Case Report

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Facial Nerve Palsy in a Patient with Acute Pancreatitis: A Complication of Systemic Inflammation or a Mere Co-existence?

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ABSTRACT

Acute pancreatitis can trigger a systemic inflammatory response leading to wide range of complications that could be local or systemic. Viruses have been implicated in most of the infective etiology of acute pancreatitis. Cranial nerve palsy in acute pancreatitis patient is a rare event. A 35-year-old male had mild pancreatitis which resolved with treatment. After a week of admission, he developed right sided facial palsy which gradually improved with oral steroids, acyclovir, and physiotherapy. Whether it was a complication of acute pancreatitis or a mere co-existence secondary to some infective cause could not be proven.

Keywords: Acute pancreatitis; facial nerve palsy; viruses

INTRODUCTION

Almost 80% patients with acute pancreatitis develop a mild or self-limited course but about 20% of the patients suffer from moderate to severe course of the disease, with a mortality rate varying from 13 to 35%.[1,2] A cascade of inflammatory events throughout the body can lead to multiple organ dysfunction syndrome (MODS) in approximately 25% patients.[3] Neurological complications are rare. Various viruses can cause acute pancreatitis as well as facial nerve palsy. Whether cranial nerve palsy is a complication of acute pancreatitis or a coexisting manifestation of an infective virus is not clear.

CASE REPORT

A 35-year-old male was brought to the emergency department with complaint of fever for past one week. He developed shortness of breath, mild abdominal pain in the epigastrium and an episode of vomiting 48 hours before admission. His symptoms gradually worsened over time. The patient denied similar history in the past, any significant past medical or surgical history, intake of drugs, alcohol or smoking. At the time of presentation, the patient was conscious and oriented. However, his HR was 118 beats/min, RR: 35 breaths/min, BP: 90/60 mm Hg, T: 98.7°F and SaO2: 88% in room air. Abdomen examination revealed mild distention and epigastric tenderness. Rectal examination was normal. Cardiac auscultation was normal. Respiratory auscultation revealed crackles over right lung and absence of breath sounds in basal areas of both lungs.

Laboratory investigations at the time of admission showed Hb: 11.3gm/dL, WBC: 22,710/cu mm and platelets: 151,000/cu mm. Urea and creatinine were normal. Serum sodium and potassium were mildly decreased at 129 and 3.4 mmol/L respectively. He had mild elevation of bilirubin but his AST, ALT and ALP were within normal limits. Erythrocyte sedimentation rate and CRP were raised significantly. Random blood glucose was elevated. Lipid profile was within normal range except low HDL levels. Chest X ray showed B/L pleural effusion. SARS- COV- 2 RT PCR was negative. Initial USG of abdomen and pelvis revealed mild splenomegaly and hepatic steatosis. Emergency management was done with oxygen supplementation, IV fluids, Noradrenaline and antibiotics targeting local microbiological profile. The patient was transferred to the ICU for further treatment. Serum amylase level was within normal range but lipase level was elevated to 426 U/L. Computed tomography abdomen performed on the second day of admission revealed bulky pancreas with minimal peri-pancreatic fat strandings (Figure 1) : suggestive of acute mild pancreatitis with CT severity index of 4/10. Mild pericardial effusion, bilateral pleural effusion and mild ascites were other notable findings. Serological tests for HIV, HBV, HCV and Tropical fever panel for (Leptospira, Scrub, Brucella, Malaria, and Dengue) came negative. Blood and urine culture revealed no growth.

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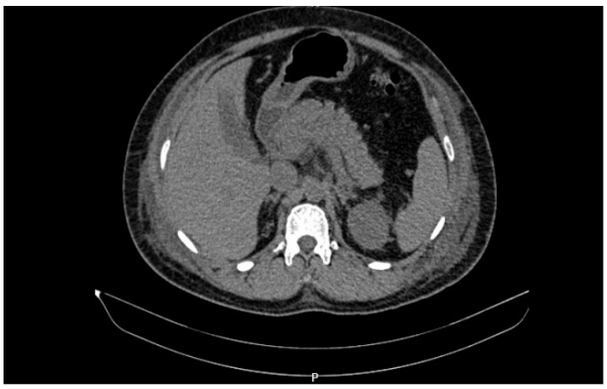

Figure 1. Computed tomography scan of abdomen showing features of pancreatitis.

