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CHRONIC CALCIFIC PANCREATITIS PRESENTING WITH STUNTING AND DIABETES MELLITUS

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ABSTRACT
We report the case of a 16 year old boy who presented with 8 months history of weight loss, 3 months history of polydypsia, polyuria and polyphagia. The child had poor growth since age of 5 years, during which time he developed recurrent abdominal pain for 4 years. A diagnosis of chronic calcific pancreatitis complicated by stunting and diabetes mellitus was made on the basis of weight/height ratio less than 5th NCHS percentile for his age, fasting blood sugar of 233mg/dl, and presence of calcifications over the pancreatic area on a plain abdominal X-ray. This case is reported due to the rarity of this condition in children. It is also the first to be seen in our hospital. It will serve to alert the Paediatrician to such clinical condition in children with chronic abdominal pain. In this case, symptoms of diabetes mellitus were the reasons for seeking medical attention and it also shows how chronic pancreatits led to insulin dependent diabetes mellitus.

Key Words: - chronic calcific pancreatitis, stunting, diabetes mellitus. *(Accepted 7 August 2007)*

INTRODUCTION

Chronic pancreatitis was first described by Largerloef in 1942 and subsequently by Comfort in 1946.[1,2] They described the condition as being characterized by episodic or continuous upper abdominal pain and weight loss, and sometimes complicated by cyst/pseudocyst, pancreatic calcification and diabetes mellitus.[1] Chronic calcific pancreatitis is a tropical disease[3] and is one of the causes of malabsorption in many parts of the tropics .[3,4] Olurin and Olurin in1962 recorded 5 cases during an 11-year period in Ibadan. Protein energy malnutrition was considered the most common important aetiological factor in 90 percent of the cases.[5] This patient presented with diabetes mellitus and growth failure and calcific pancreatitis was implicated. It is important to have this entity in mind in children with recurrent abdominal pain. The association of diabetes mellitus, abdominal pain and stunting must always raise the suspicion of pancreatic disease.[6]

Case Report

This sixteen year old boy presented at the University of Benin Teaching Hospital with eight month history of polydypsia, polyuria, polyphagia, and two weeks history of parotid swelling.

The patient was said to have failed to grow well since the age of five years. Past medical history revealed a recurrent abdominal pain at age of five years and which lasted for four years. The pain was periumbilical, intense and was accompanied by low grade fever and anorexia . He had about five episodes a day, each lasting for a few minutes and relieved by applying pressure on the abdomen. There was no history of passage of bulky malodorous stool and no family history of diabetes mellitus.

Physical examination revealed a chronically ill looking but alert boy. There was obvious stunting. The weight was 17kg and the height was 121cm, giving a Wt/Ht ratio less than the 5th NCHS percentile[7,8] for a 16 year old boy. He had bilateral parotid swelling measuring 4cm5cm that was soft and non tender (fig. 1). There was neither pallor nor finger clubbing. There was no evidence of development of secondary sexual characteristics. The breath sounds were normal. The liver was palpably enlarged, by 4cm below the right costal margin, firm and non tender.

The investigations done revealed elevated fasting blood sugar of 233mg/dl, elevated serum amylase level of 7.4g/dl, albumin of 4.4g/dl and globulin of 3g/dl. Liver function tests were normal. Haemoglobin genotype was AA. Urinalysis revealed glycosuria of 3+, and no ketonuria. Plain abdominal X ray showed multiple varied sized calcific densities overlying the 12th rib and extending to the right transverse process of L1 in keeping with pancreatic calcification (fig2).

The diagnosis of insulin dependent diabetes mellitus and growth failure secondary to chronic calcific pancreatitis were made. The diabetes mellitus was controlled, and thereafter he was placed on 12 hourly injections of combined lente and soluble insulin. His daily dietary intake was gradually raised to 2000kcal/day. The concerted efforts of paediatrician, dieticians, social workers and good nursing care helped achieve good diabetic control. At the time of discharge, 2 weeks after admission, he had gained

