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From our Medical Director



Welcome to the second issue of our pathology team newsletter. We believe 2010 has started well for Labtests as we continue to streamline our operations. We have noticed a marked improvement in clinical relationships as GPs, midwives and other

referrers get to know our pathologists.

Dr Craig Marshall and I continue to visit practices and find it highly enjoyable getting to know doctors and practice staff in their own environments. These visits are also a source of very useful feedback, and we continue to welcome and take on board your comments and suggestions.

Completion of haematology team

We are very pleased to have concluded our haematologist staffing with the appointments of Dr Andre Simmance and Dr Shahid Islam. They join Drs Russell O'Neil, Lochie Teague and Nigel Patton to complete the Labtests haematology team.

Andre Simmance joins us from his role as consultant haematologist at Healthscope's Gribbles Pathology in Melbourne. Shahid Islam has recently completed his fellowship at Auckland Hospital and will be working for us part time from 12 March.

Pathology update meetings

This week we begin the first of our quarterly pathology update meetings at Waipuna Lodge and the Bruce Mason centre.

Presentations will include anatomical pathologist Dr Vladimir Osipov on 'Problematic Cases in Dermatopathology' and chemical pathologist Dr Jeffrey Barron on the interpretation of GFR and estimated GFR.

One focus this year will be ascertaining whether these quarterly meetings are an effective way of providing CME for general practitioners.

Labtests pathologists are also looking forward to speaking at the Goodfellow Symposium, a widely-attended educational meeting for GPs, at the end of March. We will be addressing commonly-asked questions from practitioners in the fields of microbiology, chemistry, haematology and immunology.

Please don't hesitate to contact us here in the pathology team with your clinical questions, or to get in touch with me directly with any questions or concerns.

Dr Richard Lloyd

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MEET THE TEAM

Janet Wilson

HOD Microbiology

My background

I was born and brought up in Zimbabwe and trained and worked in Zimbabwe and Scotland. I moved to New Zealand in 1980 and worked at Dunedin Hospital until 1990, when I joined Southern Community Laboratories. In 2001 I moved to Auckland to set up SCL Auckland.

After SCL closed in 2005, I went to work for the New Zealand Food Safety Authority. In October 2006 I was approached to join Labtests (Mark 1). While waiting for the court decision I worked at Northland Pathology in Whangarei. In February 2009 I moved back to Auckland to join Labtests (Mark 2).

My role

As HOD Microbiology, my role is to oversee a diverse team of 61 full- and part-timers and to ensure the efficient running of this busy department. As a team, our job is to deal with the specimens that you don't talk about at dinner parties – faeces, urine, semen, pus etc. It's interesting work – and someone's got to do it!

Highlights of the job

The biggest highlight for me so far was being personally responsible for interviewing and employing this great team of people from all over the world – from Bulgaria to Ethiopia to China to Fiji. And the satisfaction of transforming 400 square metres of empty space into a well-equipped, smooth-running department processing more than 3000 samples per day.

Where you'll find me outside work

You'll usually find me with my family. I moved to Auckland on my own, thinking I had no responsibilities (I even bought a two-seater sports



car) and then my kids all moved here after their OEs! And my mother has joined us from the UK.

I'm a water person and I love the warm weather in Auckland. I swim, kayak and sail. I race with the Ponsonby Cruising Club and the Royal New Zealand Yacht Squadron throughout the year. I also enjoy cruising, having sailed from Auckland to Tonga and Australia to New Zealand.

Investigating sterile pyuria



Normal urine can have a white blood cell (WBC) count of up to 10×10^6 WBC/L. A count greater than 10×10^6 WBC/L is considered abnormal (pyuria).

The most common cause of pyuria is bacterial infection, which is usually detected by routine bacterial culture. Sterile pyuria is the finding of a count greater than 10×10^6 WBC/L in urine in the absence of bacteria on routine culture.

However, with increasingly sensitive microscopy methods, low-level pyuria (for example, $10\text{--}40 \times 10^6$ WBC/L) may not represent clinical disease.

In addition, contaminated specimens (where there are more than 10×10^6 epithelial cells/L) can have an elevated WBC due to mucosal contamination.

Dr Arlo Upton and the urine team will soon be doing a study in healthy volunteers in order to establish normal reference ranges for this technology in our community.

