2013 1

SA PATHOLOGY Newsletter

INSIDE

For our patients and our population
www.sapathology.sa.gov.au

From the inside

From the Executive Director

MR KEN BARR IS EXECUTIVE DIRECTOR OF SA PATHOLOGY

I AM DELIGHTED to present this edition of the *SA Pathology Newsletter* (formerly the *IMVS Newsletter*). SA Pathology is proud to be the public provider of pathology services within South Australia to ensure all our population, communities and patients have 24/7 access to a comprehensive range of services, irrespective of their income and location.

The priorities and direction contained within our 2013-16 strategic plan outline our clear intention to build on a longstanding record of quality and excellence as a regulated and accredited pathology service across all disciplines and our expanding Point of Care Testing network. Our plans for service, IT and transport improvement, will enable us to meet future challenges and maintain our hard earned reputation.

As part of a highly regarded health system that provides outstanding patient care, integrated research and teaching activities we aim to continually improve the depth and breadth of our services whilst returning value for money to South Australian taxpayers.

As a clinical support service SA Pathology recognises that it needs to positively respond to the changing clinical landscape in order to meet the needs of hospitals, clinicians and the community. We also recognise the need to manage demand and budget pressures, while ensuring our plans are consistent with the major developments that are occurring within the health system, including the new Royal Adelaide Hospital and the South Australian Health and Medical Research Institute (SAHMRI), plus the major research and teaching goals of the three universities.

SA Pathology will be explicitly moving away from the 'one hospital, one laboratory' model as advances in analytical, transport and IT technology provide new opportunities for us to build a more flexible, efficient and effective state-wide pathology network.

Be assured that our services will continue to be clinically led, and that patient safety and improving health outcomes remains our primary goal. We look forward to working with you so that we can continue to provide the very best pathology service to support our patients and our population.

Mr Ken Barr

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by John William Waterhouse 1891
See page 10 for Ulysses syndrome

Complement method change

To improve turnaround times for complement C3 and C4 results, SA Pathology changed the method and analyser platform to the ADVIA 2400 on 26 August 2013. Results for both C3 and C4 using the new method are approximately 10% higher than those of the old method. The reference interval has changed to reflect this shift and a paediatric range is included.

C3	
<4 weeks	0.58 to 1.08 g/L
<3 months	0.67 to 1.24 g/L
<6 months	0.74 to 1.38 g/L
<9 months	0.78 to 1.44 g/L
<10 years	0.80-1.50 g/L
>10 years and adults	0.85-1.60 g/L
C4	0.12-0.36

DGA reference interval change

SA Pathology changed the Deaminated Gliadin Antibody (DGA) method and analyser platform on 9 December 2013. Whilst the new method is clinically identical to the current method the results will be considerably different.

New reference interval

The new reference interval for DGA will be: <11 U/mL.

If you have any questions regarding these changes please contact the Immunology Consultant via SA Pathology Enquiries on (08) 8222 3000

Red Blood Cell Folate (RCF)

SA Pathology has moved from serum folate to Red Blood Cell Folate (RCF) testing as the standard measurement to assess folate nutritional status.

Due to the 120 day average lifespan of the red cell, RCF folate is less susceptible to rapid changes in diet compared with serum folate, and will provide more reliable and accurate results. In practice this change will:

- identify an additional 5% of patients with folate deficiency with spuriously high serum levels
- more accurately reflect the patient's folate status over the preceding 2 to 3 months.

Q: What do I need to do differently?

A: You will need to collect an EDTA specimen (purple top).

Q: Can I specifically request serum folate testing?

A: Yes. Serum folate levels will continue to be available on request.

Q: Can I request tests to differentiate between B12 and folate deficiencies?

A: Yes. Specific tests to identify and differentiate between B12 and folate deficiencies, such as homocysteine and methylmalonic acid levels, will continue to be performed. For B12 and RCF please collect both serum (white top) and whole blood (purple top).

Did you know?
Mandatory folate fortification of flour has been in place since 2009 in Australia.

Clinically related questions

Please contact Professor Luen Bik To, Haematology Clinical Director on (08) 8222 3633 or Dr Penelope Coates, Chemical Pathology Clinical Director on (08) 8222 3391.

Warfarin reversal guidelines

In-patients receiving warfarin and experiencing bleeding is a relatively common event, with approximately 2-3% of such individuals having a major complication each year.

Patients on warfarin will also often have high INR values without clinically evident bleeding. Guidelines for the management of both bleeding and non-bleeding patients with an elevated INR result have been developed by the Australian Society of Haemostasis and Thrombosis.

