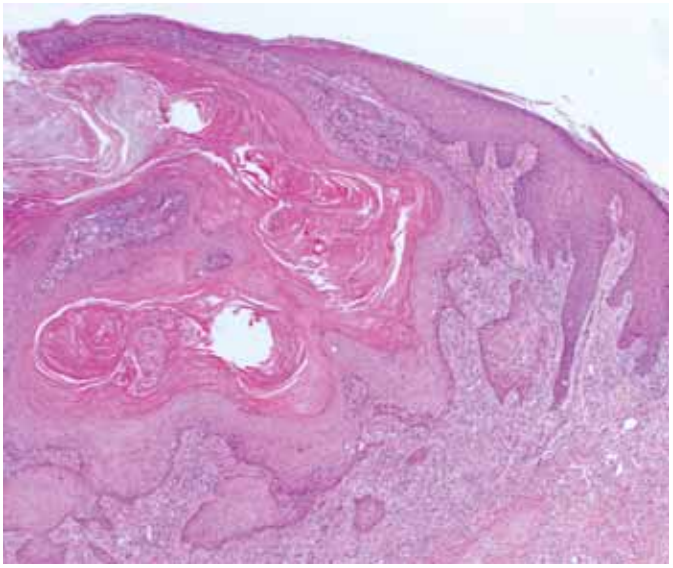


Fig. 3 Keratoacanthoma

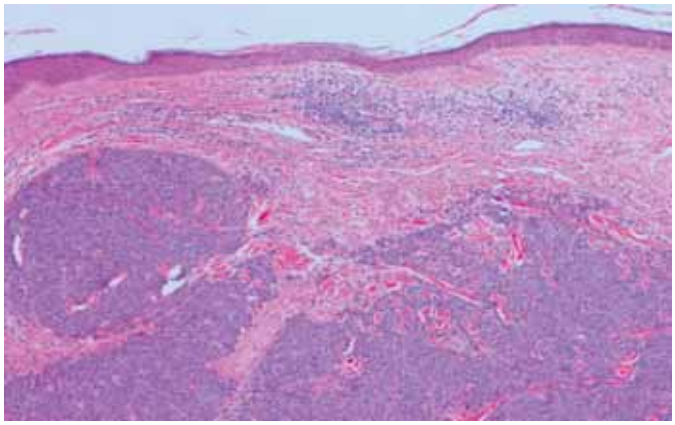


Fig. 4 Merkel Cell Carcinoma

Merkel cell carcinoma

This is a highly aggressive clinically non-distinctive tumour that arises in the sun-damaged skin of the elderly. It is a cutaneous neuroendocrine carcinoma and has a high risk of lymph node metastasis (up to 75%) and significant tumour-associated mortality (35%). Recent evidence suggests that the best results are obtained with multimodal treatment including wide local excision, early radiotherapy and, in advanced cases, chemotherapy.

Dermatofibroma

Dermatofibroma is a common lesion that is usually of only cosmetic significance. Dermatofibromas are not infrequently thought to be cysts clinically whilst the aneurysmal form is often rapidly growing and may simulate a melanocytic or vascular tumour. The recurrence rate depends on the subtype with the usual type of DF recurring in less than 2% of cases. A higher recurrence rate is seen in the following types: aneurysmal (approx. 25%); atypical (25%); cellular and deep penetrating (25%); facial lesions (15%), and subcutaneous forms (30%).

Metastasis of dermatofibroma is a very rare occurrence and has been seen only with the more aggressive subtypes noted above. The usual type of DF is a biologically benign lesion.

Atypical fibroxanthoma

This is an uncommon lesion that occurs on the sun-damaged skin of the elderly. The clinical appearance is often of an ulcerated nodule. When strict criteria are used to make the diagnosis the risk of recurrence is low following local excision and metastasis is very rare.

Other cutaneous carcinomas

There are a seemingly bewildering array of adnexal carcinomas that vary in their propensity for both local recurrence and metastasis. These tumours show differentiation towards adnexal epithelium (hair follicle, sebaceous, apocrine and eccrine).

