

Erythema induratum: a case of mistaken identity

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TO THE EDITOR: In a recent issue of the Journal, Chew et al described a woman from Vietnam with skin nodules that, on histological examination, showed lobular panniculitis with granulomatous inflammation.¹ No mycobacteria were visible and a polymerase chain reaction test for *Mycobacterium tuberculosis* was negative. Two months after starting quadruple antituberculous therapy (including rifampicin), her lesions had resolved. Erythema induratum (ostensibly due to hypersensitivity to *M. tuberculosis*) was diagnosed, despite the absence of evidence of tuberculosis. Other possible diagnoses were considered, but leprosy was not mentioned.

In regions of Australia where leprosy is not endemic, the disease is frequently overlooked.² Birrell³ described a man from Malta with recurring skin lumps. Biopsy showed panniculitis with giant cells, and the man was initially misdiagnosed as having “Weber–Christian syndrome” or “relapsing febrile non-suppurative nodular panniculitis”. Soon after, another Maltese patient presented similarly. This time, leprosy was suggested, and a biopsy revealed the presence of *Mycobacterium leprae*.⁴ Re-examination of slides from the first case showed similar organisms, confirming leprosy.⁵

The patients described by Chew et al and Birrell had migrated from countries in which leprosy was endemic, and biopsies revealed granulomatous panniculitis. Weber–Christian syndrome and erythema induratum are rare, ill-defined conditions with confused aetiologies, and both lack a specific diagnostic test. Therefore, cases of leprosy can be easily misdiagnosed as one of these conditions. That the biopsy in this patient did not show visible *M. leprae* is against a diagnosis of leprosy. But in my experience, even in lepromatous (multibacillary) disease, occasionally a skin smear of a lesion or (more rarely) a biopsy specimen may fail to reveal bacilli. Of course, this would be likely if the patient had received specific treatment for leprosy previously.

Respectfully, I suggest that Chew et al should attempt to exclude lepromatous leprosy in their patient by looking for possible missed stigmata of leprosy, enquiring whether she has ever been treated for lep-

rosy, asking whether any close acquaintances have had the infection or a chronic skin condition, and following up the patient in the long term.

- 1 Chew GY, Henderson C, Quin JW. Erythema induratum: a case of mistaken identity. *Med J Aust* 2005; 183: 534.
- 2 Bennett N Mck. Diagnosis of leprosy in Victoria — a non-endemic area of Australia. *Med J Aust* 1977; 2: 349-351.
- 3 Birrell JHW. Weber–Christian syndrome: report of a case. *Med J Aust* 1952; 2: 124-127.
- 4 Mancy ES. Report of two cases of leprosy. *Med J Aust* 1953; 2: 20-21.
- 5 Birrell JHW. A note on leprosy as an aetiological factor in the Weber–Christian syndrome. *Med J Aust* 1953; 2: 7. □

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TO THE EDITOR: One of the Journal’s recent *Lessons from Practice* illustrates common errors in the approach to dermatological conditions.¹ As in all areas of medicine, an accurate diagnosis is crucial to the management of any skin disease. This is especially so if a medical practitioner institutes treatments, such as oral steroids, that have considerable potential for causing morbidity. The lessons I would draw from the case of erythema induratum described are as follows.

If you suspect an unusual presentation of a common condition, perform investigations to confirm your suspicions. Although erythema nodosum classically occurs on the anterior lower leg, lesions above the knee may occasionally be seen.

To make a diagnosis, investigations need to be appropriate. The battery of blood tests ordered in the case described would not have shed light on the pathological process occurring in the skin. There is a reluctance among the general medical community to perform skin biopsies. These procedures cause little morbidity, have a high diagnostic yield, and should be within the skill set of any medical graduate. Concern over causing a scar is often cited as a reason for not doing a biopsy. But, in my experience, patients are rarely worried about such a prospect. Missing the diagnosis is surely of much greater concern. Taking a simple biopsy, including fat, at the initial presentation would have saved the patient in question a lot of trouble and risk.

