TROPICAL CALCIFIC PANCREATITIS

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SUMMARY

Two cases of tropical calcific pancreatitis were reported with a brief review of its aetiology and clinical feature.

INTRODUCTION

The classical picture of chronic calcific pancreatitis as a disease mainly of the middle aged male, who is often an alcoholic presenting with symptoms of abdominal pain, diabetes mellitus and steatorrhoea is well known (Sarles *et at.*, 1976 and Mark and Bank 1976). There is another form of calcific pancreatitis reported widely in the tropical "developing" countries. These patients are typically non-alcoholic, have history of malnutrition with diabetes mellitus as the main presenting feature. We are presenting two cases which we believe are examples of the latter.

Case 1

S.B.T. was a 35 year old Orang Asli ambulance driver, he was referred by Gombak Hospital for management of poorly controlled diabetes mellitus.

His symptoms dated three years ago when he developed polyuria and polydipsia. Despite this he did not seek any medical attention. About a year ago, he had frequent paraesthesia over both lower limbs. About nine months ago, his sex life was affected with penile erection not sustained. He thus sought treatment from Gombak Hospital and was diagnosed to have diabetes mellitus. He has since been tried on insulin injection which did not control his diabetes satisfactorily. This is partly because he was not regular with the injection and was inconsistent with his diet. He claimed that there was no weight loss. The other significant history was that in the last few years, with fatty meal, his stool was noted to be more "greasy".

In the past history, he claimed to have frequent abdominal pain while in childhood, often associated with the passage of large round worms. Socially, he came from a large family of eight children. The family situation was poor and difficult in his childhood days. Meat was a scarcity in their diet; there was often insufficient rice to eat so that they had to go hungry. Fish, twice a week, was relatively more frequent in their menu. However, since he started working as an ambulance driver when he reached adulthood, his diet has improved. He is presently having to support two wives and seven children. He has a deceased elder brother who probably had diabetes mellitus - the detailed medical history he could not provide as the brother stayed in another Orang Asli camp.

On physical examination, he appeared to be a thin and small man with no sign of malnutrition. Besides a cataract over the left eye, prominent both parotid glands, absent both knee and ankle reflexes, there was no other abnormal sign.

Investigations showed normal full blood count and urine microscopy. The random blood glucose on admission was 277 mg%. The serum urea, electrolyte and amylase were normal. Serum total

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protein was 6.6 gm% with albumin at 3.0 gm%, globulin at 3.6 gm%. The protein electrophoresis was normal. Serum calcium was 4.7 mEq/1 and serum phosphate was 49 mgm%. Serum iron, TIBC, vit. B12 and folate level were all normal. Xylose absorption test was normal with xylose excretion at 6.3 gm in five hours (normal 4.5-8 gm). Stool microscopy for neutral fat and meat fibres was positive. Plane abdominal x-ray showed irregular spotty calcification of the pancreas (fig. 1). Oral cholecystography was normal. A diagnosis of tropical calcific pancreatitis from previous protein malnutrition with diabetes mellitus and malabsorption was made.

The patient was then tried on dietary control and oral hypoglycaemics for his diabetes (in view of his past history of poor compliance). Despite daily dose 3 gm of metformin and 500 mg of chlorpropamide; later daily dose of 3 gm metformin with 15 mg of glibenclamide, his diabetes was uncontrolled. Insulin was then reintroduced. This again failed to bring a satisfactory level of blood sugar before he insisted on being discharged from the ward.

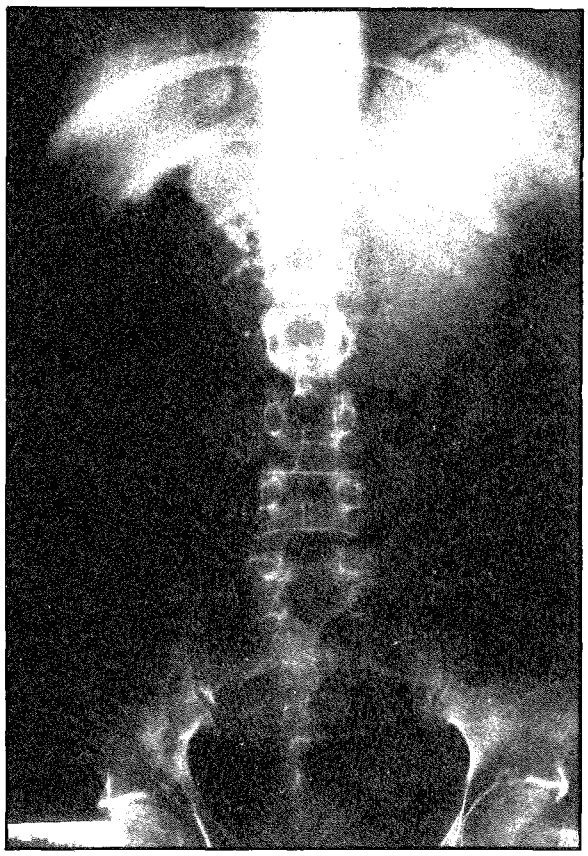

Fig. 1. Plane abdominal x-ray of Case 1 showing irregular, spotty pancreatic calcification.

