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Case Report in Gastroenterology

Reactive Hyperlipasemia Or True Pancreatitis? Pancreatic Involvement In Salmonella Infection: A Case Report And Review Of The Literature.

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Abstract

Pancreatic involvement during *Salmonella* infection is uncommon and its clinical significance remains debated. While some reports describe true acute pancreatitis, others suggest reactive hyperlipasemia without structural pancreatic injury. We present a case of *Salmonella* gastroenteritis with marked pancreatic enzyme elevation but no radiological evidence of pancreatitis. A 49-year-old healthy man developed abdominal pain, fever, vomiting, and profuse diarrhea after consuming contaminated food. Laboratory tests revealed markedly elevated amylase (385 U/L) and lipase (1814 U/L), along with severe inflammation (CRP 249 mg/L). Stool culture was positive for Salmonella group; blood cultures were negative. Contrast-enhanced CT showed no pancreatic abnormalities. The patient improved with supportive therapy and was discharged asymptomatic after eight days, with almost normalization of laboratory values.

This case highlights the diagnostic challenge of differentiating true pancreatitis from reactive pancreatic enzyme elevation in Salmonella infection. Literature suggests that pancreatic hyperenzymemia is relatively common, whereas true pancreatitis is rare. Clinicians should consider pancreatic involvement in Salmonella infection when pancreatic enzymes are elevated, but imaging is essential to avoid overdiagnosis. Further studies are needed to clarify the mechanisms and clinical relevance of pancreatic enzyme abnormalities in enteric infections.

Keywords: Acute pancreatitis; Gastroenteritis; Pancreatic hyperenzymemia; Reactive hyperlipasemia; *Salmonella* infection.

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INTRODUCTION

Acute pancreatitis is a common disease with a broad spectrum of clinical severity and potential local and systemic complications. Gallstones and alcohol account for 50-70% of cases and remain the most frequent etiologies [1]. Other causes include hypertriglyceridemia, post-endoscopic retrograde cholangiopancreatography (ERCP), genetic predisposition, medications, as well as rarer causes such as hypercalcemia, infections and toxins [2]. Among infectious agents, enterovirus, measles, and mumps are the most frequently implicated, whereas Salmonella-induced pancreatitis is rarely reported and remains poorly understood [3]. The clinical significance of pancreatic enzyme elevation during Salmonella infection is debated, with some authors suggesting true pancreatic involvement and others proposing reactive hyperenzymemia without structural pancreatic injury [3].

We describe a case of Salmonella gastroenteritis presenting with abdominal pain and marked enzyme elevation, raising suspicion of acute pancreatitis.

CASE REPORT

A 49-year-old healthy man presented to the Emergency Department with abdominal pain, fever, vomiting, and diarrhea. Symptoms began the day after consuming fish and vegetables imported from the Philippines, together with 12 other individuals. Seven diners—specifically those who had eaten the vegetables—developed similar symptoms. The patient was initially discharged with supportive care.

He returned the following day due to persistent symptoms, including more than 15 diarrheal episodes and mild fever. He denied recent alcohol intake.

On admission, vital signs were stable: afebrile, heart rate 80/min, blood pressure 105/70 mmHg. Physical examination revealed moderate dehydration, but no abdominal tenderness or peritoneal signs.

Laboratory investigations are summarized in Table 1. Notably, serum amylase was 385 U/L (reference 10–100) and lipase 1814 U/L (reference 13–60); C-reactive protein was markedly elevated (249 mg/L). Stool culture was positive for *Salmonella* group, while blood cultures were negative. Serology showed positivity for O antigen of paratyphoid B.

Contrast-enhanced abdominal computed tomography (CT) revealed no pancreatic abnormalities.

The patient received supportive therapy and was discharged after eight days, fully asymptomatic and with almost normalization of laboratory values.

Table 1. Laboratory investigations on admission and at discharge.