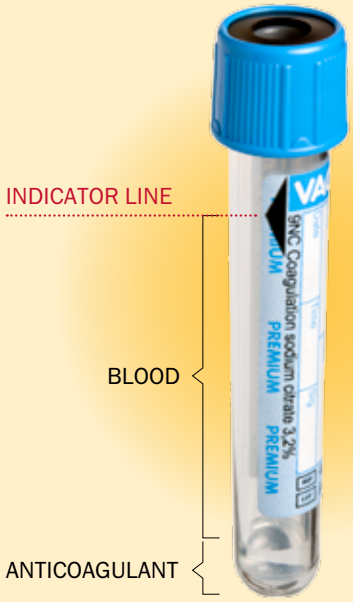
To aid decision making in this patient group the recently updated Warfarin Reversal Guidelines are now available on the SA Pathology internet site. From the Home page go to: For Clinicians | Quick Guide for Clinicians | Emergency Information.

Further advice can be obtained from the duty haematologist on-call through the RAH switch board, phone (08) 8222 4000.

DID YOU KNOW?

Citrate Blood Tubes

When collecting blood into citrate tubes (blue tops) you must fill to the indicator line.



The reason is that the **ratio of blood to anticoagulant is critical** for clotting tests.

An under-filled tube will have too much citrate making results invalid; similarly, overfilling the tube dilutes the citrate concentration invalidating the result.

Collection by vacuum is the method preferred as this takes the correct amount of blood. However if the tubes are old the vacuum may be reduced resulting in under filling; please replace old or defective tubes.

Over or under filled samples will be rejected and a new sample will be required. This applies to all tube sizes.

For additional information please call the Haemostasis Reference Laboratory on (08) 8222 3918.

Significance of ANCA

DR TATJANA BANOVIC – CONSULTANT PATHOLOGIST – IMMUNOLOGY

Anti neutrophil cytoplasmic antibodies (ANCA) are associated with a spectrum of systemic vasculitic conditions affecting small and medium vessels throughout the body.

The syndromes of small vessel vasculitides (SVV) systemic vasculitides are characterised by:

- overlapping clinical and histological features with frequent involvement of major organs
- the need for aggressive immunosuppressive treatment
- serious morbidity and a significant mortality.

The management of these vasculitides often requires critical and timely decision making to prevent the consequences of disease and the hazards of mistreatment. The importance of understanding the tests for ANCA used in the diagnosis cannot be overemphasised.

Screening

The limits of ANCA testing need to be understood. To maximise the predictive value of ANCA screening it should only be performed on appropriately selected patients. The clinical indications of suspected ANCA-associated vasculitis include:

- glomerulonephritis
- pulmonary haemorrhage
- cutaneous vasculitis, especially with systemic features
- multiple lung nodules
- chronic destructive disease of the upper airways; long standing sinusitis or otitis
- subglottic tracheal stenosis
- retro-orbital mass.

False positive results have been reported in a variety of disorders including infections, drug-induced vasculitis, other autoimmune diseases, malignancies and inflammatory bowel disease (IBD). Conversely, negative results have been reported in patients with biopsy proven vasculitides. Results therefore should always be evaluated in the context of other laboratory and clinical findings and should not be used as the principal indication for treatment.

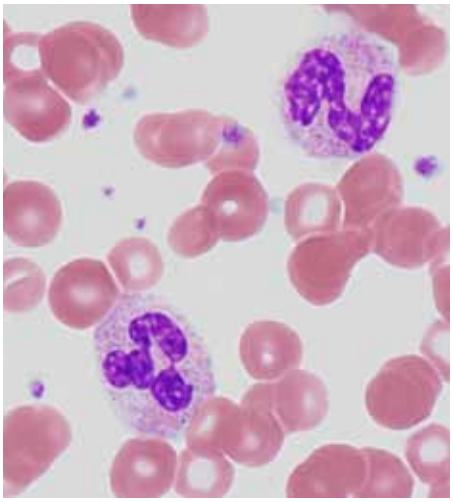
There are three major patterns associated with a positive ANCA screen:

1 Cytoplasmic or cANCA

2 Perinuclear or pANCA

3 Atypical-ANCA pattern

The atypical ANCA pattern has been reported in IBD, cystic fibrosis, autoimmune liver disease, drug-induced ANCA and rheumatic diseases. In rheumatoid arthritis the prevalence of atypical-ANCA has been detected in 20 – 70% of patients and been associated with more severe and long-standing disease.



The ANCA group of autoantibodies are directed against cytoplasmic components in human neutrophilis

Confirmation

There are many different antigen specificities in the ANCA group of autoantibodies but only two have proven clinical associations, anti proteinase 3 (PR3) and anti myeloperoxidase (MPO) antibodies. Tests for other ANCA specificities are not currently clinically useful and none have been proven specific or diagnostically important for any particular disease. Therefore all positive ANCA screens must have their identity confirmed to PR3 and MPO antibodies.

Results should be evaluated in context of other laboratory and clinical findings

What is ANCA?

