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.

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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.

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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-

tory nerve endings were larger than usual, particularly on the right. The whole of the bone of the cribriform plate was paper thin and mobile. The defect was closed with a fascia lata patch obtained from the thigh. Post-operative recovery was uneventful. Histology of the olfactory bulb showed chronic inflammatory change and gliosis.

Comments

Cocaine is a psychologically addictive drug and its abuse is greatly increasing.³ It is derived from the coca leaf and is used as a local anaesthetic in otolaryngology because of its useful vasoconstrictor activity. The mechanism of this effect is by inhibiting reuptake of catecholamines into the sympathetic nerve ending, thus potentiating their effect.⁴ Chronic abuse of the drug by intranasal sniffing may lead to chronic inflammatory changes and perforation of the nasal septum, owing to the effects of intense vasoconstriction.⁵ Similar chronic damage probably resulted in the changes in the cribriform plate and olfactory nerves in our patient, which ultimately lead to a leak of cerebrospinal fluid.

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Phaeochromocytoma: an unusual cause of chest pain

High catecholamine concentrations due to a phaeochromocytoma are known to cause myocarditis and myocardial fibrosis.¹ Electrocardiographic findings suggestive of myocardial infarction with minimal serum enzyme changes have been attributed to a toxic myocarditis in view of the rapid reversibility of these changes after treatment with adrenergic blocking agents.²⁻³ We describe a patient who presented with myocardial infarction, hypertension, and, later, anginal pain. We suggest that coronary artery spasm was the underlying cause due to the high catecholamine concentrations.

Case report

A 38 year old housewife was admitted to hospital in March 1976 because of severe central chest pain. Pulse was 80/min regular and blood pressure 190/140 mm Hg. An electrocardiogram showed appreciable ST segment elevation in the anteroapical and inferior leads. Twenty four hours after admission serum creatine kinase activity was 1500 IU/l (normal 0-130) and serum lactic dehydrogenase activity 1721 IU/l (normal 115-465).

During the first 24 hours she developed several episodes of severe central chest pain. Monitor printouts (figure) showed considerable variable ST segment elevation. After recovery from the myocardial infarction she was discharged taking propranolol and triamterene, blood pressure being 140/100 mm Hg. One month later the blood pressure was 190/135 mm Hg. After a phentolamine test the blood pressure became normal. Twenty four hour urinary catecholamine excretion was normal. Anginal pain was then

noted, and she was referred for further investigation. Catecholamines in a 24 hour urine collection were raised. This was discounted as it was thought to be due to a drug interaction.

During 1978 she had three admissions to hospital with severe central chest pain and one episode of paraesthesia in the left arm and hand. In 1979, when taking prazosin, acebutolol, and triamterene, she underwent an uneventful cholecystectomy for gall stones.

In 1982, after further anginal episodes, she was readmitted. She was taking prazosin, acebutolol, and hydrochlorothiazide, and her blood pressure was 120/70 mm Hg. Left ventricular angiography showed a discrete apical aneurysm with clot in the aneurysmal sac. The coronary arteries were normal. Three measurements of 24 hour urinary catecholamine excretion were 2.6, 2.9, and 7.03 μ mol (normal <0.65 μ mol/24 h). Computed tomography of the adrenals showed a lesion in the left adrenal measuring 4x4 cm and lying behind the pancreas in front of the renal sinus. Renal arteriography showed the lesion to be lying on the anterior surface of the left kidney, and appearances were suggestive of a phaeochromocytoma. The tumour was excised under alpha-blockade and confirmed pathologically as a phaeochromocytoma. She made an uneventful recovery and was discharged taking warfarin because of the apical aneurysm; blood pressure was 120/80 mm Hg without antihypertensive treatment. Six weeks post-operatively she had no chest pain.

Changes in ST segments associated with chest pain (monitor lead). (a) 3 15 am on 31 March 1976; blood pressure 210/150 mm Hg; no pain. (b) 4 00 am; blood pressure 170/130 mm Hg; severe chest pain with appreciable ST segment elevation; intravenous diamorphine given. (c) 4 30 am; massive ST segment elevation; (d) 8 00 am; ST segment elevation settling; (e) 12 noon; ST segment elevation similar to (a).

Comment

Peripheral vascular ischaemia severe enough to cause gangrene has been described in association with phaeochromocytoma.³ Transient cortical blindness and transient neurological defects have been described,³ but so far as we are aware coronary artery spasm has not been reported in patients with a phaeochromocytoma. Hausmann and Getzowa⁴ were the first to describe myocardial degenerative and fibrotic lesions associated with tumours of the adrenal medulla, which were probably phaeochromocytomas. Van Vliet *et al* described "active catecholamine myocarditis" in a high proportion of patients with phaeochromocytomas proved at necropsy.¹ The cardiomyopathy associated with phaeochromocytoma subsided clinically after resection of the tumour.¹ Electrocardiographic changes of myocardial infarction have been shown in patients with phaeochromocytoma, but subsequent necropsies have shown only severe myocarditis or diffuse myocardial damage.²

In this patient, who presented with a myocardial infarction and severe hypertension, the cause of the myocardial infarction and subsequent angina was probably severe coronary artery spasm as shown by appreciable variation in ST segments. Subsequently, a left ventricular apical aneurysm with normal coronary arteries were shown. Symptoms ceased on removal of the left adrenal phaeochromocytoma.