rigidity of right side with tremors of tongue and right half of the body with weakness. Initially we suspected space occupying lesion in the brain, as child had predominant unilateral signs. Investigations shows decreased total counts with mesenteric lymph nodes with hepato-splenomegaly on ultrasonography of abdomen. CT scan of brain was normal. Bone marrow examination shows classic Niemann-Pick cells. Acid sphingomyelinase assay confirms our diagnosis of Niemann-Pick disease Type A. Prenatal diagnosis can be made by assay of acid sphingomyelinase activity in cultured chorionic villi or amniocytes [3].

Unilateral involvement with tremors in the initial stage, absence of cherry red spot despite neurological involvement, absence of lung involvement are unusual presentations. There is no literature available on this kind of unusual presentation.

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Diabetic Ketoacidosis Following Mumps

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A-13-year-old girl presented with diabetic ketoacidosis with convincing clinical signs of parotitis (fever, drooling of saliva, inability to swallow with development of bilateral parotid swelling) and pancreatitis (fever, abdominal pain and vomiting), along with high serum amylase and positive mumps IgM titer. This suggests that mumps virus may have been the causative factor, probably as a result of concomitant involvement of the pancreas.

Key words: *Diabetic ketoacidosis, Mumps.*

The common complications of mumps are meningitis with or without encephalitis, gonadal involvement and myocarditis while pancreatitis is an uncommon complication of mumps. Epidemiological studies have suggested that mumps pancreatitis may be associated with subsequent development of diabetes mellitus, but a causal link has not been established. The following is a report of a case of diabetic ketoacidosis (DKA), apparently due to mumps pancreatitis, with involvement of parotid glands.

CASE REPORT

A-13-year-old girl, developed low-grade fever, drooling of saliva and vague abdominal pain. Next day, it progressed to inability to swallow and speak, and vomiting. Two days later, she had twitching of face and generalized convulsions and became unconscious. She was then admitted to our institution.

She was drowsy but oriented and following verbal commands. Moderate dehydration was present. Her temperature was 101ºF, pupils were bilateral normal size and reacting and Glasgow coma score was 15. Her cardiac examination was normal. The abdomen was soft and non-tender. Liver was 2 cm below costal margin, soft, smooth and non-tender. Spleen was not palpable. On central nervous system examination, there was no focal neurological deficit or signs of meningeal irritation.

Investigations revealed high random blood sugar values (462 mg/100mL), urine was postive for sugar and ketone, and blood gas analysis revealed metabolic acidosis. She was diagnosed as a case of diabetic ketoacidosis and treated with intravenous fluids, regular insulin and other supportive treatment. Hyperglycemia was controlled within 24 hours. On the third day of admission, she developed swelling of right parotid gland followed by involvement of the left parotid gland following day. Serum amylase was 918 U/L. USG abdomen showed. hepatomegaly and bilateral enlarged

kidneys (right kidney- 11.5 × 4.5 cm, left kidney- 11.2 × 4.6 cm) with increased cortical echotexture. Cortico-medullary distinction was maintained. Mumps IgM by ELISA was positive.

On 5th day of admission, drooling became less and she was able to swallow liquids. Her parotid swellings subsided on the 8th day of the admission and she was discharged on subcutaneous human mixtard insulin.

DISCUSSION

Type I diabetes mellitus (T1DM) results from the interaction of genes, the environment, and the immune system. The presence of disparate geographic prevalence, rising worldwide incidence, and 50% discordance rate in identical twins provides evidence that environmental agents are operative. As there is a latent period between the appearance of T1DM-associated autoantibodies and onset of disease, additional environmental factors – probably interacting with genetic factors – also seem to modulate the rate of development of the disease [1].

Early nutrition and infection have been the most frequently implicated early environmental influences. There is, however, no direct evidence to date that either nutrition or infection plays a major role in causation, albeit one example of prenatal rubella infection which is associated with beta-cell autoimmunity in up to 70%, and diabetes in up to 40% of infected children [2,3]. A relationship between beta-cell autoimmunity and intrauterine exposure to enteroviral (mainly Coxsackie B_4) infection has been proposed [3-5]. Studies from Finland and Sweden suggested that maternal enterovirus infection may increase the likelihood of subsequent T1DM development in offspring [5]. Higher levels of antibodies to procapsid enterovirus antigens were found in the pregnant sera of mothers of children who developed diabetes. However, the presence of antibodies against enteroviruses in people with autoimmunity does not prove a causal relationship. Persons with autoimmunity also may be more prone to enteroviral infection, may have a stronger humoral response to infection because of their particular HLA genotype, or may be in a nonspecific hyperimmune state marked by elevation of antibody levels to various exogenous antigens. Islet related autoantibodies also have been detected after mumps, measles, chickenpox and rotavirus infections [7,8]. But of all the viral infections, mumps seems to be most frequently associated with diabetes [8-9]. In most of these instances, symptoms of diabetes developed within one-to-eight weeks after infection [8,9].

In the case presented in this report, presence of clinical features of mumps and the postive IgM indicates a recent mumps infection. This report further strengthens the reported association of mumps infection and diabetes.

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