

CASE REPORT

Acute Pancreatitis from Mumps Re-infection in Adulthood. A Case Report

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ABSTRACT

Context Acute pancreatitis is a complication of mumps which mainly affects children who then usually acquire permanent immunity. We present the case of a woman with acute pancreatitis caused by mumps re-infection in adulthood.

Case report A 34-year-old woman developed mild acute pancreatitis caused by re-infection with mumps, as confirmed serologically by enzyme-linked immunosorbent assays mumps-specific IgM and IgG. Acute pancreatitis was indicated by the elevation of amylase and other pancreatic enzymes such as lipase and elastase-1 as well as by swelling of the pancreatic head visualized by abdominal computed tomography. The abdominal symptoms were resolved soon after the administration of a pancreatic enzyme inhibitor. As the swelling of the right and left parotids decreased, serum amylase levels also gradually normalized.

Conclusion We believe that this is the first reported case of acute pancreatitis caused by mumps re-infection in an adult. Such re-infection should be considered a possible though rare cause of acute pancreatitis in adulthood.

INTRODUCTION

Mumps is a common viral infectious disease which mainly affects children who then

usually acquire permanent immunity [1]. In the early stage of infection, mumps can have various complications such as orchitis, aseptic meningitis, oophoritis, and pancreatitis [2]. Symptoms compatible with mumps-associated pancreatitis have been reported in 0.31 to 15% of patients [3]. The latest British epidemic report in 2005 gives an incidence rate for acute pancreatitis of approximately 5% [2].

Even though one attack of mumps usually confers lifelong immunity [1], cases of mumps virus re-infection have been reported [4]. We describe an adult woman who developed acute pancreatitis after a re-infection with mumps which was confirmed by serologically, enzyme-linked immunosorbent assay (ELISA) mumps-specific IgM and IgG. To the best of our knowledge, this is the first reported case of acute pancreatitis caused by mumps re-infection in an adult.

CASE REPORT

A 34-year-old woman presented to our hospital with increasing epigastric pain which had developed the day before. Serum amylase was 463 IU/L (reference range: 35-133 IU/L). Abdominal computed tomography (CT) showed a slight swelling of the pancreatic head and edema of the adjacent duodenum (Figure 1). She was admitted with a provisional diagnosis of mild acute pancreatitis. She had a 17-year history of anorexia nervosa; on admission, her body-

mass index (BMI) was 15.1 kg/m². Abdominal CT showed a dilated common bile duct, but the hepatobiliary enzymes were not elevated with the exception of gamma-glutamyl transpeptidase (GGT: 936 IU/L; reference range: 3-54 IU/L): aspartate aminotransferase 32 IU/L (reference range: 12-35 IU/L), alanine aminotransferase 11 IU/L (reference range: 6-33 IU/L), alkaline phosphatase 276 IU/L (reference range, 120-362 IU/L) and total bilirubin 0.77 mg/dL (reference range: 0.2-1.0 mg/dL). The laboratory data on admission did not show hypercalcemia and hypertriglyceridemia: calcium 8.8 mg/dL (reference range: 8.6-10.5 mg/dL) and triglycerides 73 mg/dL (reference range: 31-150 mg/dL). She drank about 80 mL of alcohol per day, but the amount of intake had not changed notably in the days before admission. Two years previously when her alcohol intake was at the maximum (twice the present intake), her GGT was 2,196 IU/L. Other pancreatic enzymes in serum at the present admission also indicated acute pancreatitis: lipase 455 U/L (reference range: 11-53 U/L) and elastase-1 2,600 ng/dL (reference range: 100-400 ng/dL). We performed abdominal ultrasonography several times during the period of hospitalization, but dilatation of the common bile duct was not detected.

The patient was treated intravenously with a pancreatic enzyme inhibitor. Serum amylase



Figure 1. Abdominal computed tomography on admission showing a slight swelling of the pancreatic head and adjacent edema of the duodenum (circle).



Figure 2. Bilateral parotid swelling on hospital day 9.

declined to 348 and 186 IU/L on the second and third day, respectively. Epigastric pain and tenderness were resolved completely on day 3. On day 4, she complained of marked bilateral parotid swelling (Figure 2) and numbness at the side of the forehead. She had noted some parotid swelling as early as the day after admission, with gradual worsening. We consulted with an otolaryngologist. Mumps was suspected. She did not declare a past history of mumps, and she had not received measles-mumps-rubella (MMR) vaccine. Serum amylase peaked at 1,229 IU/L on day 6 when p-amylase was 93 IU/L (reference range: 8-49 IU/L).

