Severe acute pancreatitis of gallblader origin with sequels: pancreatic necrosis, pseudocyst and splenic vein thrombosis

PANCREATITIS AGUDA GRAVE DE ORIGEN BILIAR CON SECUELAS; NECROSIS PANCREÁTICA, PSEUDOQUISTE Y TROMBOSIS DE LA VENA ESPLÉNICA

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40 YEAR OLD MALE WITH ULCERATIVE GASTRITIS, CHOLELITHIASIS AND SEVERE ACUTE PANCREATITIS WITH LONG-TERM HOSPITAL STAY, REQUIRING TRACHEAL INTUBATION AND TRACHEOTOMY. CHYLOPERITONEUM, CHOLELITHIASIS AND PANCREATIC PSEUDOCYST WITH A 90% NECROSIS WITHOUT ACTIVE BLEEDING WERE FOUND DURING SURGERY. CHOLECISTECTOMY, WASHING AND INTRACAVITARY NECROSECTOMY, STITCHING OF THE SMALL VASCULAR INTRACYSTIC STUMP, ROUX-EN-Y CYSTOJEJUNOSTOMY, DRAINING OF THE CHYLOPERITONEUM AND OF THE CAVITY WERE PERFORMED. CHYLOUS ASCITES PERSISTED AND WAS TREATED WITH OCTEOTRIDE, DIURETICS AND FAT-FREE DIET SUPPLEMENTED WITH MEDIUM-CHAIN TRIGLIYERIDES, PROTEINS AND VITAMINS. HOWEVER, SEQUELS REMAINED INCLUDING: RECURRENT ACUTE PANCREATITIS, SPLENIC VEIN THROMBOSIS AND LEFT PORTAL HYPERTENSION. AS THE PATIENT WAS NOT DIABETIC AND THE PLATELET COUNT WAS 140000 CELLS/MM^3 SPLENECTOMY OR EXPECTANT MONITORING WERE POSED AS POSSIBLE CHOICES.

THE INTEREST OF THE CASE IS ROOTED IN THE DIAGNOSIS, TREATMENT AND EVOLUTION OF THE CHYLOUS ASCITES, 90% PANCREATIC NECROSIS, PSEUDOCYST AND SPLENIC VEIN THROMBOSIS, AS WELL AS THE REPEATED ADMISSIONS.

KEYWORDS: SEVERE ACUTE PANCREATITIS, CHYLOUS ASCITES, PANCREATIC PSEUDOCYST, LEFT PORTAL HYPERTENSION, SURGERY.

PALABRAS CLAVE: PANCREATITIS AGUDA GRAVE, ASCITIS QUILOSA, PSEUDOQUISTE PANCREÁTICO, HIPERTENSIÓN PORTAL IZQUIERDA, CIRUGÍA.

Introduction

Severe Acute Pancreatitis is defined as acute pancreatitis associated with immediate complications such as organ failure: shock (systolic blood pressure < 90 mmHg), respiratory failure (PaO < 60 mmHg), renal failure (plasma creatinine > 2 mg/dlLdL after rehydration), gastrointestinal bleeding (> 500 ml/24 hours), disseminated intravascular coagulation (platelets < 100,000 cells/mm^3, fibrinogen < 1 g/L and degradation products of fibrinogen > 80 µg/mL), severe metabolic disturbances (Ca^{2+} < 7.5 mg/dL); and late complications such as necrosis, intra-abdominal abscess or pseudocyst obstruction and dilation of ducts (1). It is characterized by three or more criteria of the Ranson classification or eight or more points in the APACHE II (2).

Clinical case

40 years old male with ulcerative gastritis treated with omeprazole, is admitted to the Emergency department due to an acute abdomen. He is conscious and oriented and presents severe epigastric pain radiating to the back, persistent vomiting, muscular defense, positive Murphy sign, diet intolerance and dysthermia.

Blood analysis showed glucose 175 mg/dL, total bilirubin 1.51 mg/dL, direct bilirubin 0.86 mg/dL, glutamic-pyruvic transaminase (GPT) 871 LU/L, lactate dehydrogenase (LDH) 1300 U/L, amylase 3089 U/L, K 3.60 mE/L, 24000 leucocytes per cubic millimeter (mm^3) (83% neutrophils, lymphocytes 7%), hemoglobin (Hb) 18.5 g/dL, hematocrit 54%, Prothrombin Activity (PA) 85%, activated partial thromboplastin time (APTT) 19s, international normalized ratio (INR) 1.13.