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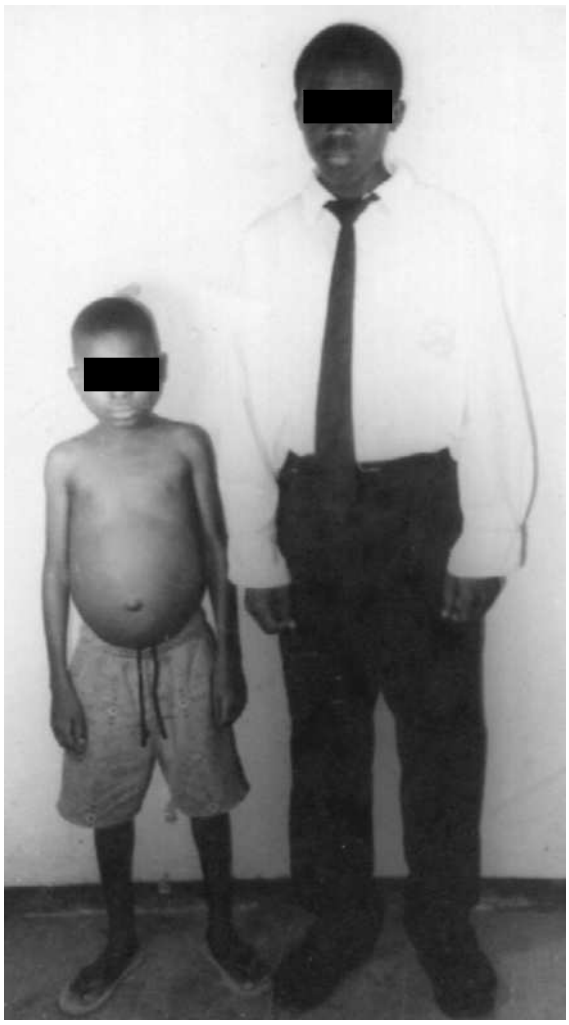
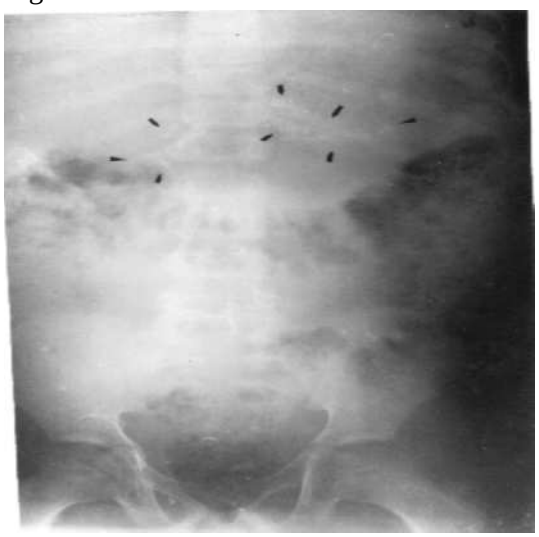
2kg and was able to inject himself with insulin and monitor his urine sugar.

Fig 1



Picture showing the patient (left) standing next to a boy his age

Fig 2



Pain abdominal X-ray with black arrows indicating the calcific densities overlying the 12th rib.

DISCUSSION

Chronic calcific pancreatits is a rare condition in children.[1] High alcohol consumption is the most important aetiologic factor causing chronc pancreatitis in adults notably in Europe and USA[1,9], whereas in the tropics protein energy malnutrition has been considered to be responsible for pancreatic calcification.[3] Other causes include hereditary pancreatitis an autosomal dominant disease, hypercalcaemia, hyperlipoproteinamia type- I, IV, V, cystic fibrosis, viral infections like mumps and hepatitis, ascaris infestation, as well as biliary and pancreatic duct anomalies.[3,9]

Studies have shown that there is some evidence of permanent pancreatic damage after recovery from malnutrition.[9,10] Fibrosis follows atrophy of the pancreatic acinar cells in Kwashiorkor. However, in this patient there was no confirmed past history suggestive of protein energy malnutrition. There is a strong association between dietary deficiency and development of chronic calcifying pancreatitis.[6,9] It is possible that an unsuspected lesion may have affected the pancreas during acute protein energy malnutrition, such as an ascending low grade pancreatitis from bacterial contaminants of the duodenum.[6] Sarles suggested that certain patients have genetic disposition to the spontaneous development of chronic calcifying pancreatits and this is brought about by continous alcohol consumption, dietary imbalance or rarely hypercalcaemia. Recurrent chronic relapsing pancreatitis is a feature of hereditary pancreatitis, a rare autosomal condition reported chiefly among Caucasians in USA.[9,13] The first changes in chronic pancreattis is the involvment of the finer branch ducts and their catchment areas of acinar parenchyma. The ducts are blocked by protein plugs. The ducts and the acini supplied undergo inflammatory reaction and eventual destruction of first exocrine and later endocrine tissues in the vicinity.[3,6]

A large part of the pancreas remains unaffected in the early stages but as the lesions enlarge, more numerous and larger plugs are precipitated in wider ducts, and layers of calcium accumulate on their surfaces. The main pancreatic ducts and principal branches may become occluded by calculi or by scarring fibrosis in nearby parenchyma.[6]

Finally, the pancreas is reduced to a narrow hard fibrous cord in which acini and ducts are scattered and islet cells are progressively replaced by fibrous tissue. Clinically the majority of patients present with abdominal pain which is usually the dominant syndrome, and after years may present with diabetes

mellitus and marked growth failure.

The serum amylase analysis in children with recurrent abdominal pain is important for early diagnosis or exclusion of chronic pancreatitis. The marked weight loss is attributable to the severe abdominal pain, which may result in anorexia as well as protein maldigestion secondary to pancreatic exocrine enzyme deficiency. In this patient the growth failure was so pronounced that at 16 years he weighed 17kg, was 121cm tall and had not developed any secondary sexual characteristics.

The association of diabetes mellitus with abdominal pain and marked weight loss must always raise the suspicion of chronic pancreatic disease.

An exclusion of growth hormone deficiency was considered as a possible causative factor of the short stature of the child. It is known that the diagnosis of classic growth hormone deficiency is suspected in cases of profound postnatal growth failure, [14] but our patient had normal postnatal growth status. However acquired growth hormone and other endocrine disorders could not be excluded because of our limitations in the tools for their diagnosis.

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