ANCA is a group of autoantibodies directed against cytoplasmic components in human neutrophils, the two main antigenic targets being proteinase-3 (PR3) and myeloperoxidase (MPO). Clinical studies done at SA Pathology have found the combination of an ANCA screen and confirmation tests yield the best diagnostic information.

If an ANCA screen on initial presentation is positive but negative for both PR3 and MPO antibodies it is more likely to be related to other diseases than ANCA associated vasculitides. However results must be interpreted in context with clinical findings as it may represent a limited form of disease. Patients for whom there is a high index of suspicion should be monitored with PR3 and MPO antibodies, which may become detectable as the disease progresses.

Clinical associations

To date only PR3 and MPO antibodies have been shown to be of value in the diagnosis of vasculitides.

The strongest disease association is between anti PR3 antibody and Wagner's granulomatosis, which has been reported in about 80% of active cases, however sensitivity varies according to disease activity and extent. The more limited forms have sensitivities in the order of 67%, while generalised forms are nearly 100% of patients.

PR3 antibody levels appear to parallel disease activity. In general high levels of anti-PR3 antibodies indicate active vasculitis and a sharp rise in levels signifies disease flare. Monitoring ANCA levels may be useful in discriminating between a disease flare and non-specific infections in patients with SVV.

The presence of anti MPO antibody strongly suggests necrotising vasculitis. It has a reported sensitivity around 60% for microscopic polyangiitis and 50% for Churg Strauss syndrome. About 25% of patients with Wagner's Granulomatosis are also anti MPO positive. Anti MPO levels do not appear to reflect the disease activity of patients with primary vasculitides and the effects of treatment are not well documented.

Anti MPO antibody is occasionally found in other forms of glomerulonephritis. It is present in about 30 – 40% of patients with anti glomerular basement membrane (GBM) disease, and these patients appear to have a better prognosis than those with GBM antibodies alone. Antibodies also occur in drug-induced lupus and occasionally in certain other connective tissue diseases.

In general, anti MPO and anti PR3 do not occur in the same patient concurrently.

When to treat?

Systemic necrotising vasculitides are serious life threatening diseases, fatal if untreated. Since the introduction of combination corticosteroid and cyclophosphamide therapy clinical outcomes have improved dramatically. The current treatment schemes also include aggressive immunosuppressive therapy.

Treatment should be started early as there is good evidence that the extent of organ involvement at onset determines the ultimate prognosis, hence prompt diagnosis is critical.

Other factors besides diagnostic tests need to be considered before deciding to administer toxic immunosuppressive therapy, they include the:

- probability of improvement and potential side effects with additional biopsy investigations
- consequences and costs of mistreating nonvasculitic disorders
- consequences and costs of delaying or missing the diagnosis.

In the appropriate clinical setting for those patients with renal findings suggestive of vasculitis, initiation of immunosuppression based on ANCA results alone without renal biopsy appears justified.

In other clinical settings, a positive ANCA screen and MPO/PR3 antibodies are not sufficient for diagnostic decisions on patient treatment. Positive ANCA results in these settings must be confirmed with biopsy investigation.

In a successfully treated vasculitis patient ANCA levels should disappear or decrease significantly. If this is not the case, or the levels reappear, a clinical exacerbation is likely to occur within the next few weeks or months. ◘

Type 1 diabetes: early diagnosis essential

DRS JESSICA PHILIPS, JENNY COUPER, JAN FAIRCHILD, ALEXIA PEÑA AND ELAINE THAM –
PAEDIATRIC; ENDOCRINOLOGY WOMEN'S AND CHILDREN'S HOSPITAL

Every day in Australia two children will be diagnosed with type 1 diabetes. Currently Australia has the world's sixth highest rate for new diagnoses of type 1 diabetes. The incidence is highest amongst teenagers, with a second smaller peak amongst 5-9 year olds, but it can occur at any age, including infancy.

A combination of genetic and environmental factors is thought to precipitate the autoimmune destruction of the pancreas which leads to Type 1 diabetes. Despite years of research, improvements in insulin and insulin delivery devices a cure remains elusive.

Over 30% of children with type 1 diabetes still present in diabetic ketoacidosis (DKA), a life threatening complication and the leading cause of death in children with type 1 diabetes. Recognising the symptoms and signs of the disease and starting insulin early can prevent morbidity and mortality.

The following case study illustrates the importance of early diagnosis and treatment.

CASE STUDY

Mia is 4 years old. Her mother takes her to the GP as she is concerned Mia might have a bladder infection. She has been going to the toilet frequently and has started wetting the bed again after being mostly dry overnight for over a year. Today Mia complained of a sore tummy. She has had no fever or vomiting, but has been quite thirsty.

Mia has no significant past medical history. Her grandfather has type 2 diabetes.

Her GP agrees a urinary tract infection is likely, though is wondering about type 1 diabetes.