Computed Tomography (CT) shows mild bilateral pleural effusions, enlarged pancreas, ill-defined borders, and heterogeneous parenchymal density with no signs necrosis. Moderate free-liquid quantities were found in peripancreatic, perihepatic and perisplenic locations, as well as paracolic gutters and around the pouch of Douglas. Symptoms persist despite analgesic treatment. Diagnosis: Edematous Acute Pancreatitis stage C (according to Balthazar index) (3).

Rapid evolution with signs of severity; after 48 hours, the APACHE II score was 10 points and Imrie-Glasgow score was 4 points (2). He is admitted to Intensive Care Unit, conscious and oriented, but showing psychomotor restlessness and sinus tachycardia at 110 beats per minute (bpm). The patient's condition worsens, presenting encephalopathy with delirium, severe hypoxemia, arterial hypertension (AHT) and tachycardia.

Exhaustive blood volume replacement was performed. Antibiotic prophylaxis for necrosis was set with Imipenem. He was also given enteral nutrition through nasojejunal, analgesia (third step), high flow nasal oxygen therapy with increased inspired oxygen fraction (FiO_2) and 50 bpm flow. The patient showed tachypnea with great use of accessory muscles. Abdominal distension: intra-abdominal pressure (IAP) 12 to 15 mmHg, with decreased PA coagulopathy and thrombocytopenia, progressive anemia with C-reactive protein (CRP) > 200 mg/L and neutrophilic leukocytosis. Multiple organs dysfunction appears with worsening of the renal function. Treatment only improved the internal milieu.

Endotracheal intubation and mechanical ventilation connection (VM) and alveolar recruitment measures against acute lung injury secondary to pancreatitis were done. CT showed increased necrosis and free fluid. A CT-guided puncture was scheduled, although eventually it was ultrasound-guided. The resulting fluid shows a negative culture. Fever persists, accompanied by an increase in the acute phase reactants, with hypoxemia and pulmonary condition that requires diuresis with furosemide. Coagulation is fixed, but the patient shows progressive anemia.

Blood cultures were performed. The central line was replaced and bronchial aspirates were obtained. The patient had a Candida Score of 3, which prompted us to start treatment with Fluconazole due to potential candidiasis before culture results. As results were positive for Multi-Drug Resistant Pseudomonas, treatment was readjusted. The patient showed hyperglycemia, requiring insulin for adequate control, leading to suspension of enteral nutrition.

