

# Acute Pancreatitis Following Resection of Juxtarenal Abdominal Aortic Aneurysm

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## Abstract

A case of acute pancreatitis following resection of a juxtarenal abdominal aortic aneurysm is reported. The patient was a 73 year old man who underwent resection of a juxtarenal abdominal aortic aneurysm. The aneurysm was repaired with a 20 mm. gelatin coated Dacron graft. Proximal control of the aneurysm was performed with supraceliac aortic cross clamping. The clamping time was 50 minutes. Postoperatively, he developed progressive abdominal distension with deterioration of renal and pulmonary function necessitating relaparotomy on the 7th postoperative day. The second operation revealed evidence of saponification and fat necrosis in the omentum. The pancreas was edematous and swollen compatible with acute pancreatitis. The aortic graft and other intraabdominal organs appeared normal. Despite intensive supportive care, the patient died 2 weeks later from multiple system organ failure. The possible causes of acute pancreatitis following aortic surgery described in the literature are 1. systemic and regional hypoperfusion, 2 atheromatous emboli to arteries supplying the pancreas and 3. direct trauma to the pancreas during the operation from retractors or surgical dissection. All of which may be the etiology of acute pancreatitis in our patient. Avoidance of such factors during aortic surgery is recommended to prevent this potentially fatal complication.

**Key word :** Acute Pancreatitis, Abdominal Aortic Aneurysm, Resection

Acute pancreatitis is occasionally observed following aortic surgery. Warshaw and O'Hara found a 5-year incidence of 2 per cent in survivors after aortic resection<sup>(1)</sup>. Significant mortality associated with this entity has been reported owing to 1. the aggressive nature of this specific type of pancreatitis, 2. the complication usually occurring in

high risk patients, and 3. the diagnostic difficulty following a major abdominal operation. Diagnosis is frequently delayed because the clinical symptoms and signs of acute pancreatitis may be overlooked in the early postoperative stage of the aortic surgery. Management is usually difficult. Sepsis, multiple organ failure, aortic graft infection, and aortic

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anastomotic leakage may be the ultimate fatal event(2-4).

We report a case of acute pancreatitis following a juxtarenal abdominal aortic aneurysm resection. The pathogenesis of this entity is reviewed and discussed.

### Case presentation

A 73 year-old male was admitted to Chulalongkorn Hospital on the 1<sup>st</sup> of October 1997 for resection of a large abdominal aortic aneurysm (AAA). He had had asymptomatic AAA for years which had markedly increased in size during the last few years. He also complained of progressive dysphagia for 2 years. He used to be a heavy smoker but stopped smoking 20 years ago. Physical examination revealed an elderly patient who could get about quite well despite some shortness of breath on exercise. The heart sounds were normal. Occasional wheezing was heard at both sides of the chest. A large AAA was detected on abdominal examination. The peripheral pulses were within normal limits.

Abdominal computed tomography revealed a 8 cm diameter juxtarenal AAA (Fig. 1). The esophagography revealed an abnormal dilatation of the esophagus with a stenotic lesion at the esophagogastric junction (Fig. 2). The esophagoscopy revealed a megaesophagus with no intraluminal lesion and normal mucosa at the distal esophagus. The scope could be passed into the stomach which also revealed normal gastric mucosa. At this stage, the diagnosis of achalasia cardia was also made.

Preoperative evaluation included pulmonary function test, electrocardiography, and exercise stress test. Pulmonary function test revealed severe obstructive lung disease. The electrocardiography and exercise stress test were within normal limits. After a careful evaluation of the operative risks, it was concluded that surgery should be performed for fear of rupture of the AAA. Concomitant resection of the AAA and esophagomyotomy were scheduled after improvement of the pulmonary function with active pulmonary physical therapy and medication.

The operation was performed under general anesthesia with a long midline incision. A 8 cm diameter AAA involving from just below the left renal artery to the aortic bifurcation was found. Proximal control of the aneurysm was performed at the aortic hiatus (supraceliac aortic clamping). The aneurysm

was replaced with a 20 mm gelatin coated knitted Dacron graft. The aortic cross-clamp time was 50 minutes. The graft was completely covered with the aneurysm sac and the retroperitoneum. Transabdominal esophagomyotomy was subsequently performed. The total operative time was 5 hours. Six units of blood transfusion were used. The hemodynamic status was stable throughout the operation. Postoperatively, the patient was admitted to the intensive care unit.

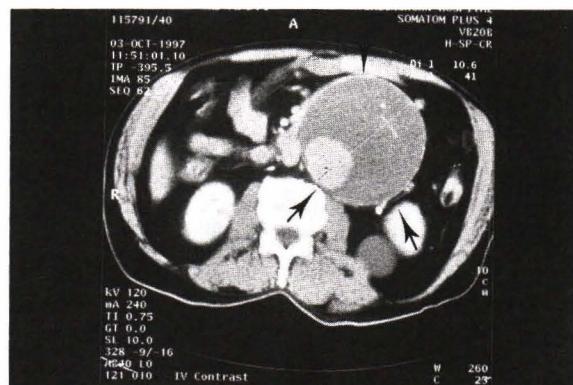


Fig. 1. Computed tomography of the abdomen showing a large abdominal aortic aneurysm (arrows).



