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SHORT REPORTS

Toxocariasis and eosinophilic meningitis

Toxocariasis is usually manifested as visceral larva migrans in young children with pica.[1] Lesions in the eye are more common in older children and adults,[1 2] and rarely other parts of the central nervous system may be affected.[2] A benign, self limiting meningitis has not been described and we therefore report such a case.

Case history

A previously healthy 11 year old girl was referred by her general practitioner with a 10 day history of worsening neck stiffness and back pain. She had initially presented with severe bilateral frontal headache and dizziness but these symptoms had resolved within 48 hours after beginning treatment with paracetamol.

On examination the child looked alert and well and was not feverish. There was pronounced meningism and she complained of severe pain at the level of T12-L1, accentuated by movement. Kernig's sign was positive and both optic discs were blurred. There was generalised hyperreflexia. The rest of the physical examination, routine blood tests, and radiography showed nothing unusual. An urgent CT scan showed no abnormality. Lumbar puncture the day after admission yielded 150 white cells, reported initially as being predominantly lymphocytes with normal protein and glucose. In view of the atypical history Romanovsky staining was performed on a cytospin of cerebrospinal fluid. This showed that 30% of the cells were eosinophils and that the remainder were lymphocytes. Culture and serology for common viral and bacterial infection were unhelpful. Two days after admission an eosinophilia of $1{\cdot}3\times10^9$/l was noted, which persisted to follow up at six weeks.

The patient recovered spontaneously over the next three days and was sent home. Results of serological screening for toxocaral infection suggested active infection (see table), both acute and convalescent serum samples having appreciable titres. Anti-A agglutinin titres were also raised but fell during convalescence, were IgG only, and the patient had never had a blood transfusion, all points suggestive of acute toxocaral infection. She denied pica but both neighbours had recently acquired puppies, ensuring a plentiful supply of toxocara ova.

She was treated with diethylcarbamazine 8 mg/kg for 10 days and tolerated the treatment well. She continued to be asymptomatic nine months after presentation.

Comment

This case was unusual in its presentation and is instructive in the method of diagnosis. Pronounced eosinophilia in the cerebrospinal fluid is rare in Britain but there are many recognised causes.[3] Other

reports have emphasised the severity of neurological and systemic disorder in patients with toxocaral meningoencephalitis.[1 2 4] Apart from neck stiffness our patient was surprisingly well with no evidence of encephalitis. Her diagnosis might have been that of an atypical viral meningitis had Romanovsky staining not been performed. We think that this investigation may be useful in culture negative meningitis when there are no signs of viral infection.

Toxocara enzyme linked immunosorbent assay titres using T canis excretory-secretory products as antigen

	Acute serum	Convalescent serum
IgG	1/400	1/200
IgE	1/320	1/320
Anti-A agglutinins:		
IgM A_1	1/16	1/16
IgM A_2	1/8	1/8
IgG A_1	1/8192	1/4096
IgG A_2	1/8192	1/2048

Note: erythrocytes 0^+.

We decided to give specific antitoxocaral treatment because of the risk of ocular lesions or recurrence in the central nervous system,[1] although there is little evidence for the efficacy of diethylcarbamazine or thiabendazole[1 2] and there is the risk of release of larval antigen and reaction to this.

This is the first reported case of a mild, self limiting eosinophilic meningitis due to *Toxocara canis*, but overt manifestations of toxocaral infection are uncommon in Britain. It is interesting to speculate how common this presentation of toxocariasis may be in view of the fact that over 2% of the population in Britain have seroconverted.[5]

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Size of pancreas in diabetes mellitus: a study based on ultrasound

We have shown that exocrine pancreatic deficit is common in diabetes and that the degree of this deficit parallels the endocrine deficit.[1-4] From necropsy data we also know that the size of the pancreas in patients with insulin dependent diabetes is smaller than in normal people.[5] In order to confirm this observation in insulin dependent diabetes[5] and to determine whether pancreatic size is also altered in non-insulin dependent diabetes mellitus we studied the pancreatic size in patients with diabetes mellitus using ultrasound techniques.