Sebaceous carcinomas are most common around the eyes and diagnosis is not infrequently delayed as they may mimic a chalazion or chronic conjunctivitis like other sebaceous tumours they can occur in Muir Torre Syndrome. These tumours are prone to local recurrence and there is a 20% tumour-associated mortality. In general, eccrine and apocrine carcinomas are locally invasive, necessitating wide local excision. Perineural invasion is particularly common with some eccrine carcinomas. The metastatic risk varies from negligible (e.g. microcystic adnexal carcinoma / sclerosing sweat duct tumour) to high (e.g. porocarcinoma, ductal type carcinomas). Some of the tumours can be subclassified as low and high grade, with corresponding differences in metastatic potential (e.g. hidradenocarcinoma). Information regarding this will usually be provided in the histology report.

Metastatic carcinoma in the skin either may be the first manifestation of an occult tumour or occur in the setting of known disease. The differentiation from a primary cutaneous tumour can be difficult in some cases and may require correlation with the clinical and radiological findings. Providing the history, when known, is extremely helpful.

Superficial sarcomas

Various sarcomas can occur in the skin and subcutaneous tissue. The diagnosis is made histologically by identifying the direction of tumour cell differentiation. These tumours usually have infiltrative margins and therefore a propensity for local recurrence. Myxofibrosarcoma ("myxoid MFH"), the commonest superficial sarcoma arising in the extremities of elderly patients, is often unexpectedly extensive at the time of diagnosis and therefore at particular risk for local recurrence unless widely excised.

The risk of metastasis of superficial sarcomas depends on the tumour type (e.g. epithelioid sarcoma ultimately metastasises in approx. 50%, whilst dermato-fibrosarcoma protuberans does so in only approx. 3%); the grade of the tumour in some cases (e.g. myxofibrosarcoma: superficial grade 1 lesions do not metastasise, whereas superficial grade 2 and 3 tumours do so in 25% of cases); and the depth at which the tumour arises. In general, sarcomas confined to the dermis have a very low risk of metastasis, whilst similar tumours arising primarily in the subcutaneous fat tend to be more aggressive. For leiomyosarcoma the risk is < 10% for dermal and 30% for subcutaneous primaries.

Cutaneous lymphoma

Cutaneous lymphoid infiltrates can resemble rashes, cysts or tumours and are often unsuspected clinically. The differentiation of reactive lymphoid hyperplasia from lymphoma can be difficult and frequently involves the use of ancillary techniques such as immunostaining, flow cytometry and studies for clonality (gene rearrangement testing). Once the diagnosis has been established, classification of cutaneous lymphoma involves ancillary testing, correlation with the clinical findings and staging.

Further reading: Weedon D. Skin Pathology (2nd Ed)
Churchill Livingstone 2002.

Dr Gordon Harloe
Anatomical Pathologist
T: (08) 9471 5279
E: gharloe@clinipath.net

Adapted from the article written by Dr Richard Williamson published by our affiliated laboratory, Sullivan and Nicolaides in Synopsis: Issue 34: December 2004

Haematology of Pregnancy

Blood volume changes in pregnancy:

Both red cell mass and plasma volume increase during pregnancy. Since the increase in red cell mass is around 25%, well below the plasma volume increase of 40% (up to 55% in multiple pregnancies) the haemoglobin level falls, particularly between weeks 24 and 32 of pregnancy. This decrease in haemoglobin level occurs even when iron and folate stores are adequate, but is accentuated by deficiencies. The consequent decrease in blood viscosity facilitates placental perfusion. Women who do not exhibit a fall in haemoglobin during a pregnancy have a high incidence of complications such as pre-eclampsia and stillbirth, according to the Swedish Medical Birth Register [1].

Although 110g/l is commonly accepted as the lower limit of normal haemoglobin in pregnancy there are many patients in whom the haemoglobin falls as low as 95g/l without any deleterious effects or evidence of iron or folate deficiency.

Folate status at conception and in pregnancy:

It is now widely accepted that folate deficiency at conception is strongly correlated with neural tube defects. It is strongly recommended that all women take folate supplementation for 3 months prior to conception and throughout pregnancy. Women with a family history of neural tube defect, with diabetes or on anti-epileptiform drugs constitute a high risk group and require a daily 5 mg folate dose. For standard risk women the recommended folate supplement is 0.5mg daily. It should be noted that the standard Fefol tablet only contains 0.3mg of folate, well short of the recommended pre-conception intake.