Her urinalysis results are:

Specific gravity	1.01
pH	5.0
Leukocytes	negative
Nitrites	negative
Ketones	+
Glucose	+++

Because of the glucosuria, her BGL is checked and is 14mmol/L. Her GP thinks Mia may have type 1 diabetes and asks them to return in the morning for a fasting blood glucose level.

Overnight Mia starts vomiting and the abdominal pain worsens. Her mother takes her to the emergency department. Her BGL on arrival is 18mmol/L with blood ketones of 3.7mmol/L. A blood gas shows a metabolic acidosis with a pH of 7.15 and bicarbonate of 10mmol/L. Mia is admitted to the paediatric intensive care unit and an insulin infusion started.

Symptoms

Children with type 1 diabetes usually present with a 2-6 week history of polyuria, polydipsia, and weight loss. Bedwetting is also common.

These symptoms are often attributed to urinary tract infections or psychogenic polydipsia. If these early symptoms are not recognised and ketoacidosis develops, vomiting, abdominal pain, dehydration, reduced consciousness and hyperventilation will ensue, and can be mistaken for gastroenteritis, acute abdominal pain, asthma or pneumonia.

Diagnosis

Once suspected, type 1 diabetes can be easily diagnosed with a blood glucose meter. The diagnosis is made if the blood glucose level (BGL) is elevated:

- fasting BGL ≥7mmol/L or
- random BGL ≥11.1mmol/L

A fasting BGL, oral glucose tolerance test (OGTT) or an HbA1c are not required for diagnosis. Waiting for the results of extra tests will only delay the diagnosis and management.

Testing blood or urine ketones will help determine if ketoacidosis is likely.

Children diagnosed with type 1 diabetes require immediate referral to a hospital with paediatric services to commence insulin and organise multidisciplinary education and management.

Children with diabetes can deteriorate quickly, and it is not uncommon for a child to present in severe DKA whilst waiting to have a fasting BGL.

The fasting BGL is not necessary for a symptomatic patient with elevated random BGL. Very early on in the disease post-prandial BGLs are the first to rise and fasting BGLs remain normal.

Summary

Type 1 diabetes can occur at any age, and is easily diagnosed if suspected.

Polyuria, polydipsia, bedwetting and weight loss are usual early symptoms.

A random blood glucose level >11.1mmol/L, in a symptomatic child is enough to make the diagnosis.

Fasting blood glucose, OGTT or HbA1c are not necessary to make the diagnosis and can delay treatment.

Early diagnosis and immediate referral to a doctor experienced in the management of type 1 diabetes in children can prevent diabetic ketoacidosis.

Children with diabetes can deteriorate quickly

Recognising risk

Raising awareness within the community about the four common signs of diabetes – weight loss, increased thirst, increasing urination and fatigue, reduces the number of children diagnosed late as in Mia's case.
A recent Australian study demonstrated that a population awareness campaign was effective in reducing the number of children who present in DKA by 64%.

RESEARCH SPOTLIGHT

Leading Light Award

Associate Professor Susan Branford

Associate Professor Susan Branford of the Leukaemia Unit, Department of Molecular and Genetic Pathology, Centre for Cancer Biology at SA Pathology won the Australian Society for Medical Research (ASMR) SA Leading Light Award in September this year.

This prestigious award recognises the exceptional research output by mid-career researchers who have pursued their own research direction, and highlights the outstanding work being undertaken by up and coming researchers in South Australia. The award was presented by Professor Ian Frazer.

Young Investigator Award

Dr Julia Kuliwaba

SA Pathology's Dr Julia Kuliwaba, a researcher in surgical pathology, is the recipient of an International Bone and Mineral Society (IBMS) 2013 Sun Valley Young Investigator Award – the Alice L. Jee Award – for her research investigating the pathophysiology of osteoarthritis.

The award was presented at the 43rd International Sun Valley Workshop on Musculoskeletal Biology, Sun Valley, Idaho, in August 2013.

Clinical utility of bone turnover markers

DR DEVIKA THOMAS

Osteoporosis is a major public health issue.

Diagnosis relies on a bone mineral density (BMD) measurement, using dual energy X ray absorptiometry, and is defined as a bone density more than 2.5 standard deviations below the young normal (peak bone) values at the lumbar spine or the hip. Bone turnover markers (BTM) can be used as a complement to BMD in monitoring treatment response as well as fracture prediction in patients with osteoporosis.