Case 2

L.P. was an 18 year old Indian girl from Kelang, Selangor seen in 1979. She was admitted for growth retardation and weight loss for two months prior to admission.

Her problem dated back to 1971 when she was first admitted to the University Hospital, Kuala Lumpur. Her problem then was facial and limb swelling for three years, treated intermittently in the district and estate hospitals. The patient was a full term normal delivery. She had delayed developmental milestones, she sat unaided at 12 months and stood with support at 2 years. The social history was the main clue to her problem. She came from a very poor socio-economic background. Her father was a rubber tapper earning M$70/- per month. Her mother had passed away. She was the seventh of eight children in the family. The family diet consisted of rice, vegetable with curry and fish or meat once every fortnight. On physical examination then, the patient appeared pale, with oedema of face and both ankles. There was tachycardia with cardiac enlargement. Investigations showed a hypochromic microcytic anaemia with serum iron at 18 mgm%, normal serum folate and Vit. B12 level. Stool examination demonstrated presence of ankylosmatosis, trichuris and ascaris ova. The serum albumin was 1.7 gm% and serum globulin was 3.6 gm%. The serum electrolyte, urine microscopy were normal. Chest x-ray showed a small left pleural effusion. Plane abdominal x-ray showed calcification of the pancreas. Studies for malabsorption carried out then showed normal faecal fat, normal xylose absorption test and barium study. Diagnosis of malnutrition and

TABLE I
COMPARISON BETWEEN CLASSICAL AND TROPICAL CALCIFIC PANCREATITIS

Characteristics	Classical	Tropical
Locality:		
	Temperate countries	Tropical developing countries
Aetiology:		
	Alcoholism	Protein malnutrition
Age group:		
	Middle age	Teens and twenties
Diabetes	+	++
Abdominal pain	++	+
Steatorrhoea	+	+

helminthiasis with iron deficiency anaemia causing congestive cardiac failure was made. The patient was treated accordingly with replacement therapy, anti-helminthics and high protein diet. She made remarkable recovery with Hb of 10 gm%, serum albumin at 3.7 gm% on discharge.

She was lost to follow up until two years later, when she was admitted again with the same problem of facial and limb oedema. Physical examination then showed a pale patient with generalized oedema in cardiac failure. Investigations showed similar finding of iron deficiency anaemia with helminthiasis and low serum protein. Additional test done during this admission showed a diabetic glucose tolerance curve and normal duodenal biopsy. She again improved with replacement therapy and high protein diet. As for the diabetes, blood sugar was in acceptable level without drug therapy.

The patient again did not turn up for follow up until the third admission in 1979. The social and economic circumstances of the family did not improve over these years. The patient never went to school and was not employed.

On examination, she was of a small build weighing 16.3 kg and 118.5 cm in height. There was no secondary sexual characteristics. She was mildly pale. Both eyes showed early cataract. There was no other abnormal physical sign.

Investigations showed Hb of 9.9 gm%. Total white and differential count was normal. Stool examination showed the presence of ankylostoma, trichiaris and ascaris ova. Fasting blood sugar was 270 mg%. Plane abdominal x-ray again demonstrated extensive calcifications of the pancreas. Blood urea, serum electrolyte, urine microscopy, serum calcium, phosphate, thyroid function test, xylose absorption test, chest x-ray were all normal.

A diagnosis of tropical calcific pancreatitis with diabetes mellitus and growth retardation was finally made. The diabetes was controlled satisfactorily with Lente Insulin 52 units daily. She gained 3 kg in weight during the hospital stay. The patient again failed to turn up for follow up.

DISCUSSION

The classical picture of calcific pancreatitis usually involves middle aged patients with alcohol as the main aetiological factor. The patient usually complains of recurrent abdominal pain. In a typical patient with alcohol induced pancreatitis, the abdominal pain occurs some 12-48 hours after the end of drinking bout; the onset of persistent pain is classically on the 'afternoon after the night before'. Each attack lasts 3-7 days unless a complication ensues. Majority of the patients suffer 2-4 attacks a year. Painless alcohol induced calcific pancreatitis does occur, but is uncommon (Sarles *et al.*, 1976 and Mark and Bank, 1976). Diabetes mellitus occurs in 10-50% of the patients and steatorrhoea in 17-38.8% (Olurin, 1969).

