dium, activated charcoal, water and electrolyte replacement, pyridoxine, vitamin K, traxenamic acid, and fresh plasma. The reference liver transplant unit was contact because of suspected poisoning by *Amanita phalloides*. The cytolysis pattern and coagulation changes started to improve on the third day of stay at the ICU, and the patient was discharged to the gastroenterology ward. On the fourth day of stay at the ward (7 days since mushroom intake), creatinine levels of 4.2 mg/dL (as compared to a previous value of 1.5 mg/dL) were reported to the nephrology department. Urine: Na 95 mmol/L, K 49.08 mmol/L, urea 14.94 g/L, creatinine 100 mg/dL, protein 0.5 g/L, no RBCs. Normal complete blood count without eosinophilia, and normal C3 and C4. A further evaluation ruled out a prerenal cause, nephrotoxic agents, and an obstructive cause (by ultrasonography). Since a relationship with mushroom intake was suspected, mushrooms were analysed by an expert mycologist, who identified several species, including *Amanita phalloides* and *Cortinarius orellanus*. Support measures were started and an adequate water balance was ensured. Patient remained asymptomatic with a preserved urine output and maximum creatinine levels of 7.1 mg/dL with metabolic acidosis. Liver enzymes and coagulation were normal. Renal replacement therapy was not required at any time, and kidney function gradually improved until basal creatinine levels of 2 mg/dL were achieved. These levels have been maintained to date.

Mushroom poisoning is classified into two large groups based on whether the time elapsed from intake to symptom occurrence is shorter or longer than 6 hours. Poisonings caused by the *Amanita* and *Cortinarius* genera belong to the latter group (2-21 days). The potential occurrence of mixed syndromes due to the concomitant intake of several species, as occurred in our case, should also be taken into account.

Species from the genus *Cortinarius* have two types of toxins, cortinarins and orellanines. Orellanines show a high renal tropism, inhibiting protein synthesis in tubular cells. Orellanine degradation produces oxygen free radicals and glutathione depletion. Orellanines remain in renal tissue for up to 6 months after intake.

Renal failure occurs in 30%-75% of all poisonings depending on individual sensitivity and the amount ingested. End-stage chronic renal failure occurs in approximately one third, temporal haemodialysis is required in another third in which total or partial recovery of kidney function is subsequently achieved, and the remaining third experience no renal damage.

Non-specific gastrointestinal symptoms initially occur. These are associated to urinary frequency, that is occasionally followed by an oliguric phase with onset of uremic symptoms. Hepatic damage is rare, and some cases of transient cytolysis have only been reported.

Renal biopsy mainly shows interstitial nephritis with tubular necrosis and infiltration by lymphocytes, plasma cells, and PMNs with no glomerular involvement.

There is no specific antidote. Treatment should be supportive and symptomatic. Haemodialysis and plasmapheresis are not effective for toxin removal because of the long symptom-free period involved in late diagnosis. However, good results have been reported in some cases when performed within 5 days of poisoning. Use of corticoids and N-acetylcysteine for its antioxidant and glutathione-donating effect has been reported, but their efficacy is controversial.

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Acute pancreatitis and polycystic kidney disease

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To the editor: Adults with polycystic liver and kidney disease have cysts in the kidneys and, in many cases, asymptomatic cysts in the liver, pancreas, ovaries, and spermatic duct.[1,2] A patient with polycystic kidney disease and pancreatic cysts who experienced acute pancreatitis is reported.

The patient was a 47-year old male without no toxic habits. He had been on regular haemodialysis since September 2006 due to adult polycystic liver and kidney disease, and had underwent nephrectomy because of multiple complications derived from his renal cysts (infections, ruptures…).

The patient reported nausea, vomiting, severe abdominal pain, and loose stools.

The most common extrarenal complications in polycystic kidney disease include cerebral aneurysms, hepatic cysts, cardiac valve disease, colonic diverticulosis, and abdominal and inguinal hernias.[3]

Physical examination revealed diffuse abdominal pain, liver increased of

size, and peristalsis with no signs of peritoneal irritation.

Laboratory tests showed the presence of high amylase, lipase, and CRP levels and triglyceride levels of 218 mg/dL, with normal bilirubin, transaminase, LDH, and alkaline phosphatase values. Electrocardiogram was normal. No changes were seen in chest and abdominal X-rays.

Antibiotic coverage and fluid therapy were started, and absolute diet was maintained.