Bone Turnover Markers

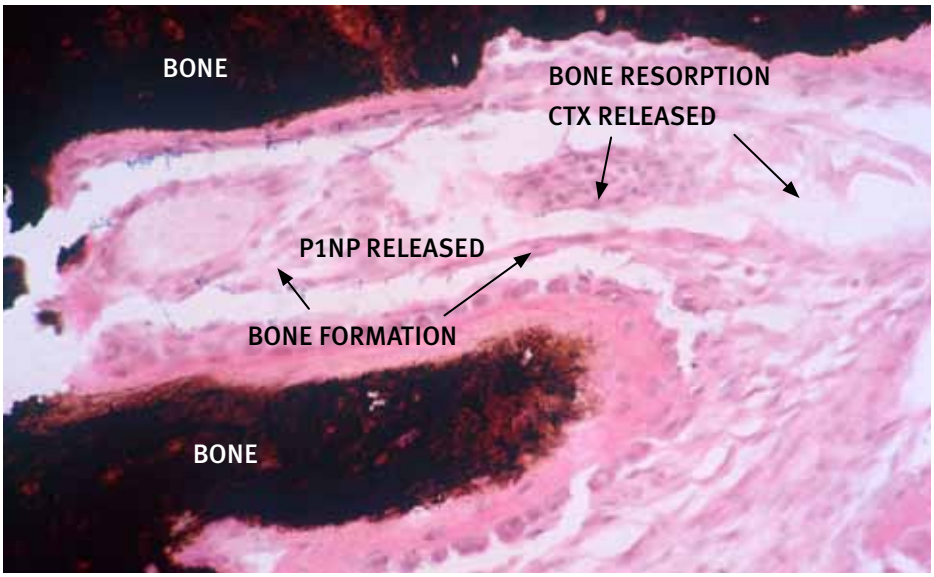
BTM are products of bone formation and resorption. Bone is dynamic tissue with formation and resorption occurring concurrently at many multi-cellular bone remodelling units, hence the measurement of BTM reflects bone turnover and rates of formation and resorption.

Bone formation

Products of osteoblasts (osteocalcin, bone specific alkaline phosphatase) and type 1 procollagen extension products (P1NP) are markers of bone formation that can be measured in serum. Total alkaline phosphatase may be used in place of bone specific alkaline phosphate in the absence of liver disease.

Bone resorption

Products of osteoclasts and terminal telopeptides of mature type 1 collagen (like serum crosslaps – CTX) are bone resorption markers. Currently CTX is the most widely used bone resorption marker having replaced the urine-based crosslinks test. Products of osteoclasts are not routinely measured in clinical practice.



Which markers?

Fasting morning serum CTX and P1NP are convenient measures of bone turnover. Diurnal variations and effects of food intake affect marker levels, and serial collections for monitoring should be collected under the same conditions, at the same time of day and analysed by the same laboratory to minimise biological and analytical variability.

Who to test and when?

BTM testing is not a screen for osteoporosis and has not been validated as a diagnostic test, which still requires bone density measurement. However, BTM testing is useful for monitoring the treatment response to antiresorptive agents in patients with osteoporosis, and in the early recognition of non responders. This is an important indication because alternative treatment may be offered to non responders.

BTM testing may be requested prior to commencement of treatment. Following initiation of anti-resorptive therapy it is useful to measure BTM again at three to six months to ensure adequate response, followed by re-assessment once or twice a year while treatment continues.

Follow up with BTM testing is still useful even without a baseline value, if the blood samples are collected under recommended conditions (ie: morning fasting samples) and analysed by the same laboratory to minimise variabilities.

Clinical Utility

BTM are not specific to a disease and therefore cannot be used for diagnosis, however raised bone resorption as reflected by raised CTX may suggest bone loss and loss of bone microarchitecture not captured by bone density measurements. Therefore changes in BTM precede changes in BMD.

BTM are raised in Paget's disease, osteoporosis and other conditions where bone turnover is high, such as rheumatoid arthritis, hyperparathyroidism and hyperthyroidism. They are also generally higher in patients with renal impairment due to low clearance.

Monitoring therapy

There is no consensus on a BTM target with as many as 50% of women with osteoporosis having a BTM in the premenopausal range. The aim of treatment would be to return BTM to premenopausal levels. In patients treated with anti-resorptive agents, levels of serum CTX of <400ng/L or at least 30% reduction from baseline level should be the therapeutic target.

P1NP has been shown to convincingly and significantly rise after treatment with teriparatide. When treated with anti resorptive agents such as bisphosphonates, strontium ranelate and denosumab, BTM fall significantly compared to baseline values.

When monitoring treatment response the target would be a 30% fall in bone resorption markers within weeks of commencing therapy. The desirable limit for P1NP in postmenopausal women is <75 ug/L.

Table 1 P1NP reference range

FEMALES	
AGE	P1NP (ug/L)
<30 years	25-90
30-39 years	15-80
40-49 years	15-60
>50 years	15-75
MALES	
AGE	P1NP (ug/L)
>25 years	15-80

BTM are also useful in recognising non compliance and sometimes, helps identify those who may take the medication incorrectly (i.e. taking bisphosphonate tablets with food or calcium tablets).