If there is no response to your treatment, it may well be that the initial diagnosis was incorrect. For example, it is common to see “steroid-resistant eczema” that is actually

intraepidermal carcinoma. Erythema nodosum will usually show at least some response to non-steroidal anti-inflammatory treatment. Lack of response to a treatment that usually works should lead to a re-evaluation of the diagnosis.

Systemic steroids should not be used for a dermatological condition without a firm diagnosis. Firstly, they can suppress many of the clinical and histological changes that allow a diagnosis to be made. Appropriate investigations need to be done before starting steroids. Secondly, a drug like prednisolone may well make matters worse, especially if, as here, there is an infectious aetiology.

Patients from areas in which tuberculosis is endemic should have this condition excluded before being given systemic steroids. A lack of obvious exposure to or symptoms of tuberculosis is not unusual in patients from such areas who are subsequently shown to harbour this infection.

The authors state that, as erythema induratum can resolve with corticosteroid treatment, this can lead to an erroneous diagnosis of erythema nodosum. Using response to treatment as a quasi-diagnostic test is dangerous indeed. Steroids will cause many conditions associated with significant inflammation to improve or even appear to resolve. But this does not mean that there is no infectious or malignant aetiology.

- 1 Chew GY, Henderson C, Quin JW. Erythema induratum: a case of mistaken identity. *Med J Aust* 2005; 183: 534. □

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IN REPLY: We thank Bennett and Muir for their pertinent comments. Our patient did not have any history or clinical evidence of lepromatous leprosy. The skin biopsy did not reveal any dermal granulomatous involvement, and there were definitely no organisms seen on an auramine stain of the biopsy specimen.

Subcutaneous involvement in leprosy is uncommon except in erythema nodosum leprosum or as a neurotropic phenomenon. When present, it tends to be a neutrophil-rich hypersensitivity necrotising vasculitis — no features of which were seen in this

case. Neither the woman's partner nor child had a chronic skin condition or clinical history of leprosy or tuberculosis. Furthermore, the patient has been followed up for 12 months, with no recurrence of the rash.

We agree with Muir that an accurate diagnosis is crucial to managing any skin disease and that there were many lessons to be gathered from this case apart from the five points we listed. It is our usual practice not to begin definitive treatment until we have examined a skin biopsy of any suspicious lesion and made a diagnosis. As this patient was very concerned about getting a scar, we did not perform a skin biopsy initially, but informed her that we may need to do so if the condition did not respond to treatment.

We agree that patients from areas where tuberculosis is endemic should have tuberculosis excluded before instituting systemic steroid treatment. In this case, the patient was given a chest x-ray by the appropriate authorities before her migration to Australia. She has not returned to Vietnam since then. Furthermore, the patient had failed a trial of a non-steroidal anti-inflammatory drug and found the lesions cosmetically distressing. Consequently corticosteroids were instituted. □

Declining iodine content of milk and re-emergence of iodine deficiency in Australia

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TO THE EDITOR: Iodine is essential for production of thyroid hormone. The recommended daily intake is 100 µg for children, 150 µg for adults and 250 µg for pregnant and lactating women.¹ Sporadic surveys of population iodine intake in Sydney, New South Wales, between 1985 and 1992 showed median levels of urinary iodine excretion (UIE) >200 µg/L, indicating iodine sufficiency.² However, a recent national study demonstrated mild iodine deficiency (median UIE <100 µg/L) in New South Wales and Victoria, borderline levels in South Australia and adequate intake in Queensland and Western Australia.³

The major sources of dietary iodine are dairy milk and dairy products, seafood and iodised salt. In Australia, few people purchase iodised salt, and, except in Tasmania, the food industry does not use iodised salt in the production and preparation of food.⁴ For decades, milk contaminated with iodine residues from sanitising solutions (iodophors) used in the dairy industry has probably been the largest source of iodine in the Australian diet.