In addition, since intolerance to the iron in Fefol may hinder compliance, it makes sense to prescribe folate alone initially, switching to a combined iron/folate preparation only if the serum ferritin is reduced.

Folate and Vitamin B12 metabolism in pregnancy:

A slight increase in mean red cell volume (MCV) may occur in normal pregnancy. However, an increase in MCV to over 105fl (given a normal range of 80-100fl) warrants assessment of red cell and serum folate and serum Vitamin B12 levels. Neutrophil hypersegmentation is not a reliable indicator of megaloblastic anaemia in pregnancy due to the tendency for a left shift to occur.

Decreased folate intake due to such causes as vomiting in pregnancy and malabsorption, combined with increased requirements at pregnancy, make folate deficiency a much more common cause of megaloblastic anaemia than Vitamin B12 deficiency. A fall in the inactive transcobalamin I during pregnancy results in a decrease in Vitamin B12 levels which does not accurately reflect either the active transcobalamin II or body stores, which on average last for 2 years. In fact the B12 levels may fall to 100pmol/l or even below without any functional disturbance of B12 metabolism. When the patient has a macrocytic anaemia, a balanced diet, normal folate levels but reduced Vitamin B12, it is worth checking serum homocysteine and urine methyimalonic acid (MMA) levels and anti-parietal cell and anti-intrinsic factor antibodies before giving empiric parenteral Vitamin B12. If genuine Vitamin B12 deficiency is confirmed investigation can then be undertaken at leisure once breast feeding has been completed.

Iron deficiency in pregnancy:

Iron deficiency accounts for 75% of anaemias of pregnancy. The markedly raised red cell mass, together with the requirements of the placenta and foetus necessitate a considerable increase in recommended daily iron intake of around 30mg daily, compared with 15mg daily in the non pregnant state. A serum ferritin estimation at the initial antenatal visit will help to determine whether iron supplementation will be needed and whether this should be taken as Fefol or equivalent or whether iron containing multivitamin preparations will suffice.

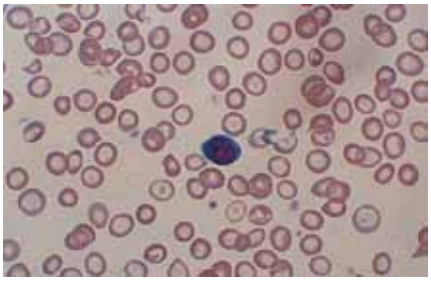


Fig.1 Iron deficient anaemia.

In the second half of pregnancy symptomatic iron deficiency, developing in the setting of intolerance to oral iron can be effectively treated with intravenous total dose infusion (TDI). These infusions are safe and effective but need to be given by experienced staff adhering to a strict protocol.

Additional important causes of a microcytic blood film (MCV<8Ofl) are thalassemias/haemoglobinopathies. As thalassemia trait may be masked by iron deficiency, it is important to check not only Hb electrophoresis and HbH bodies but also serum ferritin. This also avoids the unnecessary administration of iron supplementation to patients whose primary problem is not iron deficiency but thalassemia. Checking the partner is important once a diagnosis of thalassemia trait/haemoglobinopathy is confirmed so that genetic advice can be given promptly if appropriate.

Leucocytes in pregnancy:

The normal range of the leucocyte count increases with pregnancy, the upper limit extending from 11.0/nl to 15.0/nl. This increase is largely due to a neutrophilia, which may be accompanied by “toxic” granulation of neutrophils, and left shift with band forms, metamyelocytes and myelocytes. If any symptoms are present, infection, particularly of the urinary tract, needs to be considered. A more extreme left shift with the occasional promyelocyte and myeloblast is occasionally found in a normal pregnancy. The absence of symptoms, normal examination findings and normal platelet count together with serial blood films should allow exclusion of serious disease such as acute leukaemia.

