

Necrotising lymphadenitis without granulocytic infiltration: case from Western Samoa

Kikuchi's disease (necrotising lymphadenitis) was first reported by Kikuchi¹ and Fujimoto *et al*² in Japan in 1972. It is characterised by cervical lymphadenitis in young patients, and may simulate malignancy both clinically and histopathologically. Patients present with tender cervical or submandibular lymphadenopathy for a couple of weeks. There are generally no other abnormal physical signs and laboratory findings. The principal microscopical features are effacement of the lymph node architecture, coagulative necrosis to a varying degree, and infiltration of histiocytic cells without polymorphs; coagulative necrosis with an absence of polymorphs is the most striking histopathological finding of this disease.

Since 1972 cases outside Japan have also been reported in North America, the Far East, and the United Kingdom.³ This report concerns a case of Kikuchi's disease encountered in the island of Western Samoa believed to be the first such case report in that country.

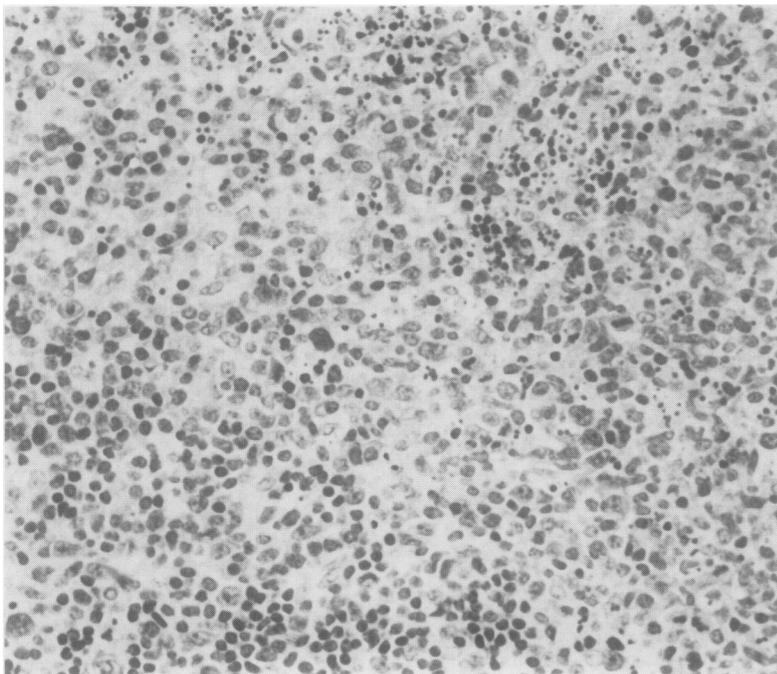
A 25 year old Samoan (Polynesian) woman presented with left cervical and axillary lymphadenopathy which she had had for a few months. She first developed tender left cervical adenopathy, and later a painful swelling in the left axillary. Routine radiological and laboratory investigations showed no other important findings. A Paul-Bunnell test

was negative. Finally, the cervical node was biopsied with the presumptive diagnosis of tuberculosis.

Macroscopically the node was 2 cm in diameter, hard in consistency, with a grey-whitish cut surface. Microscopically the node showed essentially similar features to those mentioned above: complete loss of the nodal architecture with varying degrees of necrosis, nuclear debris, and phagocytic histiocytic cells (figure). There was no neutrophil polymorph infiltrate. Acid fast stain was negative, as was bacteriological culture for tuberculosis. After consultation with the Department of Anatomical Pathology of the Sydney Royal Prince Alfred Hospital, Australia (Dr S McCarthy) the appearances were thought to be those of Kikuchi's disease. The disease is thought to be benign; the aetiology is unknown, although a viral aetiology has been suggested.

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- 1 Kikuchi M. Lymphadenitis showed focal reticulum cell hyperplasia with nuclear debris and phagocytosis. *Acta Haematol Jpn* 1972; 35:379-80.
- 2 Fujimoto Y, Kozima Y, Yamaguchi K. Cervical subacute necrotising lymphadenitis. A new clinicopathological entity. *Arch Intern Med* 1972;30:920-7.
- 3 Ali MH, Horton LWL. Necrotising lymphadenitis without granulocytic infiltration (Kikuchi's disease). *J Clin Pathol* 1985; 38:1252-7.



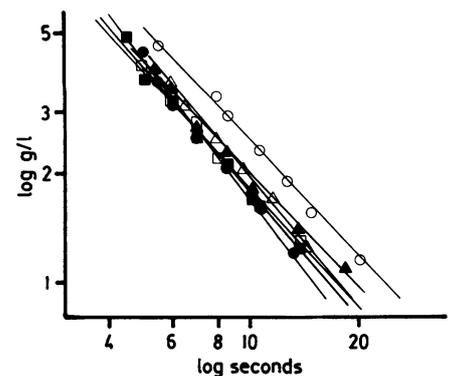
Lymph node biopsy specimens showing necrotic foci and histiocytic cells with phagocytic debris (haematoxylin and eosin).

Fibrinogen standards

In our laboratory fibrinogen concentrations are determined by the method of Clauss¹ using a semiautomated technique (Fibro-System BBL; Becton Dickinson). The precision of our method is high (coefficient of variation of replicate samples <4%). Recently, however, we have noticed a discrepancy between results obtained when

using different commercially available fibrinogen standards.

At present there is no international standard available for fibrinogen which would permit assessment of these standards (GK Cook, National Institute for Biological Standards and Control). We therefore purchased fibrinogen standards from six commercial sources and prepared a standard curve from each of these (figure). A variety of methods



Fibrinogen standard curve (reference standards A = ●; B = ▲; C = ■; D = □; E = △; F = ○).

are used to assign fibrinogen values to these standards, such as "clot weight," Kedjahl protein determination, and Clauss methods.

There was close agreement between standards with one exception. The fibrinogen values determined using this standard produced higher values which could be clinically misleading.

There is a strong case for the production of an international standard for fibrinogen and perhaps guidance on which method to use when assigning values to commercial standards. Meanwhile we would recommend that when producing a standard curve for fibrinogen using the Clauss technique, that at least two (preferably three) commercial standards be used: this will enable standards with discrepant values to be identified.

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- 1 Von Clauss A. Gerinnungsphysiologische Schnell method zur Bestimmung des fibrinogens. *Acta Haematol* 1957;17:237-46.

ANTI-HBc IgM assays and diagnosis of acute hepatitis B

Although a high titre of IgM class antibody to hepatitis B core antigen (anti-HBc IgM) is considered to be diagnostic of recent acute hepatitis B virus (HBV) infection, such an antibody response occurs not only during episodes of acute HBV infection but also in 5% of patients with chronic HBV infection.¹ As anti-HBc IgM antibody titres decline after infection,¹ the disappearance of reactivity in serial serum specimens tested for high titres of this antibody could perhaps be diagnostic of acute infection. Detection of "e" antigen to "e" antibody seroconversion is an alternative method of diagnosis in this situation.¹ Personal observations and published data show that the disappearance of high titres of anti-HBc IgM antibody is of lesser value than the detection of "e" antigen to "e" antibody seroconversion as a diagnostic marker for acute HBV infection.

A study of hepatitis B surface antigen, hepatitis B "e" antigen and "e" antibody, and high titre (titre of $\geq 1/400$) anti-HBc IgM antibody responses by commercial immunoassays in serial sera collected from three patients with acute hepatitis B (table) suggested that seroconversion from "e" antigen to "e" antibody positivity preceded the dis-