Infectious causes of sterile pyuria

- Resolving urinary tract infection on antibiotics
- Urethritis due to:
 - *Chlamydia trachomatis*
 - *Neisseria gonorrhoeae*
 - *Trichomonas vaginalis*
 - *Ureaplasma*
- Herpes simplex virus
- Urinary tract infection due to 'fastidious' (difficult to grow) organisms such as *Haemophilus* species, anaerobes
- Renal tuberculosis
- Prostatitis

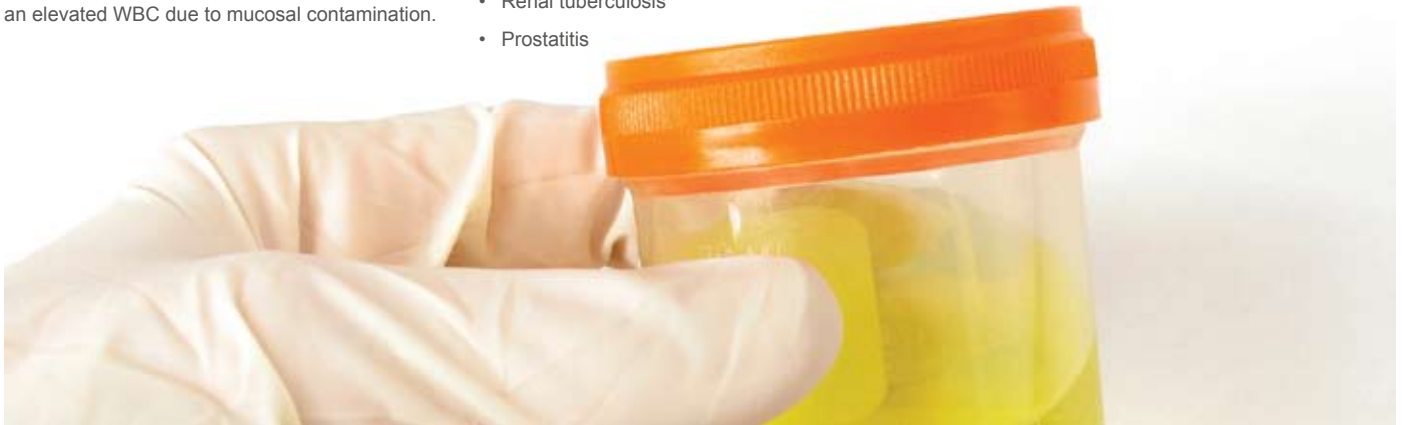
Non-infectious causes of sterile pyuria

- Urinary tract tumour
- Renal calculi
- Trauma (such as recent instrumentation)
- Inflammatory condition of adjacent bowel (e.g. appendicitis)
- Renal disease (often drug-related)

The investigation of sterile pyuria should be patient-specific, taking into account gender, age, risk factors and clinical history. The guidelines below provide a general approach that can be used in conjunction with clinical findings.

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Guidelines for investigating sterile pyuria

Contaminated specimen	Repeat specimen
Recent antibiotics	Repeat specimen in approximately two weeks, after completion of antibiotics
Sexual activity	Request first-void urine for chlamydia PCR and collect swabs for STI check
History of trauma	Repeat specimen in approximately two weeks
History consistent with possible calculi	Repeat specimen/consider investigating for calculi
Proteinuria/elevated creatinine	Consider intrinsic renal disease
None of above	Repeat specimen and request culture for fastidious organisms
No growth of fastidious organisms/ risk factors for tuberculosis	Three early morning urines for tuberculosis culture
Possible renal tumour	Urine cytology

References

Cutoff values for bacteria and leukocytes for urine flow cytometer Sysmex UF-1000i in urinary tract infections. Manoni F et al. *Diagn Microbiol Infect Dis*. 2009 Oct; 65(2): 103-7
Sterile pyuria: a differential diagnosis. Dieter R. *Compr Ther*. 2000 September; 26(3): 150-2

The role of eGFR in diagnosing early kidney disease



Estimated glomerular filtration rate (eGFR) is reported on every request for a serum creatinine in non-pregnant adults over the age of 18 years. The eGFR is calculated from the serum creatinine, age

and sex of the patient. This allows for correction for the patient's body surface area.

In the hierarchy of kidney function tests, eGFR is better than measured creatinine clearance in individuals who have normal body composition. Estimated GFR has not been validated in those under 18 years of age, in pregnancy, at extremes of body composition or for different ethnic groups.

Utility of eGFR reporting

The management and timely referral of patients with early kidney disease depends on the identification of kidney insufficiency by primary care physicians. It is well known that patients can have significantly decreased eGFR while the serum creatinine is still within the reference interval. Serum creatinine only starts to rise when around 50% of the glomeruli have been lost.

References

Chronic kidney disease and automatic reporting of estimated glomerular filtration rate: revised recommendations. Mathew TH et al. *Med J Aust.* 2007; 187(8): 459-463
Interpretation of the eGFR. Mathew T, Jones G. *Comm Sense Path.* July 2007; 2-8

The most sensitive indicator of minor kidney damage is a rise in serum creatinine compared to the patient's own previous result.

