

## Case report

# Acute necrotizing pancreatitis caused by atheromatous embolization during Percutaneous Transcatheter Coronary Angioplasty (PTCA)

S.C. Trygonis<sup>1</sup>, Eleni T.Tsiaousi<sup>2</sup>, S.E. Miliaras<sup>1</sup>

## SUMMARY

Although acute pancreatitis is a common disease some of its causes are quite rare such as ischemia of the pancreas. One of the causes of pancreatic ischemia is atheromatous embolization of the pancreatic vessels during angiographic procedures either for diagnosis or for treatment. We present the case of a male patient who suffered acute pancreatitis of pancreatic ischemia origin due to atheromatous embolization of the pancreas during percutaneous transcatheter coronary angioplasty (PTCA) of the left coronary artery.

**Key words:** Acute pancreatitis, angiography, cardiac catheterization

## INTRODUCTION

Acute pancreatitis (AP) is a common disease caused mainly by gallstone disease or chronic alcohol consumption. Pancreatic ischemia which can result in acute pancreatitis is quite rare. One of the causes of pancreatic ischemia is atheromatous embolization of the pancreatic vessels during transcatheter angiographic procedures. We present the case of a male patient who underwent percutaneous transcatheter coronary angioplasty (PTCA). Twenty-four hours after the procedure he developed acute pancreatitis and 22 days later died from massive pulmonary embolism.

<sup>1</sup>1<sup>st</sup> Surgical Clinic of the Aristotle University of Thessaloniki, "Papageorgiou" General Hospital, Thessaloniki, Greece,

<sup>2</sup>1<sup>st</sup> Propedeutic Clinic of Internal Medicine of Aristotle University of Thessaloniki, AHEPA University Hospital, Thessaloniki, Greece,

### Author for correspondence:

Sotirios C. Trygonis, Stilp. Kyriakidi 5, 54636, Thessaloniki, Greece, Tel.: 0030-2310-249133, 0030-6944-913388, Fax: 0030-2310-991581, e-mail: [trygonis@hotmail.com](mailto:trygonis@hotmail.com)

## CASE REPORT

A 67-year old, male patient was urgently admitted into the cardiology department suffering from acute myocardial infarction (MI). On admission, the patient's vital signs were; Blood Pressure 190/80 mmHg, Heart Rate 95/min and sO<sub>2</sub> 87%. He reported an ambiguous history of heart disease but he was not currently on any medication. He underwent an emergency cardiac catheterization which revealed severe stenosis (95%) of the anterior descending branch (ABD) of the left coronary artery. Therefore, a PTCA of ABD was performed. After the PTCA, the patient received 40 ml/h of Tirofiban Hydrochloride as antiplatelet therapy.

Twenty four hours after the procedure, the patient complained of acute epigastric pain reflecting on his back. Laboratory tests showed increased WBC (leucocytes: 21,210/mm<sup>3</sup>, poly: 93,2%) and increased serum amylase levels (AMY: 1,135 IU).

An abdominal Computed Tomography scan (CT) was performed and revealed acute pancreatitis with severe pancreatic edema and peripancreatic fluid collections. The most interesting finding was a necrotic lesion in the head of the pancreas (Figure 1). The patient was transferred into our department for further assessment and management. He was supported with intravenous fluids, analgesics and enteral feeding through a nasogastric tube. He also received the following treatment for MI: Clopidogrel, Acetylsalicylic Acid, Enoxaparin Sodium, Furosemide, Quinapril and Glycerin Trinitrate. The patient remained stable for two weeks. On day 15 his clinical condition deteriorated. A new CT scan was performed and revealed extended pancreatic necrosis which was proved to be sterile after CT-guided fluid aspiration. According to the CT Severity Index Scoring System (CTSI) proposed by Baltha-



**Figure 1.** An abdominal CT scan showing acute pancreatitis with a necrotic lesion in the head of the pancreas (white arrowhead)

zar et al<sup>[1]</sup> the patient received a CTSI score 7 (Balthazar grade D plus Necrosis score 4).

The patient was transferred to the Intensive Care Unit for invasive cardiopulmonary support and monitoring. Despite maximum support with mechanical ventilation and medication, the patient died 7 days later from massive pulmonary embolism. Autopsy was performed and confirmed the pulmonary embolism as the cause of death. Extended necrosis of the head and body of the pancreas, as well as atheromatosis of the coronary vessels and the aorta (both thoracic and abdominal) were found. As no other risk factor was apparent, we suggested that the cause of AP was atheromatous embolization of the pancreatic vasculature caused during the PTCA procedure.

## DISCUSSION

Among the various causes of acute pancreatitis, the vascular ones are the rarest. It was Panum back in 1862 who first suggested a relationship between circulatory disease and acute pancreatitis.<sup>2</sup> Other causes of pancreatic ischemia resulting in acute pancreatitis are; systematic lupus erythematosus,<sup>3</sup> polyarteritis nodosa,<sup>4</sup> hemorrhagic shock,<sup>5</sup> transcatheter arterial chemoembolization (TACE) for hepatocellular carcinoma<sup>6,7</sup> and atheromatous embolization.<sup>8</sup> Orvar and Johlin reported the largest series of acute pancreatitis after cardiac catheterization or abdominal angiographic procedures.<sup>8</sup> They studied the data of 21,680 patients who underwent angiographic procedures of any kind during a 10-year period and they discovered only 39 patients with acute pancreatitis after the procedure. They excluded 30 of them who had multiple risk factors

for developing AP and they resulted only in 9 patients out of 21,680 (0,04%) who had no other evident cause of AP than the procedure itself. They suggested that atheromatous embolization of the pancreas during the angiographic procedure was the cause of the pancreatitis, relying on the autopsy findings of 3 patients. Autopsy on all patients showed multiple cholesterol emboli in the kidneys, spleen, bowel and pancreas and severe necrosis of the gland.