Subsequently within 24 hours, he developed pedal edema, scrotal swelling and ascites. Oral feeding was commenced after 48 hours of admission. Pleural fluid analysis revealed transudate with normal amylase and cytology reports. Sputum culture came positive. Over next few days, he showed signs of improvement and was shifted to ward for further observation and management. Herpes Simplex Virus IgG and IgM serology titers came non-significant (0.26 and 0.13 respectively). Patient had raised IgG level against Mumps (70.10 AU/mL), however IgM titers were normal (0.20). Antinuclear antibody titer was within normal range. He had persistent elevation of serum lipase levels for almost a week which then gradually began to fall (Table 1).

Table 1. Serial measurement of amylase and lipase levels.

	On admission day (12/23)	Day 2 (12/24)	Day 6 (12/28)	Day 9 (12/31)	On the day of discharge (1/10)	7 wks later (2/14)
Lipase	426 U/L	998	1650	1337	1114	460
Amylase	73 U/L	148	293	-	258	125

After seven days of admission, the patient noticed weakness of his right side of face (Figure 2) which gradually increased in severity. He had difficulty in blowing and chewing meals. Water and other liquid food would dribble down his angle of mouth on right side. He couldn't firmly close his right eyelid. He had normal sensation over both sides of face. He did not have loss of taste, weakness of tongue and hoarseness of voice. Sensation and power of his all four limbs were normal and comparable on either sides. He was diagnosed with lower motor neuron type of right facial nerve palsy.

Due to new onset symptoms and possibility of other viral infections (VZV, CMV, Coxsackie etc.) clinical decision was made to start patient on antiviral and corticosteroids. After two weeks of treatment his facial weakness improved significantly (Figure 3). Amylase and lipase levels remained high for total eight weeks with gradual decline. Repeat CT abdomen scan after eight weeks showed normal pancreas.

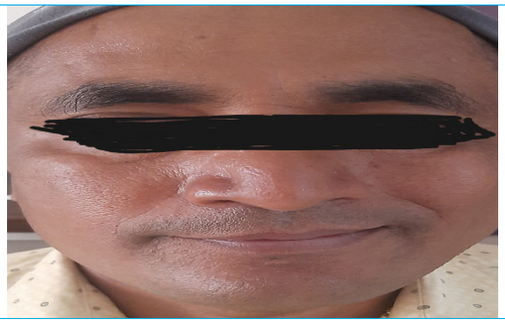

Figure 2. Right sided LMN type facial palsy.

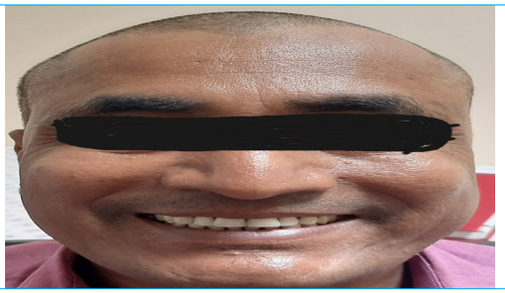

Figure 3. Improving right sided facial palsy.

DISCUSSION

Acute pancreatitis may present with wide range of complications. Local complications include peri-pancreatic/pancreatic fluid collection, pancreatic/peri-pancreatic necrosis and pseudocyst formation. As systemic complications, acute pancreatitis may lead to third space fluid loss, ARDS and DIC. Uncommon complications such as retinopathy and stroke may be seen in some patients.[4,5] Few cases of peripheral neuropathy have been reported following acute pancreatitis.[6] However cranial nerve palsy as a complication of acute pancreatitis is a rare event. One case of bilateral Adie's pupil with bilateral facial nerve palsy, following acute pancreatitis has been reported.[7] Many viruses including HSV, CMV, VZV, Mumps, etc. have been implicated for acute pancreatitis as well as LMN type facial nerve palsy.[8] One of these could have been responsible for the clinical presentation in our patient as well.

CONCLUSIONS

Cranial nerve palsy as a neurologic complication in a patient with acute pancreatitis is a rare occurrence. Systemic inflammation in acute pancreatitis may trigger

neuronal inflammation which can lead to neurological deficits such as facial nerve palsy. Similarly, various viruses can lead to both acute pancreatitis as well as facial nerve palsy. The coexistence of facial nerve palsy in a patient with acute pancreatitis demands detailed virological workup as a part of diagnostic tool which could shed some lights to clear up the confusion in such scenario.

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Competing interests: None declared

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