Serum creatinine is dependent on muscle mass. It is lower in children, women and the elderly. It is subject to interference from ingestion of creatine and cooked meat, some cephalosporins and haemolysis.

Recently laboratories have started reporting eGFR values up to 90ml/min/1.73m² because improved standardisation and greater accuracy in serum creatinine measurement have been achieved.

Studies have shown that the long-term complications associated with reduced GFR begin to appear at levels below 60ml/min/1.73m². However, chronic kidney disease may exist at eGFR values above 60ml/min/1.73m².

Patients with mild decrease in eGFR

For a patient with an eGFR of 60-89ml/min/1.73m², practitioners should take a history and an examination. Blood pressure control should be optimised and the urine tested for blood and protein using a dipstick.

A negative test for blood excludes microscopic haematuria. Because of the lower sensitivity of the stick tests for protein, a negative test for protein means that at worst, microalbuminuria maybe present.

If urinalysis is negative and blood pressure is well controlled then no further investigations are required. An annual screen of serum creatinine and urinalysis is indicated. The finding of stable eGFR 60-89ml/min/1.73m², not associated with other evidence of kidney damage, appears to be of little clinical consequence.

If urinalysis is positive for protein or haemoglobin, then an early morning urine sample should be sent for protein determination, urine microscopy and culture.

In all cases of chronic kidney disease, an ultrasound of the kidneys and bladder should be requested to exclude obstruction and confirm normal renal anatomy. A second eGFR should be performed to confirm the findings and referral to a nephrologist should be made without delay.

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Common questions about eGFR

Why is eGFR not reported with every adult serum creatinine?

eGFR can only be calculated if the age and sex of the patient are known.

When is eGFR inaccurate?

At the extremes of body composition.

Does extensive bruising increase serum creatinine? No.

Is there any place for serum creatinine clearance?

Rarely: at the extremes of body size, severe malnutrition or obesity, skeletal muscle disease.

CASE STUDY

Tinea nigra

A superficial fungal skin infection clinically mimicking melanoma

A 53-year-old woman returned from Queensland with a variably pigmented brown macule on the sole of her right foot. She said the lesion had been growing rapidly for four weeks. There were three darker 'satellite' lesions and the dermatoscopic features in one of these were interpreted as melanoma.

Histology of the excision biopsy shows acute dermatitis with fungal hyphae, some of which are pigmented, in the stratum corneum. Tiny spongiotic vesicles are present in the epidermis and lymphocytes focally infiltrate the base of the epidermis and around blood vessels in the upper dermis.

Characteristics of tinea nigra

Tinea nigra is an uncommon superficial fungal infection, usually of palmar or plantar skin and sometimes bilateral. The disease occurs mainly in the tropics and is usually caused by *Hortaea werneckii* or *H. mansonii*, two of a group of melanised yeast-like fungi tolerant of a high-salt, low-pH environment.

The infection is probably acquired by inoculation

following trauma and is more common in females and in children and adolescents. As in this case, tinea nigra can mimic melanoma or other melanocytic skin lesions and there may be an associated dermatitis.

Distinction from a melanocytic lesion depends on suspecting the diagnosis in someone who has returned from the tropics and recently noticed a pigmented lesion on a palm or sole, possibly with some pruritis. The diagnosis can be confirmed with routine mycology. Dermatoscopy may also assist in making the diagnosis.

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References

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- Enlarging pigmented patches on the hand. Muellenhoff M, Cukrowski T, Morgan M, Miller R. *Int J Dermatol*. 2003 Oct; 42(10): 810-1
- Entodermoscopy: a new tool for diagnosing skin infections and infestations. Zalaudek I, Giacomel J, Cabo H et al. *Dermatology*. 2008; 216(1): 14-23

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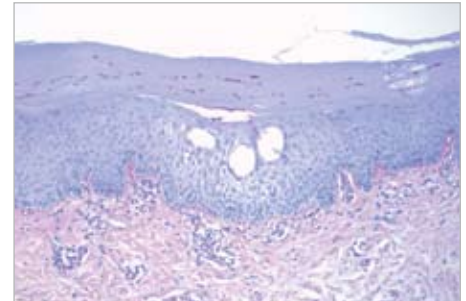


Figure 1. Fungal hyphae in the stratum corneum (PAS stained section)

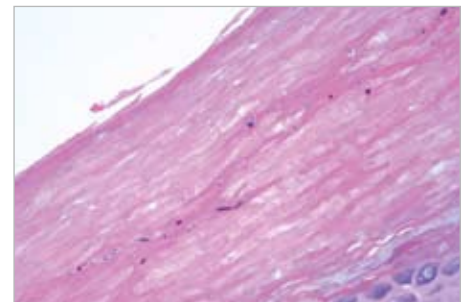


Figure 2. Fungal hyphae showing melanin pigmentation

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