

with the DF is able to separate thalassaemia trait from iron deficiency: 50 patients have an MCV of less than 75 fl; a high platelet count (more than $400 \times 10^9/l$) is found only in the cases with iron deficiency; when the platelet count is normal or low, the discriminant function differentiates the iron deficiency (positive DF) from the thalassaemia trait (negative DF). So we were able to identify 26 of 26 cases with iron deficiency and 17 of 19 cases with β thalassaemia trait.

In conclusion, the platelet count associated with the discriminant function may serve as a useful screening tool to detect the majority of patients heterozygous for thalassaemia or deficient in iron.

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A case of small-cell Sézary syndrome with null-cell features (Goldstone *et al.*, *Journal of Clinical Pathology*, 1976, 29, 848)

This case was originally investigated and treated at the Royal West Sussex Hospital, Chichester. He presented in December 1974 with a history of a rash on the thighs. A skin biopsy was reported as the plaque stage of mycosis fungoides (Dr Knowles). He symptomatically improved on therapy with Dimotane and Dermovate (1 in 3) but soon relapsed and was admitted to hospital in February 1975. At this time the rash (which was itchy) was

widespread over the trunk (back and front) and legs. A second biopsy was again reported as typical of mycosis fungoides. There was no splenomegaly nor lymphadenopathy. Haematological investigation showed the white count to be raised at $29 \times 10^9/l$ with 70% small, mature-looking lymphocytes. Haemoglobin level and platelet counts were normal. Bone marrow aspiration did not show any lymphocytic marrow infiltration.

Histological examination of a normal sized lymph node, removed from the groin, showed the presence of a well-differentiated, diffuse, lymphocytic lymphoma.

The surface characteristics of the peripheral blood lymphocytes were studied in March 1975; 70% were null cells, 20% T cells, and 10% B cells. Transmission electron microscopy showed the cells to be Sézary cells. Over the next three months the skin condition remained much the same but the WBC count rose progressively until, by the end of May, it had reached $58 \times 10^9/l$ with mature lymphocytes predominating. On 29 May 1975 treatment was started with chlorambucil, 6 mg daily. The total WBC dropped steadily over the next eight weeks and, towards the end of this time, the skin condition suddenly improved. Just before this occurred arrangements had been made for the patient to be transferred to Cambridge for consideration of whole body irradiation.

Although this additional information does not affect the conclusions of Goldstone *et al.*, it does illustrate the diagnostic problems associated with this bizarre disorder. We feel your readers will find it interesting.

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Occurrence of e antigen in acute hepatitis B

The recent paper by Gibson and Ruparelia (*Journal of Clinical Pathology*, 1977, 30, 925-927) suggests conclusions which are quite unjustified by its evidence. Of

44 patients with hepatitis B, only six were found e antigen positive, and only two became HB_sAg carriers (why substitute 13.6% and 4.6% for integers in the summary?). If presence of e antigen and becoming a carrier of HB_sAg were independent, the probability of their coincidence in this group would be about 0.006, so the finding reported proves nothing. In addition, the authors acknowledge that they may have failed to detect e antigen in some of their patients and did not do so in two of three who eventually formed anti-e. Their conclusions about the duration of HB_s antigenaemia are also open to criticism for the same reasons.

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The author comments as follows:

In reply to Mr Davey's letter of 29 November 1977, I should like to make the following comments: the results in our paper are straightforward and although no firm conclusions are reached, those that are, are self-evident. No claim of independence is made. Of six cases where e antigen was found, none became a carrier of HB_sAg, indicating that the presence of e early in the course of acute HB_sAg is not necessarily of prognostic value. Unfortunately, blood samples from the early acute phase of illness are difficult to obtain, and of 90 cases of acute hepatitis studied, only 44 satisfied the necessary criteria to be included (ie, first serum sample taken within one month of jaundice onset and the final sample cleared of HB_sAg or when it became evident that the carrier state had developed). Those not included were patients whose serum samples were taken very late during the HB_s antigenaemia and these were all e negative. The importance of this report is that in cases where blood samples were available from within one week of onset of jaundice, e was detected in these first specimens (but not in the following specimens) in 6 of 15 cases, while in the remaining 29 cases where the first specimen was obtained following one week of the onset of jaundice, e was not detected in any of the samples. The difference between these two groups is very significant.

Berquist *et al.* (1976) and Frösner *et al.* (1977) have reported similar results and reached similar conclusions. Therefore,