Our patient had no evident risk factors for acute pancreatitis, such as cholelithiasis or alcoholism. His medical history concerning his cardiovascular system was ambiguous, with only a reference of “heart disease” for which he denied to take any kind of medication.

During his hospitalization he received medication for MI (diuretics, antihypertensives and salicylates) which have been implicated as possible causes of acute pancreatitis.<sup>3</sup> However, the initial attack of AP was only 24 hours after the PTCA procedure and the initiation of MI medication followed the diagnosis of AP. The only possible relationship between pancreatitis and medication that could be suggested would be the lethal deterioration of the clinical condition of the patient. His medical history and autopsy findings of aortic atheromatosis suggest atheromatous embolization of the pancreatic vessels during the PTCA, as the probable cause of AP, since no other risk factors were present.

In conclusion, although atheromatous embolization is an extremely rare cause of acute pancreatitis, it should be considered as one of the possible causes of acute abdominal pain of patients who undergo angiographic procedures.

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## Case report

## Idiopathic portal hypertension in a twin treated with TIPS and consequent splenectomy

I.A. Mouzas<sup>1</sup>, A.A. Hatzidakis<sup>2</sup>, Erminia Matrella<sup>1</sup>, Maria Roussomoustakaki<sup>1</sup>, E.A. Kouroumalis<sup>1</sup>

### SUMMARY

**Background:** Idiopathic portal hypertension is a disorder of unknown aetiology characterized by portal hypertension secondary to splenomegaly, without cirrhosis. There are no reports on idiopathic portal hypertension occurring in twins. Variceal haemorrhage, a life threatening manifestation of portal hypertension may be treated with transjugular intrahepatic portosystemic shunt in the acute setting. **Case presentation:** A 36-year-old woman with severe variceal haemorrhage and ascites due to idiopathic portal hypertension was admitted to the Gastroenterology Department. Her twin sister underwent a splenectomy at the age of 12 due to splenomegaly and haemolytic episodes without further complications. The patient, like her twin sister, had also a history of splenomegaly since her childhood, with haemolytic episodes and need for multiple transfusions. Splenectomy was not preferred for her. In the following years, blood group incompatibilities developed after multiple transfusions that precluded any further blood transfusions. A  $\beta$ -thalassaemia trait was also present. At admission, because of active variceal haemorrhage we performed a transjugular intrahepatic portosystemic shunt (TIPS) in an emergency setting. A decline of the portosystemic pressure gradient from 26 to 12 mmHg resulted with no further bleeding and with a subsequent reduction of the spleen size from 35 cm to 20 cm in diameter. A transjugular liver biopsy, a few months after

TIPS, revealed a mild chronic hepatitis that was attributed to hepatitis C virus infection acquired from transfusions before 1990. A splenectomy was performed and the haematological parameters improved significantly. Despite TIPS obstruction that occurred later, no further oesophageal varices developed, and there was no need for further transfusions. **Conclusions:** In this patient, idiopathic portal hypertension may have had splenomegaly possibly related to haemolytic episodes as an initial cause, whereas later increased portal vascular resistance developed. In her twin sister, who also had splenomegaly at childhood, there was no development to portal hypertension due to an early splenectomy. Emergency treatment of the portal hypertension with TIPS, followed by a later surgical splenectomy was an effective management option for a follow up period of six years.

**Key words:** idiopathic portal hypertension, transjugular intrahepatic portosystemic shunt, splenomegaly, splenectomy, thalassaemia, twins

### BACKGROUND

Idiopathic portal hypertension (IPH) is a clinical disorder of unknown cause, typically associated with splenomegaly and anemia, without cirrhosis. This entity was initially named Banti's syndrome<sup>1</sup> and later when a more precise definition was available, idiopathic portal hyper-

#### Abbreviations:

IPH=Idiopathic portal hypertension  
TIPS=Transjugular intrahepatic portosystemic shunt  
ANA=Antinuclear antibodies  
AMA=Anti-mitochondria antibodies  
SMA=Smooth muscle antibodies  
EBV=Epstein-Barr virus  
CMV=cytomegalovirus  
HCV=Hepatitis-C virus

<sup>1</sup>Department of Gastroenterology, University Hospital of Heraklion, Medical School, University of Crete, Greece,

<sup>2</sup>Department of Radiology, University Hospital of Heraklion, Medical School, University of Crete, Greece

#### Author for correspondence:

I.A. Mouzas, Department of Gastroenterology, University Hospital of Heraklion, Medical School, University of Crete, Greece.  
[mouzas@med.uoc.gr](mailto:mouzas@med.uoc.gr)