Fibronectin in children with diabetes mellitus

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SUMMARY Fibronectin plasma concentrations were determined in 28 children with type I diabetes mellitus and 22 healthy children. No statistically significant difference was observed between the fibronectin concentrations in diabetic and non-diabetic children. Even in children with poor glycaemic control the fibronectin concentrations (glycosylated haemoglobin greater than 10%) were not significantly higher.

Fibronectin, a 440,000 dalton glycoprotein, is a substrate for thrombin, plasmin, and activated fibrin stabilising factor (factor XIII). acting as a non-specific opsonin, plasma fibronectin potentiates reticulo-endothelial clearance of intravascular debris to maintain microvascular integrity and vascular permeability.

Both raised and normal fibronectin concentrations have been described in non-ketoacidotic adult patients with type I diabetes mellitus, \( ^2 \) \( ^3 \) while low fibronectin concentrations have been observed in diabetic ketoacidosis. \( ^4 \) A possible relation between high fibronectin concentrations and diabetic microangiopathy has been suggested.\( ^2 \) \( ^3 \)

To our knowledge fibronectin concentrations have not been studied in diabetic children. We have, therefore, measured and compared plasma fibronectin concentrations in children with type I diabetes mellitus and healthy children.

Patients and methods

The diabetic group consisted of 28 patients (mean age 12 years, range 11.1 to 13.9) and 22 healthy children served as a reference group (mean age 11.8 years, range 10.0 to 13.8). There was no difference between the groups with regard to sex distribution, age, weight, and height. None of the children had exercised before blood sampling and none were receiving any medications, except insulin in the diabetic group. Blood samples were obtained by venipuncture after an overnight fast and before morning insulin. Fibronectin was determined by an immunoturbidimetric assay as described by Saba \( et \ al. ) \( ^5 \) Calibration was done using standard plasmas containing 1250, 2500, 5000, and 7500 \( \mu g/l \). Glycosylated haemoglobin (expressed as a percentage of haemoglobin (rel %)) was measured by a micro-column technique (BIORAD).

For statistical analysis Kruskal-Wallis test, Mann-Whitney test, and Spearman rank correlation were used.

Results

The plasma concentrations of fibronectin for both groups are shown in the Figure. The values in diabetic children were not significantly different

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**Figure** Fibronectin plasma concentrations (\( \mu g/l \)) in diabetic children and healthy controls.
from those of normal children (mean 2250, range 2000 to 3300 μg/l v mean 2750, range 2100 to 3300 μg/l respectively). Obviously, serum glucose concentrations and values of glycosylated haemoglobin were significantly higher in the diabetics than in controls (serum glucose mean 10.2 mmol/l, range 1.8 to 19.9 mmol/l v mean 4.7 mmol/l, range 4.1 to 5.5 mmol/l respectively and glycosylated haemoglobin mean 10.0 rel %, range 6.3 to 12.6 rel % v mean 5.6 rel %, range 4.6 to 6.3 rel % respectively). In neither group was there a difference between boys and girls in respect of plasma fibronectin concentrations. Even when the diabetic children were divided into three groups according to their glycosylated haemoglobin concentrations (group I less than 8 rel %; group II 8 to less than 10 rel %; group III greater than 10 rel %), the plasma fibronectin concentrations were not significantly different (Table).

**Discussion**

Vascular endothelial cells are the major source of plasma fibronectin. Fibronectin is also found in platelets and is partially released into the surrounding environment after reaction of platelets with thrombin or collagen.1 Schwarz2 described normal fibronectin concentrations in non-ketoacidotic adult patients with type I diabetes mellitus whose glycosylated haemoglobin values did not exceed 10 rel %, but increased concentrations in those with a glycosylated haemoglobin value greater than 10 rel %. He assumed that the high fibronectin concentrations in the latter group might be caused by an increase in endothelial production as well as an increased release by thrombocytes. Furthermore, he concluded that fibronectin could be of some importance in the development of diabetic microangiopathy. Seghieri et al3 found raised concentrations of plasma fibronectin in insulin treated adult diabetics suffering from retinopathy compared with those without retinopathy. He also suggested a relation between diabetic microangiopathy and raised fibronectin concentrations.

Our data indicate that fibronectin plasma concentrations in diabetic children—even in those with poor control—are no different from those of normal children. Whether the duration of diabetes is an important factor in the changes in fibronectin concentrations in adult diabetics is unknown. Unfortunately the duration of the disease was not mentioned in the report of Schwarz et al.2 In our study there was no correlation between the duration of diabetes and the plasma fibronectin concentrations. Nevertheless, this matter awaits further investigation in a longitudinal study.

