Clinical Section

President Harold Ellis MCh

Meeting May 14 1971
(continued from December 'Proceedings', *p 1310)*

Cases

Rectal and Colonic Ulceration in Behçet's Disease

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(Westminster Hospital, London SW1)

Miss M E, aged 25. Laboratory technician

History: September 1969, attended surgical outpatients complaining of painful defæcation and the passage of stool streaked with blood, associated with malaise and weight loss.

On examination: Aphthous ulcers on uvula; acute fissure-in-ano; anterior rectal ulcer 3 cm in diameter with nonspecific histology.

Investigations: Hb 9·6 g/100 ml; ESR 84 mm in 1 hour (Wintrobe); WBC 5,400/mm³ (normal differential). Serum electrolytes and urea were normal. Plasma proteins: total 6·5; albumin 2·9; α_1-globulin 0·4, α_2-globulin 0·8, β-globulin 0·8, γ-globulin 1·6 g/100 ml; fibrinogen 611 mg/100 ml. WR and Kahn negative.

Progress: Two days after the examination under anæsthetic she developed punched out necrotic ulcers on her neck, ear, right scapula region and tongue. The appearance of these lesions was compatible with a diagnosis of Behçet's disease.

She improved slowly and was fairly well until two months later, when she developed peritonitis due to a perforating ulcer at the apex of the sigmoid colon. This was widely excised and a colostomy performed. The histology showed chronic inflammation and ulceration of all layers of the colon similar to the rectal biopsy and compatible with a diagnosis of Behçet's disease.

She was discharged with the rectal and skin ulcers healed. An attempt made six months later to close her colostomy failed because of a troublesome fistula.

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Comment

Behçet's disease is characterized by relapsing inflammation in the eye and ulceration of the mouth and genital tract. Not all of these features appear at the same time. More widespread manifestations such as arthralgia, erythema nodosum, pyrexia and thrombophlebitis are recognized. Diseases such as ulcerative colitis or Crohn's disease may often be associated with skin lesions (Mountain 1970). Several authors have described the association of Behçet's disease and gastrointestinal symptoms (Boe *et al.* 1958, Jensen 1944, Ramsay 1967), and we have seen a similar patient who was too ill to attend this meeting. Ulceration with perforation of the colon has been only rarely reported (Tsukada *et al.* 1964). Now that the more general manifestations of Behçet's disease are well recognized, more cases will be diagnosed in general medical and surgical clinics, and it should be borne in mind as one of the less common causes of rectal and colonic ulceration.

(A fuller report will appear in the *British Journal of Surgery*)

REFERENCES
Boe J, Dalgaard J B & Scott D
(1958) *Amer. J. Med.* **25**, 857
Jensen J S (1944) *Ugeskr. Læg.* **106**, 176
Mountain J C (1970) *Gut* **11**, 18
Ramsay C A (1967) *Proc. roy. Soc. Med.* **60**, 185
Tsukada S, Yamazaki T, Iyo S, Nishio I, Hashimoto K & Matubara F
(1964) *The Newest Medicine (Japan)* **19**, 1533

Mr M Naunton Morgan *(Westminster Hospital, London SW1)* said that it had initially been thought that this patient might have Crohn's disease; however, repeated rectal biopsies and biopsies from the colostomy showed no evidence of this. The fistula which followed closure of the colostomy was, he

thought, due to technical reasons and not to the disease process. Unfortunately no biopsies were performed on the skin lesions. He felt sure that if this case was one of Crohn's disease at least one giant cell would have been seen in the biopsies.

Professor Harold Ellis (*Westminster Hospital*) said that until something was known about the etiology of these diseases, at present identified only by their eponyms, it was often extremely difficult to distinguish between Behçet's disease with colonic involvement and ulcerative colitis complicated by pyoderma gangrenosa.

Dr Graham Neale (*Royal Postgraduate Medical School, London W12*) said that the skin lesions of this patient had features suggestive of pyoderma gangrenosa, a condition described in 3 patients who had ulceration in skin folds (Mountain 1970, *Gut* **11**, 18).

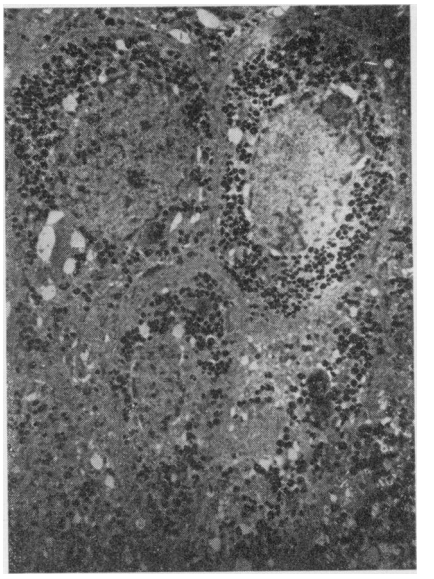

Fig 1 *Electron microscopy of liver metastasis showing electron-dense granules about 200 nm in diameter consistent with granules containing 5-hydroxytryptamine*

Carcinoid Syndrome Treated with Streptozotocin

F I Iweze FRCS, M Owen-Smith FRCS
and J Polak MD (Argentina)
(for R H Franklin FRCS)
(*Royal Postgraduate Medical School, London W12*)

Mrs V H, aged 57. Housewife

History: 1964, hysterectomy and bilateral salpingo-oophorectomy for metropathia. Two-year history of severe hot flushes and palpitations provoked by exertion, heralded by throbbing in face and scalp, and quite different from attacks of menopausal flushes which she had had for ten years. Severe watery diarrhœa and borborygmi for one year; palpable mass in right hypochondrium for two months.

On examination: She was anæmic, had a pulmonary systolic murmur, a palpable hard, non-tender gall-bladder mass, with liver enlarged to 9 cm below costal margin.

Investigations: Oral cholecystogram: no excretion of dye. Liver scan: Filling defect in region of gall-bladder bed. Chest X-ray, electrocardiogram, and phonocardiogram – normal.

Laparotomy at Kingston Hospital, and six weeks later at Hammersmith Hospital, revealed multiple circumscribed whitish tumour deposits about 1·5 cm in diameter in both lobes of an enlarged liver. The gall-bladder was thickened, hard and infiltrated by similar tumour but did not contain calculi. The gall-bladder tumour was thought to be inoperable and a biopsy of a liver secondary taken.

Histology: The cells of the liver metastasis were shown to possess the characteristics of an 'apudoma' (Szijj *et al.* 1969, Pearse 1969), secreting 5-hydroxytryptamine, i.e a carcinoid tumour (Figs 1 & 2). Her 24-hour urine excretion of 5-hydroxyindole acetic acid was 315 mg (normal 3–17 mg).

Treatment and progress: In view of preliminary reports that streptozotocin may induce at least temporary remission in patients with the carcinoid syndrome (C Moertel and G Canellos, personal communication), the patient was given two

Table 1
Excretion of 5-hydroxyindole acetic acid during treatment with streptozotocin

	24-hour urine excretion of 5-HIAA (mg)	
27.10.70	280	
28.10.70	272	Streptozotocin started
29.10.70	450	
30.10.70	436	
31.10.70	315	
1.11.70	260	Streptozotocin stopped
3.11.70	275	
4.11.70	200	
29.1.71	330	Liver scan shows at least 2 new filling defects
6.2.71	360	
7.2.71	325	
8.2.71		Second course of streptozotocin started