Fig. 2. Esophagography searching for the cause of dysphagia showing an abnormally dilated esophagus with a stenotic lesion at the esophagogastric junction. Subsequent esophagogastric endoscopy revealed normal esophageal and gastric mucosa and the diagnosis of achalasia cardia was also made.

Early postoperative period was uneventful. However, on the 5th postoperative day, the patient developed progressive abdominal distension with deterioration of renal and pulmonary function. The body temperature was 38°C, the hematocrit was 38 per cent, the white blood cell count was 15,500/mm<sup>3</sup> (neutrophil 85%, lymphocyte 15%). The patient was treated by reintubation with mechanical ventilatory support, nasogastric decompression and other supportive measures. On the 7th postoperative day, the patient's condition became worse. The abdomen was markedly distended. Re-laparotomy was considered necessary to exclude a possible surgical condition. At the second operation, there was no free intraperitoneal fluid. Evidence of saponification and fat necrosis were found in the omentum. The whole pancreas was edematous and swollen. The retroperitoneum covering the aortic graft was normal. The liver, spleen, small and large bowel appeared normal. The diagnosis of acute pancreatitis was made and three penrose drains were placed around the pancreas. In spite of the obvious operative finding of pancreatitis, the postoperative serum amylase level was within normal limits.

The postoperative period was complicated by multiple organ failure, the patient died 2 weeks later despite intensive supportive therapy.

## DISCUSSION

Castleman and McNeely first reported a case of acute pancreatitis after ruptured abdominal aortic aneurysm repair in 1967. The patient had severe pancreatitis with necrosis of both the pancreatic tissue and the surrounding fat. Although the exact cause of pancreatitis was not found, the authors hypothesised that embolization of the atheromatous debris from the aorta into the pancreatic arteries lead to ischemic injury in the pancreas<sup>(5)</sup>. Most reported cases of acute pancreatitis following aortic surgery occurred in patients who had ruptured abdominal aortic aneurysms<sup>(3-6)</sup>. Ischemic injury and direct mechanical trauma to the pancreas by operative dissection or retractor are reported to be the possible causes<sup>(1,7)</sup>.

Ischemic injury to the pancreas during aortic surgery may be caused by 1. systemic hypotension, 2. supraceliac aortic clamping and 3. atheromatous emboli to the arteries supplying the pancreas. The pancreas is highly vulnerable to ischemic necrosis and may develop ischemic injury in a fashion similar to the development of acute tubular

necrosis (ATN) in the kidney. In one study<sup>(1)</sup>, autopsy findings of patients who died from oligemic shock showed a 9 per cent incidence of acute pancreatitis if there was no concomitant ATN, but a 50 per cent incidence in those with ATN. Supraceliac aortic cross-clamping has been advocated as a method of proximal control in complex abdominal aortic reconstruction<sup>(8,9)</sup>. Although supraceliac aortic cross-clamping has the advantage of rapid aortic control without extensive and difficult and sometimes dangerous dissection of the aorta associated with supra or infrarenal control, visceral organ ischemia is a major disadvantage of this procedure. Significant ischemic complications of the visceral organs when supraceliac cross-clamping was performed have been shown to be related to the cross-clamp time<sup>(10)</sup>. When the cross-clamp time was less than 30 minutes, postoperative renal insufficiency was rarely encountered<sup>(10)</sup>. The supraceliac cross-clamp time in our patient was 50 minutes which could have produced a significant ischemic damage to the pancreas leading to the development of acute pancreatitis.

Another possible cause of acute pancreatitis in our patient is embolization of the atheromatous debris to the pancreatic vascular bed during the operation resulting in edema and cellular breakdown with release of proteolytic enzymes<sup>(1)</sup>. Atheromatous emboli have been reported to be a possible cause of acute pancreatitis by several investigators<sup>(5,11-13)</sup>. In laboratory studies, necrotizing pancreatitis may be produced by embolization of microspheres into the pancreatic circulation<sup>(14)</sup>.

