Journal of Advanced Medical and Dental Sciences Research

@Society of Scientific Research and Studies

Journal home page: www.jamdsr.com doi: 10.21276/jamdsr ICV 2018= 82.06

(e) ISSN Online: 2321-9599; (p) ISSN Print: 2348-6805

Case Report

Intracerebral Hemorrhage in the setting of Acute Pancreatitis : A Case Report

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ABSTRACT

Neurological complications like pancreatic encephalopathy and intra cerebral hemorrhage are uncommon and serious conditions associated with acute pancreatitis. It's a part of multi organ failure that accompanies acute pancreatitis, usually presenting early (within 2 weeks)in the disease. We report a case of intra cerebral hemorrhage as a complication of acute pancreatitis in a 58 year old female who presented with the chief complaint of abdominal pain located in the epigastric region and after three days of presentation developed weakness in left upper and lower limbs. Amylase and lipase levels were elevated but the other lab workup was non-contributory. MRI brain showed right parieto occipital bleed. Patient improved in terms of power of limbs and started walking with support in 10 days with the help of conservative management.

Key words - Intracerebral hemorrhage, Acute Pancreatitis

Received: 14 October, 2019 Revised: 13 November, 2019 Accepted: 25 November, 2019

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This article may be cited as: Agarwal N, Dabria T. Intracerebral Hemorrhage in the setting of Acute Pancreatitis : A Case Report. J Adv Med Dent Scie Res 2019;7(12): 64-65.

INTRODUCTION

Acute pancreatitis is a common inflammatory condition involving pancreas. Most common causes being biliary and alcoholic. Acute pancreatitis which itself can be fatal, is also known to cause neurological complications like intra cerebral hemorrhage which usually has a high mortality. The pathogenesis is due to the involvement of multiple factors in the process like release of the pancreatic enzymes in the circulation, excess release of the cytokines, phospholipase A2 activation, electrolyte disturbances and damage by oxygen free radicals to the brain.

CASE PRESENTATION

58 year old female was admitted with epigastric pain vomiting and fever but without any hemodynamic instability. No past history of Hypertension/Diabetes Mellitus/Coronary Artery Disease present. On examination there was tenderness of upper abdomen and lab tests suggested elevated lipases (254 U/L , more than three times. Normal range 0-38 U/L) and elevated serum amylase levels (111 U/L. Normal range 0-80U/L).On contrast enhanced computed tomography (CECT) of abdomen and pelvis, head and uncinate process of pancreas was bulky and showed heterogeneous attenuation. Mild peripancreatic fat stranding was seen. Findings were suggestive of acute pancreatitis. Patient was managed conservatively.

After three days of presentation patient developed sudden onset weakness in left upper and lower limb without altered sensorium .MRI Brain/ head /Cranium (Plain and contrast) was suggestive of early subacute hemorrhage in right parieto-occipital region. Subarachnoid hemorrhage seen in right sided cortical

sulci and right ambient cistern and post lumbar puncture status pachymeningeal enhancement was seen.

Contrast enhanced MRI brain was performed on 1.5 T superconducting scanner.

Non contrast axial, sagittal and coronal images of the brain were obtained using FLAIR, inversion recovery T1 and TSE T2 weighted sequences. Along with diffusion and ADC axial images are also obtained. Post contrast T1/FS images were acquired in all planes. A large area of altered signal intensity appearing predominantly hyperintense on both T1 and T2 weighted images is seen in the right parieto occipital lobe showing extensive blooming on SWI s/o early to late subacute intraparenchymal hematoma, measuring 4.4 * 3.0 cm. Moderate perilesional edema is seen which is also involving the posterior body and splenium of corpus callosum. Mass effect was seen in the form of effacement of sulcal spaces and compression of occipital horn of right lateral ventricle .Midline shift of 2mm is seen towards left side.T1/FLAIR hyperintensity is seen in the sulcal spaces in right frontal lobe and along the right sylvian fissure showing blooming on SWI s/o SAH. There is diffuse meningeal enhancement on post contrast images in right cerebral hemisphere, mild lesion in right parieto occipital lobe, likely post hemorrhage related.

Even at the time of onset of weakness blood pressure was 128/82. To rule out other causes of intra cerebral hemorrhage CT brain angiogram and MR venography were done which showed no significant abnormality. Coagulation profile was done which came out to be insignificant.

Bleeding time - 2min45sec , Normal upto 6min
Clotting time - 6min 14 sec , Normal 5-10 min
Prothrombin time - 17 sec , Normal 14-18 sec
Prothrombin time control 14 sec, Normal 14 sec
INR - 1.2 seconds

Other labs were as follows Serum sodium - 139 mmol/L, Serum potassium 3.6 mmol/L, Serum chloride 98 mEq/L, Blood urea 28 mg/dl, Serum creatinine 0.8mg/dl, Serum uric acid 6.0mg/dl

Patient improved in 10 days in terms of power of limbs and started walking with support.

DISCUSSION

Neurological complications in the setting of acute pancreatitis is thought to be due to phospholipase A2 (PLA2) activation that may lead to brain edema by increasing vascular permeability of capillaries causing changes in cell membrane thus causing hemorrhage. Acute pancreatitis also lead to generation of inflammatory cytokines and increase in generation of platelet activating factor causing increased platelet release ,aggregation and further complicating the disease by causing cerebral capillary thrombosis and hypoxia worsening the brain injury.

CONCLUSIONS

In this patient various other causes of intra cerebral hemorrhage were ruled out and there is no history of patient suffering from disseminated intravascular coagulation or hemodynamic instability like elevated blood pressures during the course of illness. Therefore vascular injury caused by acute pancreatitis can be the reason for hemorrhage. Intra cerebral hemorrhage in the setting of acute pancreatitis is a rare presentation so requires a high index of suspicion for diagnosis if a patient develops neurological deficits.

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