Hemiplegia in a Patient with Fibrocalculous Pancreatic Diabetes

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Fibrocalculous pancreatic diabetes (FCPD) is a unique form of diabetes secondary to non alcoholic, chronic, calcific pancreatitis seen in tropical developing countries of the world.[1] Being a secondary for diabetes, it was earlier believed that vascular complications do not develop in FCPD. We and others have shown that retinopathy, nephropathy and neuropathy do occur in FCPD patients and that their frequency is similar to that seen in Type 2 diabetes.[2] However, macroangiopathy is extremely uncommon in FCPD patients. We have earlier reported on the occurrence of myocardial infarction and peripheral vascular disease (PVD) in FCPD patients.[3] We now report on an FCPD patient with hemiplegia and we believe this is the first report of hemiplegia, in this form of diabetes.

SRA, a 47 years male patient presented to our centre with weakness of left upper and lower limbs. He was a known case of FCPD treated at our centre. The diagnosis of FCPD was based on the following : history of recurrent abdominal pain from childhood, steatorrhoea, pancreatic calculi on abdominal X-ray and evidence of dilated pancreatic duct with intraductal calculi on ultrasonography. He also had low faecal chymotrypsin - 2.0 unit/gm (Normal value > 13.0 unit/gms). He had evidence of non proliferative diabetic retinopathy, nephropathy and neuropathy.

He was lean with a body mass index of 17.0 kg/m^2. On examination he was conscious and well oriented. Higher functions were normal. Cranial nerves were normal except for left upper motor neuron palsy of VII cranial nerve. Motor system examination showed that he had weakness of left upper and lower limbs (power grade 2/5 power). He also had mild blunting of sensations on left side. Plantar was extensor and he had exaggerated deep tendon reflexes on the left side. Blood pressure was 140/80 mmHg and pulse rate was 80/mt. CVS and respiratory system were normal. CT scan revealed multifocal cerebral infarcts in the right lentiform nucleus, frontal subcortical regions and right corona radiata (Fig. 1). Duplex colour doppler studies of the carotid arteries were normal and there was no evidence of any plaque or stenosis.

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Laboratory investigations showed the following findings:

Fasting plasma glucose - 159 mg/dl, postprandial plasma glucose - 390 mg/dl, HbA1c - 10%, blood urea - 18 mg/dl, serum creatinine - 1.0 mg/dl, total cholesterol - 223 mg/dl, serum triglycerides - 75 mg/dl, HDL cholesterol - 49 mg/dl, LDL cholesterol - 159 mg/dl, VLDL cholesterol - 15 mg/dl, Lp (a) - 100.0 mg/dl, sodium - 141 Meq/L, potassium - 4.8 Meq/L, chloride - 103 Meq/L, bicarbonate - 24, anion gap - 14, osmolarity - 295.3.

He was treated with ticlopidine and aspirin, anti-oedema measures and insulin for control of diabetes. He was also started on physiotherapy after which he made a gradual recovery.

We have earlier published one case report each of gangrene (PVD) and myocardial infarction in FCPD patients.[3] We now present a case of hemiplegia in an FCPD patient. Macrovascular complications are rare in FCPD because of onset in youth, leanness with low body mass index and low lipid levels. In this patient, the serum cholesterol and LDL cholesterol levels are relatively high and moreover the Lp(a) level is also elevated indicating a high risk for atherosclerosis. This may explain why he developed hemiplegia. Moreover, he had grossly uncontrolled diabetes with a post prandial blood glucose of over 350 mg% and HbA1c of 10.0%. It is known that hyperosmolarity can also cause transient focal neurological signs as seen in Hyperosmolar non-ketotic coma. However there was no evidence of hyperosmolarity in this patient and CT scan showed evidence of infarcts confirming thrombosis as the cause of the hemiplegia.

References

1. **Report of the ADA Expert Committee** on the diagnosis and classification of diabetes mellitus. *Diabetes Care* 1997; 20 : 1183-97.
2. **Mohan V, Suseela L, Snehalatha C, Bharani G, Mahajan VK, Ramachandran A, Viswanathan M, Kohner EM.** Tropical pancreatic diabetes in south India : heterogeneity in clinical and biochemical profile. *Diabetologia* 1985; 28 : 229-32.
3. **Mohan V, Ramachandran A, Viswanathan M.** Two case reports of macrovascular complications in fibrocalculous pancreatic diabetes. *Acta Diabetologica Latina* 1989; 26 : 345-9.