Platelets in pregnancy:

In up to 10% of pregnancies mild thrombocytopenia supervenes after week 20. The platelet count rarely fails below 75/nl and pregnancy proceeds uneventfully. There is typically a history of similar mild thrombocytopenia with previous pregnancies but normal platelet count between pregnancies. For this reason the term gestational thrombocytopenia is usually applied. The baby's platelet count at birth is normal. Platelet antibodies are rarely found, but it remains likely that the transient thrombocytopenia is of autoimmune aetiology.

An autoimmune mechanism also accounts for most cases of severe isolated thrombocytopenia in pregnancy. Immune thrombocytopenia (ITP) which accounts for 5-10% of thrombocytopenias in pregnancy is important as the severity of the thrombocytopenia often posing major problems for pregnancy, delivery and for the baby. Systemic lupus erythematosis (SLE), the antiphospholipid syndrome (APS) and pre-eclampsia and its (HELLP) syndrome variant also need to be excluded. Platelet-specific antibodies are found in over 50% of patients with immune thrombocytopenia. However the presence or absence of antibodies does not correlate with severity. Bone marrow examination is usually noncontributory.

Typically, the platelet count falls, sometimes profoundly, as pregnancy progresses. In severe acute ITP high dose corticosteroid therapy and intravenous immunoglobulin may be required. In the most extreme cases splenectomy may be unavoidable, but can only be performed with acceptable safety to mother and foetus during the second trimester. In the milder acute ITP and in chronic ITP it may be possible to delay treatment until the last few weeks of pregnancy providing there is no active bleeding and the platelet count remains above 25/nl. In the last few weeks of pregnancy however, treatment aimed at increasing the platelet count to 75-100/nl is needed since this enables delivery and epidural anaesthesia to be performed safely. In maternal ITP, particularly in splenectomized patients there is a significant risk of severe neonatal thrombocytopenia. This holds true even when the maternal count is normal or near normal at the time of delivery. In ITP, the mode of delivery should be determined by obstetric indications.

When the thrombocytopenia is associated with pregnancy-specific causes such as pre-eclampsia, HELLP syndrome and acute fatty liver it is important to perform a coagulation profile including D-dimers to exclude DIC. Thrombotic thrombocytopenic purpura/haemolytic uraemic syndrome (TTP/HUS) is an important diagnosis since the disease complex is life threatening but responds rapidly to early intensive plasma exchange therapy. The red cell appearances on the peripheral blood film in TTP are often characteristic, with variable numbers of red cell fragments.

Myeloproliferative disorders (MPD) and other haematological malignancies occur occasionally in pregnancy. Essential thrombocythemia (ET) is the most common of these. Despite the potential hazards of thrombosis and haemorrhage it is usually possible to avoid exposure to myelosuppressive agents during pregnancy.

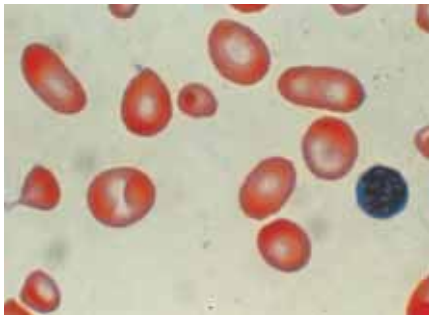


Fig. 2 Megaloblastic anaemia

Thrombosis and haemostasis in pregnancy:

The incidence of venous thromboembolism (VTE) increases three-fold in late pregnancy and ten-fold in the immediate post partum period. This risk reflects a variety of pregnancy induced changes in the haemostatic mechanism including an increase in fibrinogen and Factor VIII, the development of acquired resistance to activated protein C, a reduction in the antithrombotic factor protein S, and reduced fibrinolytic activity. In pregnancy the ESR reflects the hyperfibrinogenaemia and loses its usefulness as a marker for systemic disease.

Inherited thrombophilias, whether due to genetic mutations such as factor V Leiden and prothrombin, gene mutation or deficiencies of protein C, S and anti-thrombin (AT), increase the risk, not only of VTE in pregnancy and the post-partum period but also of recurrent foetal loss. However, the increase in risk is not considered sufficient to warrant funding of thrombophilia screening for recurrent foetal loss under the Medical Benefits Schedule. Anti-cardiolipin antibodies and lupus anticoagulant, particularly when present at high titre, markedly increase the risk of foetal loss and maternal VTE, and may warrant the use of prophylactic low molecular weight heparin (LMWH) during pregnancy and the puerperium. Patients with homozygous or multiple thrombophilic defects are also at greatly increased risk, and likewise merit LMWH prophylaxis.