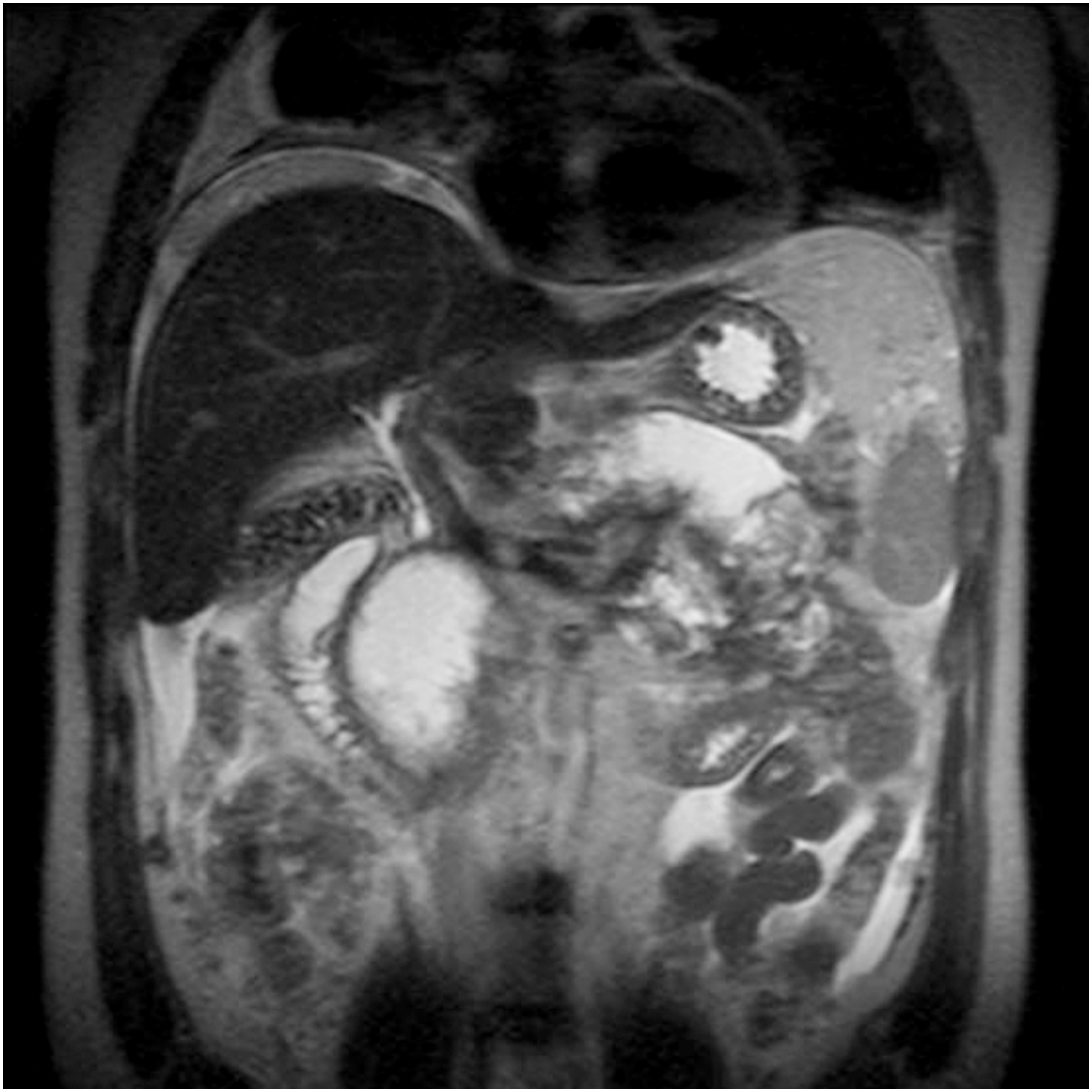
The patient was stabilized and sedoanalgesia was removed. Artificial ventilation withdrawal measures were initiated. Encephalopathy and delusions required neuroleptics. Respiratory muscle weakness required tracheotomy. The patient showed great improvement and tolerates withdrawal of respirator. The encephalopathy decreased, IAP improved, nasogastric catheter was well tolerated. Fever was controlled allowing to start treatment with Lantus Insulin in order to control glycemia. Acute phase reactants decreased in internal milieu. Two packs of erythrocytes were administered due to Hb 8 g/dL with hypoxemia and respiratory muscle fatigability. The bilateral pleural effusion was controlled. The respirator was withdrawn, and the patient tolerated this well, without exhausting speech valve. Glottic-subglottic incompetence test was negative, allowing to start a protective oral diet for the gallbladder and diabetes. Insulin doses were adjusted to meals. Decanulation was well tolerated. Management of secretions needed O_2 in nasal cannulas.

The patient was discharged to the gastroenterology department. In CT we can appreciate discrete pericardial thickening, distended stomach, pancreas only distinguishable in the tail area and pancreatic cell occupied by collections, moderate ascites especially in lower peritoneal space. Diagnosis: increased ascites without significant changes from the pancreatic area. In the next CT, fluid collection of 11x6 cm in pancreatic bed. Necrosis extended to peripancreatic and mesenteric fat with increased intra-abdominal free liquid. Diagnosis: Edematous Acute Pancreatitis degree E with 90% pancreatic necrosis (Balthazar) (3).

In magnetic resonance imaging (MRI), cholelithiasis (Figure 1) and a complex collection of 16x6.8x8.4 cm in pancreatic bed with necrotic pancreas inside were found. Left kidney size was decreased and it showed small bilateral renal cysts. Diagnosis: decrease in ascites and peripancreatic collection. Blood analysis showed gamma glutamyl transpeptidase (GGT) 78 U/L, total amylase 239 U/L, 1566 mm^3 leukocytes, erythrocytes 4,380,000 cells/mm^3, Hb 12 g/dL and hematocrit 38%.

New admission to gastroenterology scheduled in two months for endoscopic ultrasonography with pancreatic pseudocyst drainage due to abdominal discomfort. Analysis showed PT 71.4%. In endoscopy, heterogeneous content, dense, with a greater diameter of a 10 cm minimum and ill-defined limits in pancreatic cell, besides the pancreatic head area. Dense content observed with abundant detritus, not being indicated make drainage.