Alexander et al4 described reduced plasma fibronectin concentrations in severely hyperglycaemic ketoacidotic adults which returned to normal after adequate insulin treatment. His data correspond with the finding that the plasma fibronectin concentration falls during fasting and returns to normal with feeding. As none of our children had diabetic ketoacidosis their normal concentrations of fibronectin are to be expected.

In general, the fibronectin concentration seems to reflect metabolic and nutritional changes. Low plasma fibronectin measurements in adults with trauma, disseminated intravascular coagulation, or sepsis have been reported.1 There is also an alteration in fibronectin with age: children between 1 and 12 months of age have significantly lower concentrations than those aged between 1 and 15 years.6

In contrast with findings in adults, no consistent sex difference was observed among diabetic or control children. This is in agreement with a previous report on a paediatric population which suggested that hormonal influences might account for the differences in adults.6

**References**


**Table Fibronectin concentrations in relation to glycosylated haemoglobin values in diabetic children**

<table>
<thead>
<tr>
<th>Group</th>
<th>Fibronectin</th>
<th>Glycosylated haemoglobin</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Group I</td>
<td>Group II</td>
</tr>
<tr>
<td></td>
<td>(n=4)</td>
<td>(n=11)</td>
</tr>
<tr>
<td>Median (range)</td>
<td>7-4 (6-3-7-6)</td>
<td>8-6. (8-1-9-9)</td>
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</tbody>
</table>

*Not significant.*
Gastric trichobezoar associated with transient pancreatitis

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SUMMARY A Pakistani girl presented with acute abdominal pain and raised serum amylase and alkaline phosphatase concentrations. She was found to have a gastric trichobezoar with a tail extending to the mid-ileum. The altered biochemical parameters returned to normal after surgical removal of the bezoar. Irritation of ampulla of Vater by the bezoar tail is believed to have caused transient pancreatitis.

Trichobezoar has been reported to cause a large number of complications but we believe this to be the first report of trichobezoar associated with pancreatitis.

Case report

A 15 year old Pakistani schoolgirl, resident in Britain for 12 years, presented as an emergency, complaining of severe colicky upper abdominal pain of 24 hours’ duration. She had vomited bile stained fluid on two occasions during this period and had opened her bowels once noticing a streak of fresh blood in the stool. During the previous year she had had occasional upper abdominal pain and had not gained weight.

She was asthenic, pale, and ill looking; in pain and mildly dehydrated clinically. She had no fever, her pulse rate of 100/minute, and her blood pressure was 120/70 mm Hg. There was a tender, mobile, firm mass in the epigastrium and right hypochondrium, with associated guarding. The bowel sounds were normal as was rectal examination. A full blood count showed only a mild, microcytic, normochromic anaemia. Her serum amylase concentration was raised at 2000 IU/l (normal up to 300 IU/l) but urea and electrolyte concentrations were normal. Other biochemical abnormalities noted were a low serum albumin concentration of 28 g/l and a raised serum alkaline phosphatase value of 800 IU/l (less than 600 IU/l is normal for our laboratory). Serum bilirubin and liver enzyme concentrations were normal. Plain abdominal radiography confirmed the presence of an upper abdominal mass surrounded by several loops of distended small bowel. An ultrasound scan showed the mass to be solid, making the diagnosis of a possible pancreatic pseudocyst unlikely. Other possible diagnoses were those of tumour or gastric bezoar. On subsequent questioning the patient admitted to trichophagia extending over a year. A barium meal (Figure) confirmed the diagnosis. Three days after admission the patient underwent laparotomy. A trichobezoar forming a complete cast of the stomach was found with a tail extending as far as the mid-ileum (Figure). The duodenal wall was hypertrophied and there was a solitary large chronic jejunal ulcer 6 cm from the duodenjejunal flexure. The pancreas, liver, gall bladder, and biliary tree were macroscopically normal.

The bezoar was removed completely through a single gastrotomy incision. The patient’s serum amylase concentration 24 hours after surgery fell to 800 IU/l; thereafter it fell slowly and was 250 IU/l on discharge from hospital two weeks later. She had a psychiatric consultation before discharge. Five weeks later the patient was admitted to hospital for reassessment—full blood count and biochemical screen were normal.

Discussion

Trichobezoars are potentially lethal. The mortality of cases not operated on has been quoted as over 70%, a figure which falls to under four per cent when surgical removal is undertaken. Death usually occurs secondary to gastric ulceration, perforation, and peritonitis from the bacterial contamination of the bezoar which always renders it noticeably foul and putrid.