A picture of severe abdominal pain in epigastrium and the periumbilical region, often irradiating to the back, nausea, and high serum amylase or lipase levels usually confirms diagnosis of pancreatitis. Fever and ST decreases in the electrocardiogram are not uncommon.

While the main causes of pancreatitis are stones, alcohol consumption, high triglyceride levels, drugs, etc., it should also be considered in the differential diagnosis of abdominal pain in patients with polycystic kidney disease.[4]

Ultrasonography revealed a liver consistent with liver steatosis or chronic liver disease, with multiple cystic lesions. No stones were seen in the gallbladder. The bile tract was not dilated. Several cysts up to 2.6 cm in diameter were seen in the pancreas. Bilateral nephrectomy was performed.

A CT scan of the abdomen identified a cystic mass with a multilobulated contour in the pancreatic neck region, approximately 4.7 x 3.7 cm in size, that dilated the pancreatic duct at pancreas body and tail level (fig. 1).

An echoendoscopy confirmed the presence of multiple thin-walled, anechoic cysts of various sizes with no solid contents in the pancreas head and isthmus causing a 5-mm dilation in the Wirsung's duct.

No fever or leukocytosis was found at any time. The maximum amylase, lipase, and CRP levels achieved were 628 U/L, 8806 U/L, and 70 mg/L respectively. These concentrations subsequently decreased gradually during the course, abdominal pain improved, and oral diet could be restarted at 4 days with good tolerance.

The final diagnosis was acute pancreatitis, probably obstructive in nature.

As this was the first episode of pancreatitis, and given the clinical and laboratory improvement, a continued watching attitude was decided, but if the patient should experience a new episode in the future[4] or evidence of chronic pancreatitis occurred,[5] more aggressive measures already used at hepatic level,[6] such as cyst aspiration and sclerosis, surgical or laparoscopic treatment, transplant, etc. would be considered.

We think this is an interesting case, because pancreatic extrarenal cysts are usually asymptomatic.

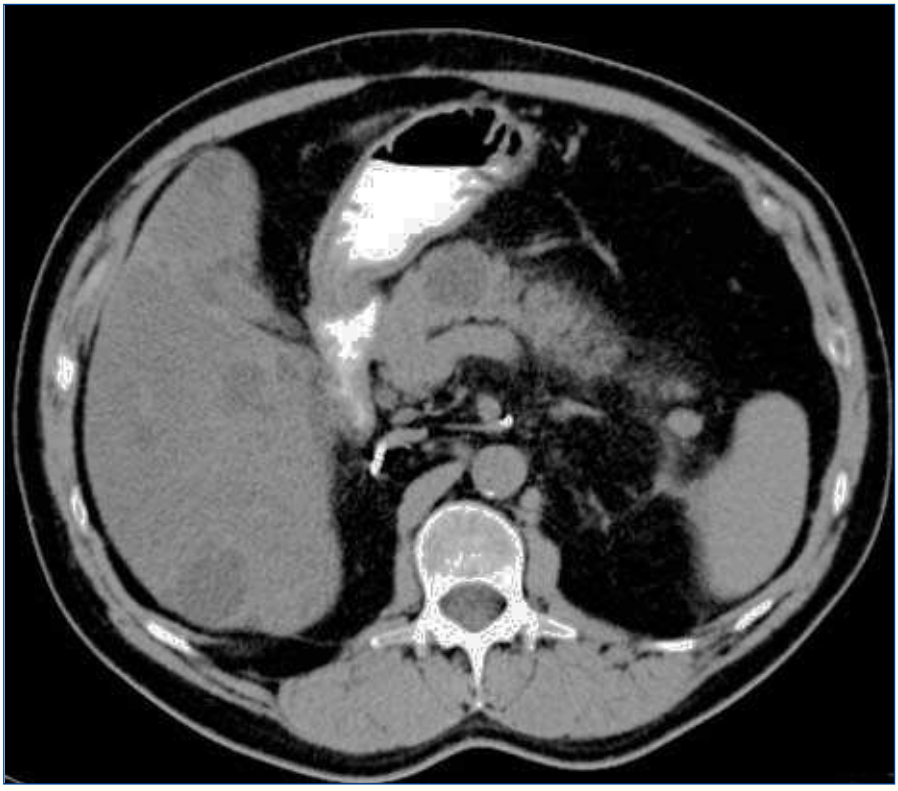

Figure 1.

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Chylotorax: an uncommon cause of pleural effusion in patients on haemodialysis

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To the editor: Chylothorax is an accumulation of lymph (containing a great amount of lymphocytes, triglyce-