

Postpartum Pancreatic Encephalopathy -A Rare Case

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Abstract: A young female of postpartum pancreatic encephalopathy is described who presented with convulsions and coma. She had deranged lipase levels in blood, and cerebrospinal fluid along with abnormal white matter signals in MRI brain. As the patient improved with medical management, deranged blood lipase levels and abnormal MRI signals also improved. Pancreatic encephalopathy is a rare complication of pancreatitis in the setting of postpartum period. In any atypical neuropsychiatric condition with abdominal symptoms the possibility of pancreatic encephalopathy should be considered after certain exclusions.

Key Words : encephalopathy,metabolic, pancreatic, postpartum

I. Introduction

A wide variety of neurological conditions can affect women during pregnancy and puerperium. Common neurological disorders seen in relation to pregnancy and post partum period are eclampsia ,venous stroke, epilepsy, demyelination and metabolic encephalopathy¹. We describe a rare case of postpartum pancreatic encephalopathy in a young female .

II. Case Report

A 22 year female developed fever , abdominal pain ,altered sensorium, generalized tonic-clonic seizures and quadriparesis on fifth post partum day.She had an uneventful full term normal delivery.. On examination she had a temperature of (100F). BP 110/70mmHg, Pulse.100/min. She was in altered sensorium with eye opening on deep painful stimuli. Pupils were bilaterally equal and reacting to light. Eye movements were full. Bilateral fundi were normal .No meningeal signs. Tone increased in all limbs. DTRs - brisk, Motor power grade 3/5 in upper limbs and grade 2/5 in lower limbs. Pin prick sensations were intact. Plantars were mute on either side. Abdomen was distended with normal bowel sounds. MRI Brain showed bilateral symmetrical hyper intense signals on diffusion and T2 FLAIR sequences with low ADC values in posterior limb of internal capsule, posterior centrum semiovale and splenium of corpus callosum suggestive of nonspecific metabolic encephalopathy/demyelination/ischemic injury (Fig1). MR angiogram and venogram did not show any vascular abnormality. EEG revealed diffuse slowing of background activity, with theta waves recorded over all the regions of the scalp.

A set of investigations was done which showed Hb-15.2gm%,TLC-16400,N87%, L11%,platelet count 1.35lac,SGPT-35 I units, SerumNa+145meq/l,SerumK+4.6meq/l, Cl-125meq/l, PT-INR 1.3, Blood Urea 59mg/dl, Serum Creatinine-0.85mg/dl.Malaria antigen negative, D-dimer- 230 microgm /L,Serum Amylase was 340 U/L(23-85 U/L normal) and Serum Lipase was 3931 U/L (0-160 U/L normal).

In view of her high amylase and lipase and abdominal distention, a CECT abdomen was ordered which showed minimal peripancreatic fat stranding and small amount of peripancreatic fluid but no evidence of any hepatobiliary disease. A presumptive diagnosis of acute pancreatitis was considered. A CSF study was done which showed an opening pressure of 15cm , glucose 59mg/dl(corresponding blood sugar 83mg/dl),total cells -2 all lymphocytes, Pandy's test- negative, protein 46mg/dl, VDRL negative with CSF lipase 48 mg/dl.

Patient was managed with intravenous fluids, broad spectrum antibiotics and nutritional support along with physiotherapy. With regular rehabilitation and medical management she gradually regained motor power and improvement in sensorium.Two weeks after onset of illness patient became conscious, oriented, following commands. Her speech had become normal, she was able to walk with partial support and her upper limb power has improved to grade 4/5. Her swallowing reflexes recovered gradually and she accepted feeds orally.A repeat MRI Brain was done which showed far less degree of abnormal signals in the same distribution as compared to previous MRI with no contrast enhancement. A repeat serum Amylase and lipase was done, which were 155 U/L and 102 U/L respectively. A final diagnosis of postpartum pancreatic encephalopathy was made

agitation, disorientation to time, space and person, and visual and auditory hallucinations. It is a diagnosis of exclusion as many situations can mimic this condition, such as electrolyte imbalance, hypo/hyperglycemia, diabetic acidosis, hypo/ hyper-calcaemia, hyperosmolarity syndrome, renal or hepatic failure, cerebral vascular insufficiency and delirium tremens,^{5,8,7}The pancreatic encephalopathy does not affect the course of the pancreatitis. The recovery usually is uneventful, as in our case⁵.

In our case all these conditions of ischaemia ,electrolyte imbalance and demyelination were ruled out. Most accepted hypothesis for the pathogenesis of pancreatic encephalopathy mentions lipase as the crucial substance⁶. Pancreatic enzymes (lipase) in the blood stream alters the blood-brain barrier and damage scattered areas throughout the cerebrum, brainstem and cerebellum, inducing increased vascular permeability. The intravascular osmotic pressure decreases and the brain becomes more vulnerable to transudation, and finally results in brain oedema⁶. These areas are picked as patchy white matter signal abnormalities in MRI as described in our patient.. Therefore in any patient with atypical neuropsychiatric condition especially associated with abdominal symptoms and deranged pancreatic parameters along with abnormal white matter abnormalities, pancreatic encephalopathy should be considered after exclusion of infective , ischemic and other common metabolic factors.

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