Pair sera for mumps IgM and IgG enzyme-linked immunosorbent assay (ELISA) from the acute period and then 2 weeks later suggested her re-infection with mumps. Mumps IgM and IgG ELISA on day 6 after admission showed 1.11 EIA of antibody indices (reference range: 0-0.79 EIA) and 9.20 EIA (reference range: 0-1.90 EIA), respectively. On day 20, they were 1.12 and 11.50, respectively. No prodromal symptoms were present in the patient.

Bilateral parotid swelling, as well as numbness at the side of the forehead, decreased gradually to resolve fully within 2 weeks of their appearance. At the same time, serum amylase fell simultaneously to 218 IU/L, with p-amylase declining to 78 IU/L 2 weeks after the symptoms had developed. Serum amylase decreased to the patient's baseline serum amylase which had been

measured one month before admission at an ambulatory clinic and, at that time, was slightly high at 161 IU/L. The amylase-to-creatinine-clearance ratio (ACCR) just before discharge was 0.71%; macroamylasemia was presumed to be present. We observed that serum amylase did not decrease to the normal level even after she recovered from parotid swelling and acute pancreatitis due to macroamylasemia. After resumption of an oral diet, there was not much change in amylase level.

Throughout the hospitalization, the patient was afebrile without pain in either parotid.

Abdominal ultrasonography on day 13 disclosed a normal pancreas and duodenum. Even with the resumption of an oral diet, no accompanying abdominal symptoms or exacerbation of parotid swelling occurred. Serum concentrations of amylase and other pancreatic enzymes also decreased each day; lipase was 101 U/L and elastase-1 was 480 ng/dL on the day of discharge, about 3 weeks after admission.

DISCUSSION

Mumps is an acute contagious RNA paramyxovirus disease typically causing painful swelling of the parotid gland [5]. Since mumps is the most common cause of inflammatory parotitis and infections caused by other viruses are rare, routine screening of patients for other causes of parotitis seems unnecessary [6]. Mumps is transmitted by direct contact with infectious droplet nuclei, fomites containing infectious saliva or possibly urine [5]. As 30% of infections are subclinical, mumps complications such as pancreatitis can occur without parotid enlargement [7]. Following an incubation period of 2 to 4 weeks, prodromal symptoms of low-grade fever, malaise and headache may be followed by characteristic parotid swelling [3]. Mumps is principally a disease of children and young adults [1]. According to a report from Britain in 1978, 92% of 15-year-old subjects had antibodies against mumps [8]. While the symptoms are similar, illnesses are usually more severe in adults and adolescents than in children [2].

Complications include orchitis, meningio-encephalitis, arthritis, and pancreatitis [3]. Symptoms compatible with acute pancreatitis were described in 0.31 to 15% of patients with mumps [3]. Current clinical reviews cite a rate of pancreatitis ranging up to 5% [2]. Experimental infection in rats suggested a relationship between the pancreas and the parotid [9], but the mechanism leading to changes of amylase activities in parotid glands as a result of pancreatitis is unknown [10].

Abdominal pain and tenderness characteristically occur at 4 to 8 days of illness [11], although abdominal symptoms occasionally begin prior to development of parotitis as in our case. Except for two deaths reported in the 1920s and two cases with severe complications in the 1960s [12] and 1970s [13], most patients recover without sequelae [3].

Mumps IgM ELISA is a more rapid and sensitive way of serologically diagnosing mumps infection [14] than conventional tests such as IgG ELISA, complement fixation (CF), hemagglutination inhibition (HI), and hemolysis-in-gel (HIG) tests [15]. According to mumps IgM and IgG ELISA, our patient was suspected of having been reinfected with mumps. Although one attack of mumps usually confers lifelong immunity [1], cases of mumps virus re-infection have been reported [4]. Such reinfected patients tend to show less severe and less typical symptoms [4]. The incidence of acute pancreatitis as a complication differs little between primary and secondary mumps infection [4] while, regarding other complications, lymphocytic meningitis is less frequent in reinfected patients, and orchitis occurs with a similar frequency in the two groups [4].

