

# EC GASTROENTEROLOGY AND DIGESTIVE SYSTEM

**Case Report** 

# Acute Pancreatitis Caused by Mumps in Adulthood: An Unusual Etiology

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#### **Abstract**

A 49-year-old female presented an acute pancreatitis caused by infection with mumps virus. The clinical context including the absence of the usual causes of acute pancreatitis and the appearance of the parotidis, oriented more specifically towards the mumps virus.

Acute pancreatitis was diagnosticated by the increase of lipase and by enlargement of the pancreas seen on abdominal computed tomography. Mumps-specific antibodies confirmed mumps infection serologically. The abdominal symptoms were disappeared spontaneously after a conservation management with analgesia, hydration and fasting and the swelling of the right parotid decreased too.

Mumps infection in adulthood is rare, and acute pancreatitis is an exceptional complication of mumps virus. However, this diagnosis have to be considered in patients presenting with acute pancreatitis without evidence of another usual cases.

Keywords: Mumps; Acute Pancreatitis; Parotidis

## Introduction

Acute pancreatitis is one of the most common gastrointestinal causes for emergency admission with high morbidity and mortality.

In most cases, acute pancreatitis is associated with gallstones and alcohol consumption, whereas in 10% of cases diverse causes like infections viruses are responsible [1,2].

Pancreatitis arises in about 5% of mumps infections [3]. In the literature, 0.31 to 15% of patients had Symptoms compatible with mumps associated pancreatitis [4].

Mumps is a usual viral infectious disease which generally concern children who then mostly get permanent immunity [5]. Mumps virus infection usually induce lifelong immunity [1], but some cases of mumps virus re-infection have been reported [5]. Those reinfected patients present less serious and less typical symptoms [6].

To prove the relation between mumps virus and acute pancreatitis, we focus on the imaging evidence of pancreatitis combined with laboratory data, and we exclude common causes of acute pancreatitis [4].

### **Case Report**

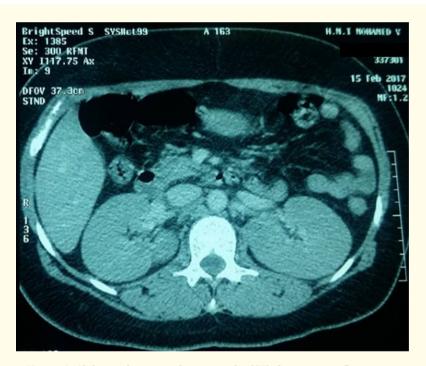
In this paper, we report a case of an adult woman who developed acute pancreatitis due to a reinfection with mumps virus. The infection was confirmed serologically by mumps-specific IgM and IgG.

A 49-year-old woman presented to the emergency and accused since 3 days an increasing epigastric pain irradiating to the back and two episodes of vomiting. She had a history of cholecystectomy 6 years ago.

Examination revealed during abdominal palpation an epigastric pain with no peritonitis signs and hyperthermia at 39°.

Laboratory investigations indicate (we include in parentheses reference ranges of laboratory): Serum lipase 5936 IU/L (< 78 UI/L), C reactive protein 256 mg/l and total leukocyte count of 33400/L with lymphocytopenia of 500/l. The hepatobiliary enzymes were normal with the exception of gamma-glutamyl transpeptidase 163 IU/L (< 32 IU/L): aspartate aminotransferase 17 IU/L (< 40 IU/L), alanine aminotransferase 11 IU/L (< 40 IU/L), alkaline phosphatase 78 IU/L (32 - 91 IU/L) and total bilirubin 11 mg/dL (3 - 12 mg/dL). There was no hypercalcemia and hypertriglyceridemia: Calcium 85 mg/L (80 - 105 mg/L) and triglycerides 0, 64 g/l (1,5 g/l). She did not drink alcohol.

Abdominal computed tomography (CT) showed inflammatory changes in pancreas and peripancreatic fat corresponding to stage C of Balthazar Grade (Figure 1). It showed a normal biliary tree and liver.

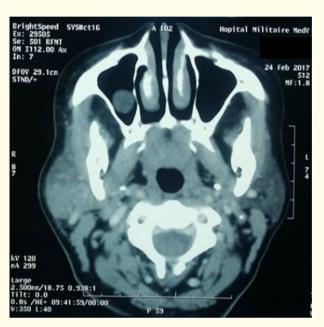


**Figure 1:** Abdominal computed tomography (CT) showing an inflammatory changes in pancreas and peripancreatic fat corresponding to stage C of Balthazar Grade.

The patient received conservation management with analgesia, hydration and fasting. No Antibiotics were given.

We tried to exclude usual causes of acute pancreatitis. Our patient was abstinent of alcohol and did not take drugs. She had a normal serum calcium and triglyceridemia. She had no family history of pancreatitis or endocrine diseases and had no history of abdominal trauma. The abdominal CT scan did not showed gallstones. We performed abdominal magnetic resonance imaging during the period of hospitalization and no dilatation of the common bile duct was detected.

