SHORT REPORTS

Platelet count: a simple measure of activity in Crohn's disease

Thrombocytosis may occur in inflammatory bowel disease and probably represents a non-specific response to inflammation.^{1,2} We have observed raised platelet counts in patients with active Crohn's disease, and we therefore investigated whether the platelet count is a useful index of disease activity.

Patients, methods, and results

We studied 57 patients with Crohn's disease attending a gastrointestinal clinic; diagnosis was based on conventional criteria. The patients comprised 27 men and 30 women aged (mean $\pm$ 1SD) 37 ± 14 years. Mean duration of disease was nine years. Distribution of disease radiologically was diffuse small bowel (nine), ileocaecal (24), ileocolonic (eight), colonic (13), and anorectal (three). Thirty patients had undergone resections, and recurrent disease was present in 22. Twenty six patients were receiving prednisolone and 15 sulphasalazine. Disease activity was measured by a simple clinical index,³ scores of 5 or more representing active disease.

Platelets were measured in all patients by an automated technique using a Clay Adams Ultra Flo 100 whole blood platelet counter (normal range $150-450 \times 10^9/l$). In most patients serum orosomucoid concentrations were measured by rate reaction nephelometry with an automated Beckman rate nephelometer (normal range < 1.0 g/l). Statistical analysis was by unpaired Student's *t* test and Pearson's moment product correlation coefficient (*r*).

Platelet counts were significantly higher in patients with clinically active disease ($511 \pm 177 \times 10^9/l$; $n=19$) than in those with inactive disease ($379 \pm 123 \times 10^9/l$; $n=38$) ($p<0.01$). Counts were similar irrespective of whether patients had undergone resection or had recurrence and irrespective of disease distribution and drug treatment. There was a strong positive correlation between platelet counts and serum orosomucoid concentrations (figure). All patients with platelet counts greater than $500 \times 10^9/l$ had serum orosomucoid concentrations greater than 1.0 g/l.

Platelet counts and serum orosomucoid concentrations in patients with Crohn's disease ($r=0.711$, $p<0.001$, $n=47$). Dotted lines indicate upper limits of normal range. Clinical activity score <5 =inactive disease; $5-8$ =active disease; >8 =very active disease.

Comment

Orosomucoid concentrations are thought to be one of the best discriminatory variables of disease activity in patients with Crohn's disease,⁴ and this study showed a strong relation between this variable and the platelet count. Others have shown a correlation between platelet count and the erythrocyte sedimentation rate, white cell count, albumin concentration, and haemoglobin concentration and have concluded that thrombocytosis reflects active disease.⁵ We

suggest that platelet counts provide a useful measure of disease activity and may be used to supplement clinical assessment especially when serum orosomucoid concentrations and the erythrocyte sedimentation rate are difficult to obtain.

¹ Morowitz DA, Allen LW, Kirsner JB. Thrombocytosis in chronic inflammatory bowel disease. *Ann Intern Med* 1968;**68**:1013-21.

² Lam A, Borda IT, Inwood MJ, Thomson S. Coagulation studies in ulcerative colitis and Crohn's disease. *Gastroenterology* 1975;**68**:245-51.

³ Harvey RF, Bradshaw JM. A simple index of Crohn's disease activity. *Lancet* 1980;**ii**:514.

⁴ Andre C, Descos L, Landais P, Fermanian J. Assessment of appropriate laboratory measurements to supplement the Crohn's disease activity index. *Gut* 1981;**22**:571-4.

⁵ Talstad I, Rootwelt K, Gjone E. Thrombocytosis in ulcerative colitis and Crohn's disease. *Scand J Gastroenterol* 1973;**8**:135-8.

(Accepted 22 February 1983)

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Cerebrospinal fluid rhinorrhoea after cocaine sniffing

By far the most common cause of cerebrospinal fluid rhinorrhoea is trauma. Non-traumatic cases are unusual, and an underlying cause may often be found.¹ This group may be subdivided into two categories, one with high intracranial pressure—commonly associated with tumours, especially hypophyseal—and one with normal pressure, often occurring in the presence of congenital anomalies and focal atrophy in the cribriform plate or sella turcica areas.² The last condition may be due to ischaemia. Iatrogenic causes form a further important group. The danger of CSF rhinorrhoea is recurrent meningitis, hence surgical closure is recommended in persistent cases. We report a case of cerebrospinal fluid rhinorrhoea which we believe was related to prolonged cocaine sniffing. This is a cause of cerebrospinal fluid rhinorrhoea which has not been reported before.

Case report

A 34 year old man presented to the casualty department at this hospital with a history of nasal discharge for six days and malaise for five. The discharge was of clear, salty fluid and stained the pillow at night. It was accompanied by total anosmia. These symptoms were noticed 24 hours after he had taken intranasal cocaine, which, according to him was of "high quality." Unfortunately a sample was not available for chemical analysis. He admitted to sniffing cocaine seven times a year for the previous 19 years. There was no history of head injury.

On admission he looked well. He was afebrile, with a nasal voice and clear fluid discharging from each nostril. Bilateral anosmia was present, but there were no other focal neurological signs and no meningism. General examination was normal. Routine blood tests and cultures gave negative results. Skull x ray films showed no abnormality. An otolaryngological examination confirmed that the fluid was coming from the region of the cribriform plate. The glucose concentration of the nasal discharge was 1.3 mmol/l (23.4 mg/100 ml). His cerebrospinal fluid was examined later and was found to be normal with a glucose concentration of 2.5 mmol/l (45 mg/100 ml). Computed tomography of the head showed fluid in the right ethmoid and right maxillary sinuses and an opacity in the right side of the nose, suggesting a leak through the ethmoid bone. Tomograms of the cribriform plate suggested a loss of bone substance, although the introduction of metrizamide into the cerebrospinal fluid failed to show a leak.

As the leak persisted surgical intervention was undertaken. A bifrontal craniotomy was performed, and both olfactory nerves examined. On the right, in particular, the olfactory nerve was covered by inflammatory changes and looked oedematous. As the patient remained totally anosmic the right olfactory nerve was examined histologically. Both cribriform plates were inspected. There was no gross defect in the dura, but the small perforations for the olfac-