If therapeutic anticoagulation is required during pregnancy LMW heparin is the drug of choice for most conditions. In view of the risk of bleeding complicating epidural anaesthesia it is mandatory to obtain expert advice concerning the timetabling of heparin dosage particularly LMW heparin.

Von Willebrand's disease is the most common inherited bleeding disorder, mild (type 1) disease accounting for 90% of cases. In pregnancy, extending through to the immediate post partum period, there is usually a temporary normalisation of the disease, measurable as a return of Factor VIII complex levels and platelet function studies to normal. A check early in the third trimester revealing that these parameters are all within the normal range clears the way for epidural anaesthesia to be performed safely if required. However, a history of PPH in a previous pregnancy may override these considerations and mandate the prophylactic use of specific therapy such as DDAVP (Minirin) or Factor VIII concentrate, depending on the severity of the von Willebrand's disease.

Dr Andrew Barr
Consultant Haematologist
T: (08) 9467 5222
M: 0419 901 329

Reference: [1] Stephansson, 0, et al. Maternal haemoglobin concentration during pregnancy and risk of stillbirth. JAMA. 2000;284:2611.

A note from our Microbiology Dept.

1. Swab for Microscopy, Culture and Sensitivity (Bacterial)

Collection:
Blue top swab with transport medium. Record the site of collection on swab container and request form.

Storage:
Store specimen at room temperature.

Note: Specimen collected in transport medium (Blue top) cannot be used for PCR.

2. Swab for PCR testing

Collection:
Orange top wire swab (dry) in empty sheath. Record the site of collection on swab container and request form.

Storage:
Store specimen at room temperature.

Common tests requested include:
N.gonorrhoea, Chlamydia, Ureaplasma/Mycoplasma Herpes I/II, Influenza, Adenovirus, Enterovirus, Bordetella.

Note: Orange top (dry) swabs are unsuitable for microscopy, culture and sensitivity.

To ensure that we can provide you with a fast and accurate service, please clearly indicate on the request form the microbiological tests required.

Our new telephone system

Easy pleasant phone access for our referrers

This was the goal we set for our new communications plan. We have recently taken major steps to make this a reality. We undertook an analysis of your feedback and all aspects of our physical platform including equipment and staff. The result was the identification of a number of areas for improvement.

To provide easy pleasant phone access we aim to:

- Have staff pick up the phone within 2 rings
- Have designated scientific staff available in each department
- Have a clear protocol to quickly access Pathologists
- Provide accurate and professional information as quickly as possible
- Avoid transfers from person-to-person
- Have our staff hand over information rather than you having to repeat yourself, if a transfer is required.

To make this a reality we have:

- Purchased a new digital phone system with improved call handling
- Designed and implemented a company wide communication plan with clear roles and responsibilities
- Increased the number of incoming lines – so there are no 'engaged' tones
- Increased the number of results staff and domiciliary bookings staff to prevent 'missed calls' or excessive waiting
- Provide on-going training for staff on phone answering
- Developed professional hold messages and background music
- Implemented monitoring of how well we are doing by using Key Performance Indicators.

This change has been a total rethink and rebuild of how we handle your calls. A rethink that has been in direct response to your feedback.

Your feedback on our service helps us to continue to be responsive to your needs. All feedback is documented, analysed and acted upon. Please keep providing us with the information that enables us to continually improve our support to you and your patients.

For more information contact

Mr Ian McPhan, Chief Operating Officer
Telephone (08) 9476 5221 or imcphan@clinipath.net

New Collection Centre Details

We have recently opened four new collection centres to serve your patients better.

Alexander Heights
44 Greenpark road
9247 2533

Halls Head
68 Mahogany Drive
9586 9168

Midland
(Relocated)
307 Great Eastern Hwy
9250 5011

Mt Claremont
14 Ashton Ave
Mt Claremont
9383 4670

Ocean Reef
81 Marina Blvd
9307 5344

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