Patients, methods, and results

Sixty adult diabetic patients were investigated: 22 had insulin dependent diabetes (group 1) and 19 non-insulin dependent diabetes (group 2) and 19 were non-ketotic patients who had to be given insulin because of inadequacy of diabetic control with oral hypoglycaemic agents (group 3). Nineteen healthy controls were also studied.

All scans were performed by one of us (LAB), using a real time linear array system (Picker LS 3000).

The patients and controls were scanned in the morning after an overnight fast. Scans were performed with the patients supine and erect. A more complete and clearer visualisation of the pancreas was achieved by scanning with the patient erect. The head (defined as the area medial to the superior mesenteric vein) and the body of the pancreas were measured separately, since these were often visualised to best advantage in different views. As the head is often oriented in the longitudinal plane parallel to the inferior vena cava, measurements were made in this plane below the portal vein as well as in the transverse or oblique plane (taking the midpoint of the confluence of the superior mesenteric and splenic veins as the marker point). In normal subjects the longitudinal section of the head was frequently larger than the transverse sections. This did not occur in any of the diabetic patients. The tail of the pancreas was well seen as it passed anterior to the left kidney, but the more distal portion extending into the splenic hilum (which represents a very small part of the pancreatic mass) was rarely seen.

The scans were recorded on photographic paper. The outline of the pancreas was mapped out and the areas computed using a Numonics Graphic Analyzer (Numonics, Japan).

Both the head and the body of the pancreas in patients of group 1 and 2 were noticeably smaller than in the controls (table). The head and body of the pancreas in patients of group 2 were significantly larger than in group 1.

Total area of pancreas and area of head and body of pancreas (in cm^2) in diabetic patients and controls. Values are medians (ranges in parentheses)

Group	No of subjects	Total area	Area of head	Area of body
1	22	8·9*† (4·6-14·7)	3·2*† (1·1-5·7)	5·7*§ (2·0-11·2)
2	19	12·9‡ (6·6-17·8)	5·7‡ (2·2-11·8)	7·8‡ (4·4-11·8)
3	19	11·9‡ (6·7-17·3)	4·8‡ (2·5-6·4)	6·7‡ (3·4-13·6)
Controls	19	18·7 (13·0-26·1)	8·8 (6·2-11·1)	10·4 (7·1-19·8)

*Compared with controls $p < 0.001$.
†Compared with group 2 $p < 0.002$.
‡Compared with controls $p < 0.002$.
§Compared with group 2 $p < 0.05$.

In group 3 the sizes of the head and body of the pancreas were intermediate between those in groups 1 and 2.

There was no correlation between size of the pancreas and body weight or duration of diabetes.

Comment

These results show that the pancreas is significantly smaller in diabetic patients than in healthy controls. Furthermore, patients with insulin dependent diabetes have significantly smaller pancreases than patients with non-insulin dependent disease. Non-ketotic patients whose diabetes was not controlled with maximum doses of oral hypoglycaemic agents and who required insulin had pancreases intermediate in size between those in the other two groups. This is the first study to document these changes systematically. The pattern of diminution in the size of the pancreas in diabetes parallels the impairment of exocrine function previously described by us. Thus patients with insulin dependent diabetes have the lowest serum concentrations of pancreatic enzymes. Patients with non-insulin dependent disease have marginally reduced serum pancreatic enzyme values, while patients with non-ketotic disease who require insulin because of inadequate control with oral hypoglycaemic agents have serum pancreatic enzyme values between those of the other two groups.[1-4]

Pancreatic enzyme values at the time of clinical onset of insulin dependent diabetes mellitus indicate that in these patients exocrine pancreatic reserve may be diminished at the time of presentation, suggesting that exocrine damage occurs not as a long term complication of and sequel to diabetes but probably as a consequence of the process which caused the pancreatic islet and β cell damage.[4] The factors responsible for the shrinkage of pancreas through a reduction of exocrine tissue may be immunological (lymphocytic infiltration has been observed); genetic; subclinical viral pancreatitis; or lack of trophic effect of insulin on exocrine pancreas. The last is particularly relevant to non-insulin dependent diabetes.

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