We undertook a survey of the iodine content of milk samples from supermarkets around metropolitan Sydney in 2001 and 2004. In each year, iodine levels were measured in 13 samples, comprising a range of milk types (including whole, full cream, lite and skim) and brands (including Dairy Farmers, Devondale, Farmdale, Farmland, Perfection, Pura and Woolworths).

Iodine concentrations were highly variable. Median concentrations were 140 µg/L in 2001 (range, 60–220 µg/L) and 195 µg/L in 2004 (range, 66–412 µg/L). Iodine concentrations varied between samples of the same brand and type by up to 100 µg/L. Many samples contained less than 200 µg/L (10/13 in 2001 and 7/13 in 2004).

A 1975 survey of iodine concentration in milk conducted by the Australian Consumers' Association found mean concentrations of 593.5 µg/L and 583 µg/L in NSW and Victoria, respectively.⁵ Because of concerns about iodine toxicity, Food Standards Australia and New Zealand specified an iodine limit of 500 µg/L in the Food Standards Code 1982. The replacement of iodophors by other sanitisers in the dairy industry appears to be the reason for the decrease in iodine content of Sydney milk. The perception that milk is a rich source of iodine is no longer true. A cup (250 mL) of milk a day would provide at most 50–60 µg iodine, approximating a third of the daily requirement for an adult.

We suggest that the reduced amount of iodine in milk is likely to be one of the explanations for the re-emergence of iodine deficiency in Sydney and perhaps elsewhere in Australia. Despite these changes, dairy milk remains an important source of dietary iodine. The iodine content in milk should be monitored.

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1 Eastman CJ. Iodine supplementation: the benefits for pregnant and lactating women in Australia and New Zealand. *Obstet Gynecol* 2005; 7: 65-66.

2 Eastman CJ. The status of iodine nutrition in Australia. In: Delange F, Glinore D, eds. *Iodine deficiency in Europe: a continuing concern*. New York: Plenum Publishing, 1993: 133-139.

3 Li M, Eastman CJ, Waite KV, et al. Are Australian children iodine deficient? Results of the Australian National Iodine Nutrition Study. *Med J Aust* 2006; 184: 165-169.

4 Eastman CJ. Where has all our iodine gone [editorial]? *Med J Aust* 1999; 171: 455-456.

5 Australian Consumers' Association. Adulterated food: is milk a hazard? *Choice* 1975; Sep: 299-302. □

Do women in rural and remote areas need different guidelines for management of low-grade abnormalities found on cervical screening?

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TO THE EDITOR: The incidence of cervical cancer in Far North Queensland (FNQ) is 10 times the national average and the mortality rate five times greater.^{1,2} Of the Australian states, Queensland has the lowest average rate of regular cervical screening (57% of eligible women), and in some FNQ communities rates of less than 40% have been reported.

Cairns Base Hospital (CBH) provides all public colposcopy services in Cairns and throughout Cape York for a population that is largely rural, remote and transient. In 2004, through the outpatients department of CBH, 12 new cases of invasive cancer were diagnosed (with additional advanced cases admitted directly to the surgical services). None of these women had undergone cervical screening in the previous 4 years.

We conducted a three-part study at CBH: a 3-month retrospective study (Feb–Apr 2004) and a 3-month prospective study (Oct–Dec 2004) comparing cytological reports with histological results, and a further study (Oct–Dec 2004) of women who were referred for colposcopy but failed to attend.

In the retrospective study, of 43 new patients with a cytology report of low-grade epithelial abnormality (LGEA) who had histology performed, 19 (44%) had a histological diagnosis of a high-grade epithelial abnormality (HGEA). ("Low-grade cytology" was defined in the 1994 National Health and Medical Research Council [NHMRC] guidelines³ as two consecutive "atypical"