Fracture prediction

There is clear and convincing evidence from epidemiological studies that BTM are an independent predictor of fractures, particularly of the spine and hip. Elevated CTX levels are associated with significantly increased risk of fragility fractures. However there is no evidence to support BTM for predicting fractures in individual patients and fracture calculators do not incorporate BTM as yet.

BTM limitations

- There is no consensus on the use and interpretation of BTM and no recommendations in clinical guidelines.
- Reference intervals and desirable limits vary between laboratories.
- The baseline BTM level cannot be used for diagnosis of osteoporosis, it does not direct treatment choice nor will it predict treatment outcome.
- Only a fall in BTM greater than the least significant change (30% for serum markers) may be regarded as a response to treatment and proof of compliance.
- It is not known to what extent BTM should fall to optimise anti-fracture efficacy.
- Bone markers may be higher in patients with renal failure and dialysis dependent patients due to accumulation over time, and may not directly reflect bone turnover rate.

Summary

BTM can be used to identify patients with high bone turnover who may be at greater risk of fragility fracture. Comparison between baseline and subsequent BTM levels can be used to monitor treatment response and patient compliance. In patients treated with anti-resorptive agents, at least 30% reduction from baseline level should be the therapeutic target.

Ulysses syndrome – is it the liver?

DR DEVIKA THOMAS

This interesting case highlights the value of clinical history when interpreting test results, particularly those like liver enzymes with significant non-specificity. It reminds us that context is crucial to unravelling the complex web of influences that produce any set of test results. For more on the liver function tests refer to *IMVS Newsletter 57*.

CASE STUDY

A 20 year old football player was admitted to the Emergency Department with a fractured mandible. Routine biochemistry tests revealed elevated AST, ALT and LD with normal GGT, ALP and bilirubin. There were no clinical or historical findings to explain the elevated Liver Function Test (LFT) results. Following surgical repair of the mandible the patient's GP was informed of the results and requested follow up. Repeat results by the GP revealed the following.

ASSAY	RESULT	RANGE
Bilirubin	9 umol/L	(2-24)
GGT	13 U/L	(<60)
ALP	90 U/L	(30-110)
ALT	130 U/L*	(<55)
AST	339 U/L*	(<45)
LD	363 U/L*	(110-230)

The GP organised a liver and biliary ultrasound which was normal. The patient was then referred to the liver clinic. Subsequent investigations were as follows:

- ALT and AST had risen to 220 and 684 U/L respectively
- Hepatitis A IgG, Hepatitis B surface antigen and Hepatitis C antibody tests were negative
- CMV and EBV IgM serology tests – negative
- Autoantibody tests including ANCA, ANA, ENA, dsDNA, anti mitochondrial, anti LKM and anti smooth muscle – all negative
- Ceruloplasmin, alpha 1 antitrypsin, haptoglobin, serum protein electrophoresis and coagulation studies were normal
- Iron studies showed normal iron status
- Chest, abdominal and pelvic CT scans – normal
- Liver biopsy revealed normal hepatic and biliary tree histology

Repeat liver function tests confirmed ALT, AST and LD remained elevated while GGT, ALP and bilirubin were normal.

The patient was referred to a haematologist but no haematological cause was found for the abnormal results. Following consultation with a chemical pathologist, and considering the patient's routine of playing football most days, a CK level was requested, which was 2605 U/L (250U/L).

Discussion

Liver enzyme patterns are used to classify liver disease into two broad categories – cholestatic disease (biliary obstruction) or hepatocellular disease. GGT and ALP are mainly located on cell membranes and particularly line the biliary cannaliculi, therefore they are raised in any obstructive biliary disease (cholestasis, metastatic lesions). They may also be induced by alcohol and a variety of medications, anti-convulsants being the most common.

Transaminases (ALT and AST are intracellular enzymes that are released due to cellular injury, and are elevated in viral hepatitis and in cell destruction due to toxins and medications such as high dose paracetamol. Levels may rise 2 to 100 fold. Other causes of raised transaminases are non alcoholic steatohepatitis, haemochromatosis and autoimmune liver disease.

AST and LD are also found in red blood cells and muscle cells including cardiac cells. Intravascular haemolysis or haemolysis of the sample after collection often raises LD and AST. In some haematological diseases LD may be markedly elevated (myelodysplasia and pernicious anaemia).

If cardiac causes are suspected then a troponin T assay is recommended

'Ulysses syndrome' is the term used to describe an unnecessary complication of the diagnostic decision making process where false-positive diagnostic test results or clinical decisions trigger a complex diagnostic work-up to elucidate the nature of what is, in fact, not a disease. The syndrome is named after Classical Greek hero Ulysses, who fought in the Trojan War (1194 to 1184 BC) and subsequently took 20 years to return home to Greece; however all his harrowing diversions proved unnecessary.

along with an ECG and clinical history. AST and LD are abundant in skeletal muscle. Although ALT is regarded as a liver specific transaminase, it is also present in skeletal muscle and with persistent muscle injury ALT may also be released.