Readmitted after two months for abdominal CT. Radiodiagnostic objectifies a bleeding pseudocyst (4), recommending hospitalization. The patient refers being tired, more epigastric abdominal distension with pain during palpation. A rounded mass of more than 10 cm can be felt. The patient also suffers from heavy digestions and weight loss of 2 kg. He goes to the Health Center twice due to intense epigastric pain with mild anemia after blood test. Self-limited biliary colic is also shown. The patient seems to be in good condition, though he shows slight mucocutaneous pallor. Urgent blood test shows glucose 147 mg/dL and CRP 9.4 mg/L.

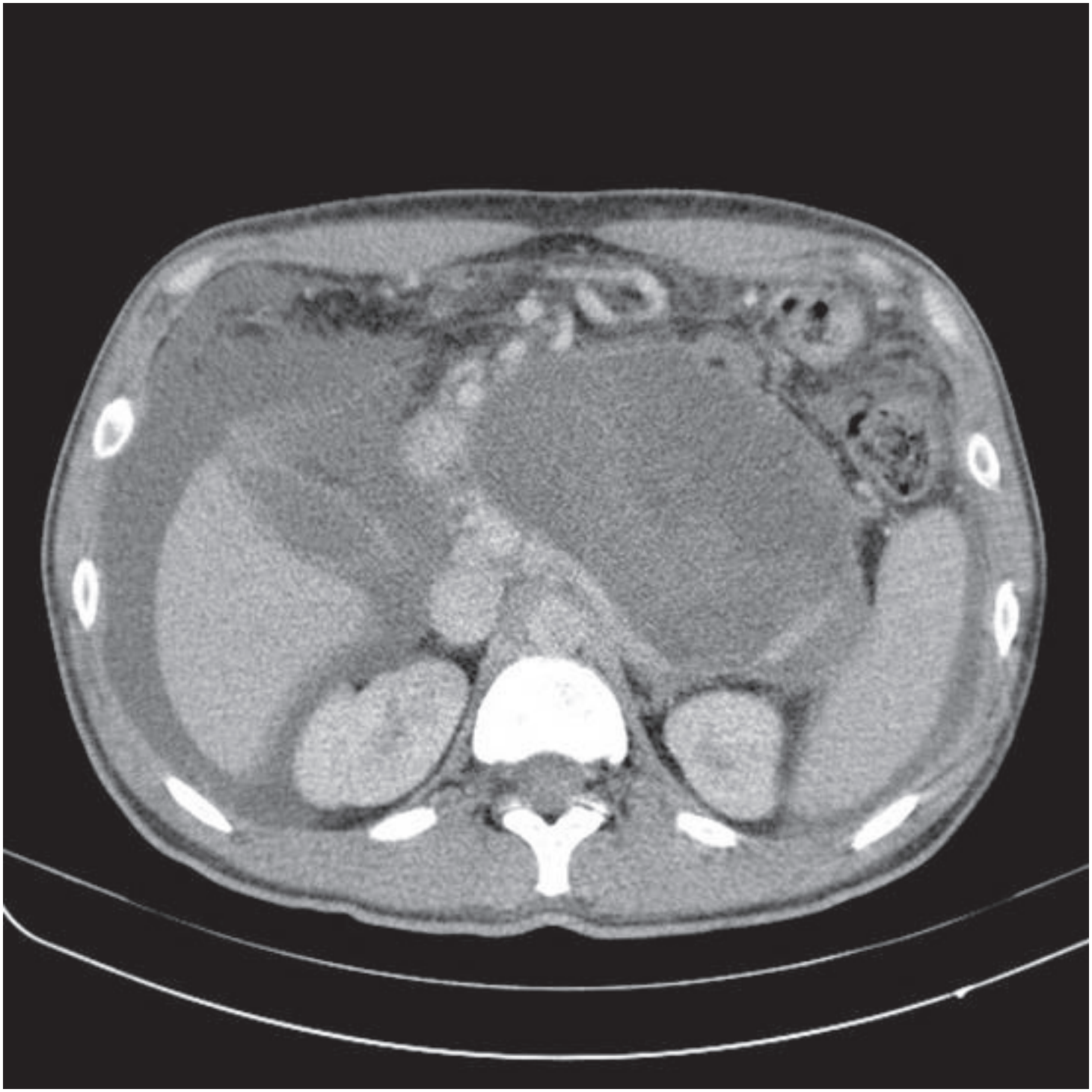


▲ **Figure 1**. *Abdominal MRI without IVC: biliary lithiasis.*

Abdominal CT with intravenous contrast (IVC) is applied, detecting a cold injury of 8 mm in segment II suggesting liver benignity, previously present and unchanged, small bilateral cortical renal cysts, fluid collection of 16x7x13 cm adjacent to pancreatic body and tail with no size changes but with high-density areas inside suggesting bleeding. Increased ascites fluid at perihepatic and perisplenic level. Both paracolic gutters and pelvis, multiple mesenteric ganglion images, some of significant size and diverticula in the sigmoid colon. Clinical diagnoses: ascites and pancreatic pseudocyst complicated with bleeding (Figure 2).

There were no signs of bleeding when abdominal arteriography was performed. The patient presented dyspeptic symptoms related to pancreatic insufficiency and compression of the stomach and intestine by the pseudocyst. Replacement therapy with Kreon and prokinetic was carried out.

After interconsultation surgery, he is submitted to intervention, finding chyloperitoneum of more than 3 liters, cholelithiasis and increased pancreatic pseudocyst a larger



▲ **Figure 2**. *Abdominal CT with IVC: ascites and pancreatic pseudocyst.*

diameter 16 cm, with necrosis inside without active bleeding. Cholecystectomy, washing and intracavitary necrosectomy, stitching of small vascular intracystic stump (with no bleeding stigmata) and Y-en-Roux cystojejunostomy were performed. Chyloperitoneum and cavity were drained. Chylous ascites persisted after intervention (verified biochemically). Dietetic and pharmacological (fat-free diet, supplements medium-chain triglycerides, proteins and vitamins), and treatment with diuretics and octreotide were prescribed.

Rating for Rehabilitation Service: myopathy due to prolonged bed rest with good evolution.