We can not deny the possibility of alcohol and anorexia nervosa as factors of acute pancreatitis, but mumps is the most probable cause of acute pancreatitis in this case. Pancreatitis is not common in anorexia nervosa, and occasional cases of acute pancreatitis have been reported in the refeeding phase [16]. The association between salivary gland swelling and eating disorders is

well known [16]. The patient sometimes noticed a slight enlargement of the submandibular gland when she repeated cycles of binge-eating and vomiting; these signs and symptoms were not present immediately before and during this admission. For this reason, anorexia nervosa was not considered to be the main cause of pancreatitis in this patient. Abdominal CT and ultrasonography on admission did not disclose stones or sludge in the gallbladder or the bile duct. Hepatobiliary enzymes elevated only her GGT. The high value of her GGT is considered to reflect the decreasing alcohol intake for this patient, and the GGT had declined over the past few years. The amount of alcohol intake had not changed notably in the days before admission, and alcohol was not considered to be the main cause of pancreatitis in this patient. Laboratory findings did not include hypercalcemia and hypertriglyceridemia. Antinuclear antibodies were not detected. Considering the clinical course and laboratory data, we diagnosed our patient as having acute pancreatitis due to mumps.

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

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References

1. Gershon A. Mumps. In: Fauci AS, Braunwald E, Isselbacher KJ, et al, eds. Harrison's Principles of Internal Medicine, 14th Ed. New York: McGraw-Hill, 1998:1127-8. [ISBN: 0071348336]
2. Gupta RK, Best J, MacMahon E. Mumps and the UK epidemic 2005. *BMJ* 2005; 330:1132-5. [PMID 15891229]
3. Parenti DM, Steinberg W, Kang P. Infectious causes of acute pancreatitis. *Pancreas* 1996; 13:356-71. [PMID 8899796]
4. Gut JP, Lablache C, Behr S, Kirn A. Symptomatic mumps virus reinfections. *J Med Virol* 1995; 45:17-23. [PMID 7714488]
5. Lane TM, Hines J. The management of mumps orchitis. *BJU Int* 2006; 97:1-2. [PMID 16336315]
6. Meurman O, Vainionpää R, Rossi T, Hänninen P. Viral Etiology of Parotitis. *Scand J Infect Dis* 1983; 15:145-8. [PMID 6308753]
7. Witte CL, Schanzer B. Pancreatitis due to mumps. *JAMA* 1968; 203:1068-9. [PMID 5694373]
8. Mortimer PP. Mumps prophylaxis in the light of a new test for antibody. *BMJ* 1978; 2:1523-4. [PMID 365288]
9. Kakizaki G, Sasahara M, Soeno T, Shoji S, Ishidate T, Senoo A. Mechanism of the pancreas-parotid gland interaction. An experimental study in parabiotic rats. *Am J Gastroenterol* 1978; 70:635-44. [PMID 742615]
10. Skrha J, Stěpán J, Sixtová E. Amylase isoenzymes in mumps. *Eur J Pediatr* 1979; 132:99-105. [PMID 499265]
11. Edgecombe W. Metastatic affection of the pancreas in mumps. *Practitioner* 1980; 80; 194-9.
12. O'Brien PK, Smith DS, Galpin OP. Acute pancreatitis and haemolytic anaemia associated with mumps-virus infection. *BMJ* 1965; 2:1529. [PMID 5850471]
13. Feldstein JD, Johnson FR, Kallick CA, Doolas A. Acute hemorrhagic pancreatitis and pseudocyst due to mumps. *Ann Surg* 1974; 180:85-8. [PMID 4835960]
14. Nicolai-Scholten ME, Ziegelmaier R, Behrens F, Höpken W. The enzyme-linked immunosorbent assay (ELISA) for determination of IgG and IgM antibodies after infection with mumps virus. *Med Microbiol Immunol* 1980; 168:81-90. [PMID 7453669]

15. Ukkonen P, Granström ML, Penttinen K. Mumps-specific immunoglobulin M and G antibodies in natural mumps infection as measured by enzyme-linked immunosorbent assay. *J Med Virol* 1981; 8:131-42. [PMID 7028917]

16. Sharp CW, Freeman CP. The medical complications of anorexia nervosa. *Br J Psychiatry* 1993; 162:452-62. [PMID 8481735]