During the follow up, abdominal pain was resolved on day 4. But on Day 5, the patient accused an unilateral parotid swelling, with gradual worsening. At contrast-enhanced CT showed an expansion of the right parotid gland (Figure 2) without abscesses or ductal obstruction. The association of acute pancreatitis and parotitis suggested highly a viral aetiology such as with mumps.



**Figure 2:** At contrast-enhanced CT showing an enlargement of the right parotid gland without abscesses or ductal obstruction.

There was no past history of mumps. She had received measles-mumps-rubella vaccine in the Childhood. We performed Mumps IgM and IgG with enzyme linked immunosorbent assay (ELISA) on day 7 after admission. It showed 1.3 EIA of antibody indices (Index value < 1 = negative) and 2,5IA (Index value < 1 = negative), respectively. The Positivity of IgG and IgM result suggests recent infection with mumps virus.

During the hospitalization, the hyperthermia disappear and the patient did not accuse abdominal pain. No exacerbation of parotid swelling or pain occurred.

#### Discussion

Mumps virus was incriminate for the first time as a cause of acute pancreatitis in 1817. Then, many reports combined pancreatitis and viral infections were published.

The Diagnosis of pancreatitis due to mumps virus is based fundamentally on clinical signs, detecting specifics antibodies and on the elimination of other causes of pancreatitis [7].

Mumps infectious is engendered by a single-stranded RNA virus belonging to the genus Rubula virus and the family Paramyxoviridae. It causes infection of the salivary glands, especially parotitis which can be complicated by orchitis, arthritis, meningoencephalitis and pancreatitis [7].

Mumps is highly infectious. The most common mumps transmission is through coughing or sneezing by respiratory droplet. The virus will be multiply during a period of 10 days of viraemia -before symptoms appearance- during which other organs can become infected [8].

Natural infection can confer lifelong immunity, but reinfection can occur and give atypical and less severe symptoms than the primary infection [9].

Pancreatitis occurs around 4% of mumps infections and appears in mild form. Severe haemorrhagic pancreatitis has been rarely reported [10].

After an incubation period of 16 days may occur prodromes as headache, anorexia, generalised myalgia and fever. Then can happen otalgia and tenderness of the parotid glands before parotitis [3].

Abdominal pain usually occur at 4 to 8 days of illness [11]. Sometimes abdominal symptoms begin prior to parotitis like in our case. Most patients recover without sequelae [4].

Suspected mumps infection can be confirmed by detection of specific antibodies. It can be approved by detection of viral RNA by reverse transcriptase-polymerase chain reaction (RT-PCR) techniques or viral cultures of microbiological samples [12,13].

Serologically diagnosing of Mumps infection relies on detection of IgM [14]. IgM can be detectable in serum 11 days after exposure, before the appearance of symptoms. It seems that the optimal time for detection is probably 7 - 10 days after the patient appearance of symptoms [15].

However, false negative IgM result can happen. It concern people previously immunised or infected [16]. In this case, a rise in serum IgG rate should be considered as an indicator of current infection [17].

In our case, the patient was suspected of having been infected with mumps because of mumps IgM and IgG positivity.

The interest of the observation concerns the rare cause of a pancreatitis occurred in a woman of 49 years.

The diagnosis of acute pancreatitis was rapidly brought to a typical symptomatology of solar pain and increased lipase.

Common causes of pancreatitis were excluded. she was abstinent of alcohol an didn't take drugs. Laboratory findings did not show hypercalcemia and hypertriglyceridemia. Hepatobiliary enzymes were normal excepted of elevate rate of GGT and Abdominal CT ultrasonography on admission did not reveal stones or sludge in the gallbladder or the bile.

In our case, the clinical context including the absence of the usual causes of acute pancreatitis, hyperthermia at 39°C and lymphocytopenia directed towards an infectious viral origin. The appearance of the parotitis then oriented more specifically towards the mumps virus. In our observation, mumps serology indicated a recent infection. In retrospect, the patient was vaccinated and did not report a history of mumps in childhood.

Our observation accurately reflects the interest of thorough research of all known causes of acute pancreatitis, regardless of age, before classifying them as idiopathic.

Considering the clinical course and laboratory data, we diagnosed our patient as having acute pancreatitis due to mumps.

Accordingly, mumps infection should be considered as a possible cause of acute pancreatitis in adults, even though reinfection in adulthood is rare.

#### **Conclusion**

We present a case of acute pancreatitis from mumps re-infections in adult woman who had no evidence of other usual case of pancreatitis. The association of acute pancreatitis and parotitis suggested highly a viral aetiology with mumps. This diagnosis should be considered in patients presenting with acute pancreatitis and no evidence of usual cases.

#### **Conflicts of Interests**

None.

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