History lesson

A good history would have given the clue to severe muscular exertion as a potential cause. In this case, persistent exertions in the form of severe exercise lead to the consistently elevated AST, ALT, LD and CK from skeletal muscle. (The half life of ALT is up to 57 hours while that of AST and CK are less than 24 hours.)

The differential diagnosis could have been supported by requesting the patient to have a repeat blood test after a few days rest to confirm that the cause was indeed exertion.

The 'health dollar' is a premium commodity, and in this case it's easy to see how much time effort and money could have been saved by including a good history with the request. ■

for list of acronyms see page 12

Test ordering standardisation

MBA

Historically ordering an MBA panel has been something of a 'movable feast'. Everyone seems to have their own interpretation on what should be reported with an MBA.
To clarify this situation SA Pathology has implemented consistent panel definitions for the basic chemistry tests.

If you request an MBA, or any of the test panels listed below, you will receive the tests as listed.

MBA
Electrolytes
Urea
Anion Gap
Creatinine
Glucose
Urate
Phosphate
Total Calcium
Albumin
Total Protein
Total Bilirubin
GGT
ALP
ALT
AST
LDH
Total Cholesterol
Globulins
Calculated Ionised
Calcium
eGFR

MBA
Multiple Biochemical Analysis

RFT
Renal Function Test

LFT

If you request an LFT plus an additional chemistry test from the MBA suite then you will receive a full MBA report.

LFT
Albumin
Total Protein
Total Bilirubin
GGT
ALP
ALT
AST
LDH (coming)

RFT or ECU?

SA Pathology has also now standardised on ECU as the renal function panel. Note that glucose is not included in the ECU panel and should be separately requested, and a grey top sample collected. The grey top provides a more stable collection environment for glucose and hence results are more accurate.

