ease represents a hyperimmune reaction to an unknown aetiological agent such as a virus.

Our use of the "unqualified term necrotizing lymphadenitis" was criticised by Dr JM Woodruff of the Memorial Sloan-Kettering Cancer Center in a letter to the editor of the *American Journal of Surgical Pathology*.[9] In response to this,[10] I proposed the eponym "Kikuchi's disease," and I am pleased to note the positive reaction to this suggestion.

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Drs Ali and Horton reply as follows:

Clearly, our literature search was not as thorough as we had thought, and we stand admonished!

We stained our material using the napthol-ASD-chloroacetate method, but were unable to identify any convincingly positive cells or debris. Feller *et al*[1] also used this technique but do not mention any positive findings.

The exact nature of the cells around the necrotic foci must remain in doubt. Professor Dorfman's study[2] suggested in the one case examined that the cells staining positively with T cell markers around the necrotic foci were cytotoxic/suppressor T cells, yet Feller *et al*[1] reported these cells to be of the helper/inducer type. Further, there are clearly some T cells that share differentiation antigens with histiocytes,[3] and we found apparent transitional forms between immunoblasts and histiocytes ultrastructurally.[4]

The advantage of the eponymous term "Kikuchi's disease" is that it cannot be reduced to an acronym which would probably be the fate of necrotising lymphadenitis without granulocytic infiltration (NLGI).

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Classification of haemolytic uraemic syndrome

An increasing number of micro-organisms have been implicated in the pathogenesis of the haemolytic uraemic syndrome.[1] This syndrome is characterised by microangiopathic haemolytic anaemia, thrombocytopenia, and renal failure; and seems to be a disorder of platelet and endothelial cell interaction.

In several cases verotoxin, an exotoxin cytopathic for monkey kidney cells (Vero), and neuraminidase, an enzyme chemically similar to verotoxin, have been identified. Of the stool isolates reported in haemolytic uraemic syndrome, various serotypes of *Escherichia coli*,[2] *Shigella dysenteriae serotype 1* (Shiga toxin),[3] and *Campylobacter fetus jejuni*[4] produce verotoxin and cause bloody diarrhoea, a typical prodromal symptom. So far, six cases of childhood haemolytic uraemic syndrome associated with *Streptococcus pneumoniae*, a neuraminidase producer, have been described.[5] Case reports of different bacterial and viral isolates in haemolytic uraemic syndrome and thrombotic thrombocytopenic purpura (an analogous adult syndrome) continue to flourish. There is often no attempt to identify the production of these important toxins, however, which may signify a common aetiology for the different micro-organisms implicated in the pathogenesis of haemolytic uraemic syndrome.

Incubation of sterile culture filtrates of verotoxin producing *E coli* with normal plasma will result in potent platelet aggregating activity.[6] This is dependent on the platelet membrane glycoproteins IIB and IIIA. The premature release of unusually large factor VIII multimers, seen in haemolytic uraemic syndrome plasma,[7] may also be an important mode of pathogenesis and result from damage to vascular endothelium by verotoxin. Neuraminidase, produced by a wide variety of micro-organisms, may have several pathogenic mechanisms. Direct platelet aggregating activity; deasialation of factor VIII to produce platelet aggregation; and exposure of the Thomsen cryptantigen of platelets, red cells, and vascular endothelium have all been postulated.

Further work is necessary to define the mode of action of verotoxin and neuraminidase in producing this disorder of platelet and endothelial cell interaction. The pathogenic role of these exotoxins, however, is becoming increasingly obvious. Early detection of free faecal verotoxin and the identification of neuraminidase and verotoxin producing micro-organisms are essential steps in the diagnosis and management of haemolytic uraemic syndrome. Classifying haemolytic uraemic syndrome into verotoxin, neuraminidase, and non-exotoxin cases could provide valuable clinical information about this heterogeneous condition. The increased morbidity recently observed in childhood haemolytic uraemic syndrome[8] and the difference in prognosis of epidemic and sporadic cases may be due