The diagnosis of acute pancreatitis following aortic surgery is usually difficult owing to vague clinical presentations of prolonged ileus and leucocytosis<sup>(7)</sup>. Although a mild form of acute pancreatitis (edematous pancreatitis) is usually a self-limiting disease which subsides in a few days on nasogastric suction and fluid administration, the fulminant form which is frequently associated with a hypoperfusion state may result in sepsis, multiple organ failure and death<sup>(1)</sup>. Diagnosis frequently comes as a surprise at autopsy<sup>(15,16)</sup>. For these reasons, antemortem diagnosis of acute pancreatitis depends on a high index of suspicion in high risk patients. Patients at risk for pancreatitis after abdominal aortic aneurysm repair are those who have experienced systemic hypotension, those who have the potential for atheromatous emboli during sur-

gery, those who have supraceliac cross-clamping and those who have difficult exposure and extensive dissection causing direct trauma to the pancreas. The diagnosis of pancreatitis should be suspected if a prolonged and unexplained ileus are present, especially if hyperamylasemia or hyperlipidemia is documented. Computed tomography should be performed when acute pancreatitis following aortic surgery is suspected. If a retroperitoneal fluid collection or necrotic mass is present, early and

aggressive drainage and debridement are recommended<sup>(3)</sup>.

Acute pancreatitis following aortic surgery is a serious complication with a high mortality rate. Surgeons should be aware of this entity when performing aortic surgery. Systemic hypotension, atheromatous embolization and inadvertent trauma to the pancreas by retractors or dissection should be avoided. Supraceliac aortic cross-clamping, if necessary, should be as brief as possible.

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## ตับอ่อนอักเสบเฉียบพลันภายหลังการผ่าตัดรักษาหลอดเลือดแดง aorta ในช่องท้อง เป็นพอง

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ได้รายงานผู้ป่วย 1 รายที่เกิดตับอ่อนอักเสบเฉียบพลัน (acute pancreatitis) ภายหลังการผ่าตัดรักษาหลอดเลือดแดง aorta ในช่องท้องเป็นพอง (abdominal aortic aneurysm หรือ AAA) เป็นผู้ป่วยชายอายุ 73 ปี มี AAA ขนาด 8 เซนติเมตร ได้รับการผ่าตัดรักษาโดยใส่หลอดเลือดเทียม (gelatin coated Dacron graft) ขนาด 20 มิลลิเมตร ระหว่างผ่าตัดได้ทำ proximal control ของ AAA โดย clamp aorta เหนือ origin ของ celiac artery (supraceliac aortic cross clamping) น่อจากตัว AAA อยู่ชิดกับ origin ของ renal artery (juxtarenal abdominal aortic aneurysm) เวลาที่ clamp ประมาณ 50 นาที หลังผ่าตัด 5 วันผู้ป่วยมีอาการอีดแน่นท้องโดยไม่ทราบสาเหตุแน่นอนและอาการไม่ดีขึ้น ถึงแม้จะได้รับการใส่สาย nasogastric tube ระบายน้ำและน้ำย่อยออกจากการเพาะอาหาร นอกจากนี้การทำงานของไตและปอดก็เจ็บ จนแพทย์ผู้รักษาพิจารณาว่าผู้ป่วยควรได้รับการผ่าตัดอีกครั้งหนึ่งเพื่อตรวจสอบภาวะแทรกซ้อนในช่องท้องเกิดขึ้น หรือไม่ การผ่าตัดครั้งที่ 2 พบว่า omentum มี saponification และ fat necrosis ตัวดับอ่อนบวมมากเข้าได้กับตับอ่อน อักเสบเฉียบพลัน ตัวหลอดเลือดเทียมและอวัยวะอื่น ๆ ในช่องท้องอยู่ในเกณฑ์ปกติ ผู้ป่วยเสียชีวิตอีก 2 ลับดาห์ต่อมา จาก multiple system organ failure สาเหตุของตับอ่อนอักเสบเฉียบพลันภายหลังการผ่าตัดรักษาพยาธิสภาพต่าง ๆ ของหลอดเลือดแดง aorta ที่เคยมีรายงานไว้ได้แก่ 1. การมีเลือดไปเลี้ยงตับอ่อนลดลงจากความดันโลหิตต่ำ หรือ การ clamp aorta เหนือ origin ของ celiac artery, 2. เศษของ atherosclerotic plaque มี embolization เข้าสู่แขนงต่าง ๆ ของหลอดเลือดแดงที่เลี้ยงตับอ่อนและ 3. การบาดเจ็บหรือชอกหัวต่อตับอ่อนในระหว่างการผ่าตัด ซึ่งเกือบทุกข้อ อาจเป็นสาเหตุของตับอ่อนอักเสบเฉียบพลันของผู้ป่วยในรายงานนี้ ผู้รายงานได้แนะนำให้หลีกเลี่ยงปัจจัยเลี้ยงต่าง ๆ เหล่านี้ในระหว่างการผ่าตัดรักษาพยาธิสภาพต่าง ๆ ของหลอดเลือดแดง aorta ในช่องท้องเพื่อป้องกันการเกิดตับอ่อนอักเสบเฉียบพลัน ซึ่งเป็นภาวะแทรกซ้อนที่มีอัตราตายสูง

**คำสำคัญ :** ตับอ่อนอักเสบเฉียบพลัน, หลอดเลือดแดง aorta เป็นพองในช่องท้อง, การผ่าตัด

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