Letters to the Editor

THE TINU SYNDROME

Sir, We write in response to *Uveitis: diagnosis and management.*¹ To quote: 'When a systemic condition is suspected collaboration with physicians is advisable'. As physicians, we would like to draw attention to the TINU syndrome.

TINU is an acronym for tubulointerstitial nephritis with uveitis. Tubulointerstitial nephritis is an uncommon cause of renal impairment being found in just 1–3% of unselected renal biopsis^{2,3} and 8–14% of biopsies for unexplained acute renal failure.⁴ It is often considered as an 'allergic' response, and in 40–60% of cases an identifiable allergen, often a drug, is found. The association of TINU with iritis was first made in 1987,⁵ since which time further reports of TINU with unilateral or bilateral uveitis or iritis have been made.

TINU is commoner in females and typical symptoms may include fever, weight loss, myalgia and those of uveitis/ iritis.⁶ Investigations may demonstrate renal impairment with or without an active urinary sediment, eosinophilia and eosinophiluria. Precise diagnosis of the renal lesion requires renal biopsy. Treatment with steroids usually results in a dramatic improvement, although spontaneous remission has been described. Whilst the renal lesion does not usually recur, further attacks of uveitis/iritis are well described.

We would recommend that the investigation of uveitis with systemic symptoms should include dipstick urinalysis and a serum creatinine. If either are abnormal, think TINU.

P Gibson and WD Plant

REFERENCES

- ¹ Akerle T, Lightman S. Uveitis: diagnosis and management. *Proc R Coll Physicians Edin* 2000; **30:**344-8.
- ² Laberke HG, Bohle A. Acute interstitial nephritis: correlations between clinical and morphological findings. *Clin Nephrol* 1980; 14:263-73.
- ³ Cameron JS. Allergic interstitial nephritis: clinical features and pathogenesis. *QJM* 1988; **66**:97-115.
- ⁴ Wilson DB, Turner DR, Cameron JS *et al.* Value of renal biopsy in acute intrinsic renal failure. *BMJ* 1976; 2:456-61.
- ⁵ Koeppen-Hageman I, Binkele-Vihlein V, Waldherr R et al. Akute granulomatöse interstitielle nephritis mit iritis. Deutsche Medizinische Wochenschrift 1987; **112**:259-61.
- ⁶ Rodriguez-Perez JC *et al.* Clinical and immune aspects of idiopathic acute tubulointerstital nephritis and uveitis syndrome. *Am J Nephrol* 1995; **15**:386-91.

HYPERTENSIVE HEART DISEASE OR ACUTE CORONARY SYNDROME?

Sir, I was interested to read the article by Drs Felmeden and Lip on the Afro-Caribbean patient with hypertension and ECG changes mimicking an acute coronary syndrome. The electrocardiogram can be difficult to interpret even in healthy Africans and the patterns to which Powell¹ drew attention 40 years ago deserve to be better known. In a group of African male nursing staff in Durban he recorded ECGs showing T wave invertion, elevated ST segments and tall T waves >20 mv in 22% of those examined. Those findings were later confirmed^{2,3} in Africans in other areas of the Continent in 22–31% of those examined, and in the Zambian series they were seen in women as well as men. Those variants may result in pericardial or ischaemic disease being wrongly diagnosed. Very similar records have been described in athletes⁴ but it is difficult to assess those findings without knowledge of the racial makeup.⁵

I am not suggesting that the ECG pattern seen in the patient described by Felmeden and Lip is of this nature; I just wish to emphasise how difficult interpretation of ECGs can be in those of African origin.

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REFERENCES

- ¹ Powell SJ. Unexplained electrocardiograms in the African. Br Heart J 1959; 21:263.
- ² Fleischman SJ. The normal electrocardiogram in the African. *S Afr Med J* 1965; **39:**177.
- ³ Davidson JC, Kolbe RJ, Heilpern JB. Variants in the electrocardiogram. *Med J Zambia* 1967; **1**:78.
- ⁴ Hockings BEF. Cardiac conditions affecting athletes. *Heart Views* 1999; 1:180.
- ⁵ Davidson JC. Letter. *Heart Views* 1999; 1:241.

ADJUNCTIVE THERAPY FOR BACTERIAL MENINGITIS

Sir, For the sake of completeness, the evaluation of adjunctive therapy for bacterial meningitis should address not just the central nervous system complications of this syndrome¹ but also systematic complications, so as to confront the paradox that 'despite (further) progress in antimicrobial therapy, the fatality rate of meningitis due to pneumococcus, which is the organism most often responsible for bacterial meningitis in adults, has remained unchanged (20–40%) during the last decades'.²

In a prospective study comprising 86 patients in the age range 15–87 with acute bacterial meningitis, systemic complications were documented in 19 (i.e. 22.1%), including septic shock, adult respiratory distress syndrome (ARDS), and disseminated intravascular coagulation (DIC).² Pneumococcus was the organism most frequently implicated in the first two complications, the risk of AIDS being prevalent throughout the first two weeks of illness, whilst DIC and septic shock predominantly occurred during the first week of illness. Acknowledgement of these systemic complications is a logical outcome of the recognition that bacteraemia can occur in as many as 35–77% of cases^{2.3} either as a primary event leading to the development of meningitis, or as a secondary event resulting from clearance of bacteria via the arachnoid villi to the bloodstream,⁴ justifying the recommendation that 'most patients with suspected bacterial meningitis should undergo initial evaluation in an intensive care setting'.⁵

When one allows for the fact that the authors of this recommendation did not even acknoweledge ARDS as a complication, an extension of this advice would be the caveat that patients subsequently managed outside the intensive care unit (ICU) should still be monitored for parameters such as oxygen saturation, arterial blood gases and PH to enable early identification of criteria for readmission to ICU⁶ for the purpose of artificial ventilation which, in this context, can be defined as adjunctive therapy for meningitis. A possible caveat, here, is that hyperventilation to pCO2 levels of the order of 27–30mm Hg should be avoided, on the theoretical grounds that it might result in a reduction in cerebral blood flow, possibly approaching ischaemic thresholds.⁷

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REFERENCES

- ¹ Leen, CLS. Adjunctive therapy for bacterial meningitis. *Proc R Coll Edinb* 2000; **30**:305-10.
- ² Pfister H-S, Feiden W, Einhaupl K-M. Spectrum of complications during bacterial meningitis in adults. *Arch Neurol* 1993; **50:**575-81.
- ³ Aronin SI, Peduzzi P, Quagliarello VJ. Community acquired bacterial meningitis: risk stratification for adverse clinical outcome and effect of antibiotic timing. *Ann Intern Med* 1998; **129:**862-9.
- ⁴ Roos KL, Tunkel AR, Scheld WM. Acute bacterial meningitis in children and adults. In: Scheld WM, Whitley RJ, Durack DT editors. *Infections of the central nervous system*. Raven Press: New York; 1991; 335-410.
- ⁵ Scheld WM. Bacterial meningitis, brain abscess, and other supprative intracranial infections. In: Fauci AS, Braunwald E, Isselbacher KJ *et al.* editors. *Harrison's principles of internal medicine*. 14th edition: McGraw-Hill Health Professions Division: New York; Chapter 377.
- ⁶ Smith G and Nielsen M. ABC of intensive care. Criteria for admission. *BMJ* 1999; **318:**1544-7.
- ⁷ Tunkel AR and Scheld WM. Acute Bacterial meningitis. *Lancet* 1995; **346**:1675-80.