The patient attends his revision in the Surgery department, showing good wound healing, weight gain, fatigue and excessive sweating. He is under treatment with prescribed anabolic steroids. In CT, lymphadenopathy and splenic vein thrombosis likely in distal region without any consequences due to collateral irrigation. Common postsurgical changes were also seen. The pseudocyst had been solved properly.

One year after surgery, he was admitted in gastroenterology. Blood test showed amylase 119 U/L, leukocytes 12700 mm^3 (87.4% and 7.3% Lymphocytes Polymorphonuclear), Hb 17.3 g/dL. Abdominopelvic CT with IVC in venous phase shows low pancreatic remnant with increasing surrounding fat density, numerous mesenteric lymph nodes of up to 12 mm in the longer axis and a small amount of peripancreatic free fluid up to the left paracolic gutter, suggesting inflammatory process. A cold 4 mm injury in segment VIII and of 5 mm in segment VI, suggestive of benignity. Multiple collateral splenorenal, peripancreatic and perigastric vessels with splenomegaly. In upper endoscopy in the esophagus to 6.8 time fully two varicose cords in lower third. The pyloric antrum showed several injuries of acute appearance. The duodenum was erythematosus. In Cholangio-MRI demonstrated a left portal hypertension: collateral circulation through short gastric vessels (Figure 3).

▲ **Figure 3**. *Cholangio-MRI. Sinistral portal hypertension: collateral circulation through short gastric vessels.*

The patient was sent to the Endrocrinology department because of a decrease in strenght, weight and nutritional parameters. He showed an alternating constipation and diarrhea with asthenia. Kreon treatment, Aerored, Omeprazole and Lexatin.

The patient came back to the consultation room two months later, presenting postprandial discomfort. In CT, collateral circulation in the pancreatic pericell. Blood test showed basal glycemia and glycosylated hemoglobin were normal, as well as pancreatic exocrine function test. A careful diet, exercise, general muscle rehabilitation, lyophilized pancreas supplements were prescribed to ensure performance digestion.

Discussion

The patients showed sequels: recurrent acute pancreatitis, splenic vein thrombosis (4) and left portal hypertension. Splenectomy is possible, considering surgical risks, immunological problems and the age of the patient. Another choice is conservative treatment, since there are no difficult-to-treat varicose cords in the stomach. Esophageal varices are controlled with beta-blockers to prevent rupture and hematemesis (5), platelet count was 140000 mm^3 and the patient was not diabetic.

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