Laboratory test	Value on admission	Value at discharge	Reference values
WBC (10^9/L)	8.40	6.60	4-10
Neutrophil (%)	68		
Lymphocyte (%)	18		
Monocyte (%)	14		
Eosinophil (%)	0		
Basophil (%)	0		
RBC (10^{12}/L)	5.50	4.48	4.0-5.7

Hgb (g/dL)	15.6	13.8	13.5-16
Hct (%)	44.4	40.0	39-49
MCV (fL)	88	89	80-96
Plt (10^9/L)	120	179	150-450
CRP (mg/L)	249	9	0-5
Glucose (mg/dL)	121	83	70-110
Urea (mg/dL)	52	25	10-50
Creatinine (mg/dL)	1.27	0.86	0.7-1.2
AST (units/L)	35	31	5-50
ALT (units/L)	28	36	5-50
Alkaline phosphatase (units/L)	29		44-129
Amylase (units/L)	385		10-100
Lipase (units/L)	1814	742	13-60
Total protein (g/dL)	7.44		6.6-8.7
Albumin (g/dL)	3.77		3.5-5.2
Total bilirubin (mg/dL)	0.37		0.2-1.0
Direct bilirubin (mg/dL)	0.14		< 0.4
Triglycerides (mg/dL)	131		< 170
Calcium (mg/dL)	9.04		8.5-10.5
Antibody research paratyphoid A paratyphoid B antigen O typhoid antigen H typhoid	Negative Negative Positive Negative		
Blood culture	Negative		
Stool culture	Salmonella group		

Note: WBC: white blood count; RBC: red blood cell; Hgb: hemoglobin; Hct: hematocrit; MCV: mean corpuscular volume; Plt: platelet; CRP: C-reactive protein; AST: aspartate aminotransferase; ALT: alanine aminotransferase

DISCUSSION

According to the revised Atlanta Classification [1], the diagnosis of acute pancreatitis requires two of the following: (1) characteristic abdominal pain, (2) serum lipase or amylase greater than 3 times the upper limit of normal, (3) imaging findings consistent with pancreatitis. Our patient met the first two criteria; however, CT imaging excluded pancreatic inflammation.

In the absence of common etiologies—alcohol use, gallstones, hypertriglyceridemia, hypercalcemia, trauma, or drug-induced injury—the marked lipase elevation raised suspicion of Salmonella-associated pancreatic involvement.

Occasionally, microbial pathogens have been suggested to induce acute non-alcoholic, non-biliary pancreatitis [4].

Among bacterial infectious agents, Salmonella—both nontyphoidal and typhoid fever—has occasionally been associated with pancreatitis.

Pancreatic involvement in Salmonella infection has been described in isolated case reports [5–27] and small retrospective or prospective studies [4, 28-33]. However, its true incidence and clinical significance remain controversial.

Renner et al. [29] conducted a retrospective study on patients with Salmonella enteritis and reported that concomitant pancreatitis was observed in 62% of patients and that the course of pancreatitis was mild to moderate in most of the patients.

Murphy et al. [4] found no clinically apparent pancreatitis among 51 infected patients, with only one showing mild enzyme elevation.

In a prospective study of 30 patients with salmonellosis, Pezzilli et al. [32] observed elevated lipase levels in 16.7% of patients, but no cases of pancreatitis.

A recent retrospective study on 99 Salmonella cases reported a 3% incidence of pancreatitis, all mild and occurring 5–11 days after diarrhea onset [3].

The study found that many patients with salmonellosis had elevated lipase levels without clinical pancreatitis. These findings suggest that many cases labeled as "pancreatitis" may instead represent reactive hyperlipasemia, without structural pancreatic damage.

The main pathophysiological hypotheses may include different mechanisms.

First, increased intestinal permeability during severe gastroenteritis may allow bacterial translocation or endotoxin migration to mesenteric lymph nodes, portal circulation, and pancreatic tissue [16].

Direct bacterial invasion of the pancreatic duct from the duodenum or biliary tree has been hypothesized [29, 34].

Secondly, Salmonella may induce the production of inflammatory and immune mediators, like arachidonic acid metabolites, nitric oxide, and pro-inflammatory cytokines [35, 36]. These mediators can amplify pancreatic inflammation, especially in predisposed subjects.

Finally, the severity of diarrhea may lead to dehydration, with consequent alteration of the hydroelectrolytic balance and duodenal motility; in this way biliary or duodenal reflux into the pancreatic duct may be favored (a mechanism similar to post-ERCP pancreatitis) [4].

CONCLUSIONS

Salmonella-associated pancreatitis is a rare, likely underdiagnosed, but documented phenomenon. Distinguishing true pancreatitis from reactive hyperlipasemia can be challenging, particularly in patients with prolonged diarrhea and abdominal pain.

Clinicians should consider pancreatic involvement in Salmonella infection when pancreatic enzymes are elevated, but imaging remains essential to avoid overdiagnosis.

Further studies are needed to clarify the pathophysiological relationship between Salmonella infection and pancreatic enzyme abnormalities.

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