Fung *et al.*, (1970) in their report on the chronic pancreatitis in Singapore showed an example of the illness. There were seven cases among 14,767 new admission to the Department of Medicine, University of Singapore over three years (incidence of 0.05%). The racial composition was 3 Chinese, 3 Indians and 1 Malay. Five of the patients were alcoholic (1.9% of the total alcoholic admissions to the same department). The average duration of the alcoholism was 23 years. Two of the patients had biliary disease. The age range of these patients was from 38-67 years. Recurrent abdominal pain occurred in three of the seven patients, diabetes mellitus in five patients, pancreatic calcification and steatorrhoea in six patients.

In contrast to this classical pattern of calcific pancreatitis, Zuidema in 1959 reported eighteen of his Indonesian patients who had calcifications on their pancreas as shown in the abdominal x-ray. They were all young adults, none had alcoholic history. Sixteen were diabetes, all were more or less emaciated and had very low protein intake. None of these patients presented with abdominal pain. Zuidema thought that the pancreatic lesion was due to long continued protein malnutrition. It is in fact known that in patients with severe protein malnutrition, the pancreas is small, atrophic and fibrosed (Davies, 1948; Trowell *et al.*, 1954). Subsequently, similar reports have appeared from the other tropical developing countries. Olurin *et al.* (1969) reported 45 patients with pancreatic calcifications from the West Nigerian town of Ibadan. The average age of these patients was 20.4 years, the male to female ratio was 1.6:1. None of the patients was alcoholic, 89% were underweight with 24% having clear signs of malnutrition. Eighty-two per cent of these cases had diabetes mellitus and 62% had abdominal pain. The authors commented that the abdominal pain was often non-specific and difficult to assess. However, only in four patients was the pain severe, three of these had raised serum amylase to above 1000 samogi units. The authors again attributed more than 90% of these patients with pancreatic calcification to protein malnutrition.

Shaper in 1960 also reported 20 patients from Uganda with calcific pancreatitis. None of his patients had a history of chronic alcoholism and 60% of them had diabetes mellitus. Ratnaike (1962) presented a similar case from Ceylon.

It thus appears that there are two common patterns of pancreatitis with calcification. The classical pattern is mainly found in the temperate countries. These patients are usually of middle aged group with alcoholic history; abdominal pain is a common symptom and diabetes is found in about 30% of them. The other pattern is mainly found in the tropical developing countries. The patients are younger in their twenties, they are non-alcoholic often with history of protein malnutrition. Diabetes mellitus occurs in 80% of these patients and abdominal pain is not a marked feature. Our patients being from low social background, poor nutrition, with diabetes and relatively minor abdominal pain, would be examples of the latter group.

The pathology of tropical calcific pancreatitis is the atrophy of pancreatic tissue with replacement by fibrosis tissue which is interlobular, periductal and panacinar. The acini are atrophied and islets of Langerhans are destroyed, the ducts and ductules are dilated, and small cysts are formed (Olurin, 1969).

The other rare causes of calcification in the pancreas are: hyperparathyroidism and familial pancreatitis. The later is inherited as autosomal dominant. The precise aetiology and pathogenesis is unknown. As our patients did not have family history of similar illness and the serum calcium levels were normal, hyperparathyroidism and familial pancreatitis were excluded.

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REFERENCES

Davies, J.N.P. (1948) The essential pathology of Kwashiorkor, *Lancet,* **1,** 317-320.

Fung, W.P., Aw, S.E., Khoo, O.T. (1970) Chronic pancreatitis in Asian patients in Singapore, *Med, J, Aust.,* **1,** 653-656.

Marks, I.N. and Bank, S. (1976) Chronic pancreatitis, relapsing pancreatitis; calcification of the pancreas, part II, clinical aspects, Gastroenterology, Vol. 3 Ed. Bockus, H.L., Saunders, W.A.

Olurin, E.O. (1969) Pancreatic calcification: a report of 45 cases, *Brit. Med. J.,* **4,** 534-539.

Ratnaike, V.T. and Rajasuriya, K. (1963) Pancreatic calcification related to protein malnutrition. *Trop. & Geographical Med.,* **15,** 1-6.

Sarles, H., Payan, H., Tasso, F. and Sahel, J. (1976) Chronic pancreatitis, relapsing pancreatitis, calcifications of the pancreas, Part I, Pathology. Gastroenterology, Vol. 3, Ed. Bockus, H.K., Saunders, W.A.

Shaper, A.G. (1960) Chronic pancreatic disease and protein malnutrition, *Lancet,* **1,** 1223-1224.

Trowell, H.C., Davies, J.N.P., Dean, R.F.A., Kwashiorkor, London, 1954.

Zuidema, P.J. (1959) Cirrhosis and disseminated calcification of the pancreas in patients with malnutrition, *Tropical & Geographical Med.,* **11,** 70-74.