ECU
Electrolytes
Anion Gap
Urea
Creatinine
eGFR

- For more information please speak to a SA Pathology Marketing representative

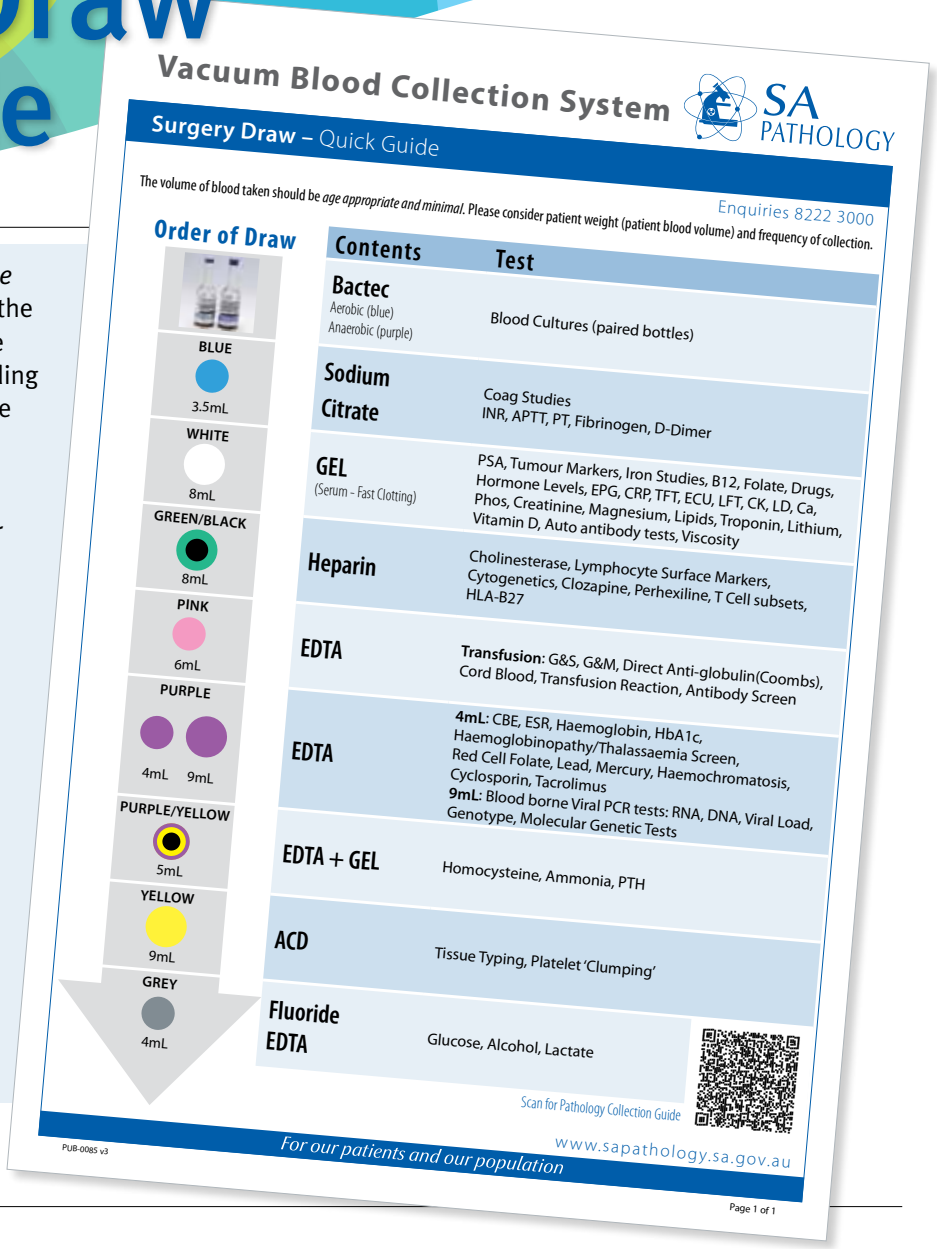
LFT
Liver Function Test

ECU
Electrolytes, Creatinine, Urea

Order of Draw Quick Guide

The SA Pathology *Order of Draw Quick Guide* has recently been updated. Whilst many of the tests and tubes remain the same there have been a number of significant updates including the introduction of the 'yellow top' for tissue typing and platelet clumping.

For comprehensive information on all the tests SA Pathology provides please visit our web site at www.sapathology.gov.au or simply scan the QR code with your smartphone to go direct to the Pathology Collection Guide page.



Vacuum Blood Collection System

SA PATHOLOGY

Surgery Draw – Quick Guide

The volume of blood taken should be *age appropriate and minimal*. Please consider patient weight (patient blood volume) and frequency of collection.

Enquiries 8222 3000

Order of Draw	Contents	Test
	Bactec (Aerobic (blue) Anaerobic (purple))	Blood Cultures (paired bottles)
BLUE 3.5mL	Sodium Citrate	Coag Studies INR, APTT, PT, Fibrinogen, D-Dimer
WHITE 8mL	GEL (Serum - Fast Clotting)	PSA, Tumour Markers, Iron Studies, B12, Folate, Drugs, Hormone Levels, EPG, CRP, TFT, ECU, LFT, CK, LD, Ca, Phos, Creatinine, Magnesium, Lipids, Troponin, Lithium, Vitamin D, Auto antibody tests, Viscosity
GREEN/BLACK 8mL	Heparin	Cholinesterase, Lymphocyte Surface Markers, Cytogenetics, Clozapine, Perhexiline, T Cell subsets, HLA-B27
PINK 6mL	EDTA	**Transfusion**: G&S, G&M, Direct Anti-globulin(Coombs), Cord Blood, Transfusion Reaction, Antibody Screen
PURPLE 4mL 9mL	EDTA	**4mL**: CBE, ESR, Haemoglobin, HbA1c, Haemoglobinopathy/Thalassaemia Screen, Red Cell Folate, Lead, Mercury, Haemochromatosis, Cyclosporin, Tacrolimus **9mL**: Blood borne Viral PCR tests: RNA, DNA, Viral Load, Genotype, Molecular Genetic Tests
PURPLE/YELLOW 5mL	EDTA + GEL	Homocysteine, Ammonia, PTH
YELLOW 9mL	ACD	Tissue Typing, Platelet 'Clumping'
GREY 4mL	Fluoride EDTA	Glucose, Alcohol, Lactate

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For our patients and our population

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ACRONYMS

	CMV Cytomegalovirus
LFT Liver Function Tests	**EBV** Epstein Barr Virus
GGT Gamma Glutamyl Transferase	**ANCA** Anti Neutrophil Cytoplasmic Antigen
ALP Alkaline Phosphatase	**ANA** Anti Nuclear Antibody
ALT Alanine Aminotransferase	**ENA** Extractable Nuclear Antigen Antibody
AST Aspartate Amino Transferase	**dsDNA** Double stranded DNA antibody
LD Lactate Dehydrogenase	**LKM** Liver-Kidney Microsomal Antibody
CK Creatine Kinase	**CT** X-ray Computed Tomography

Allergy Test Requests

The test information sheet, *'Allergen Testing Guidelines'* contains recommendations related to ordering. You can read or print the guidelines on the SA Pathology web site. From the home page go to: For Clinicians | Pathology Collection Guide and type 'allergy' into the search box; click on the More Info link.

For clinical questions please phone SA Pathology Enquiries on (08) 8222 3000 and ask for the Immunopathology Registrar or on call Immunopathologist.

The SA Pathology *Allergy Test Request* form has been discontinued.

SA Pathology enquiries Metropolitan 8222 3000 Regional and Country 1800 188 077