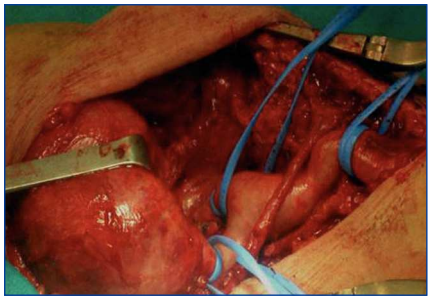

Figure 2. Surgical treatment.
Exposure of the radial aneurysm on the forearm.

struction[4] in order to guarantee adequate perfusion of the limb after the aneurysm has been excluded from circulation. Another option is endovascular treatment.[7] In our case, we opted for resection and ligation of the radial artery, since it was chronically obstructed and the perfusion of the hand was guaranteed by the ulnar and interosseous arteries.

Venous aneurysms do not require treatment unless they are associated with severe stenosis, necrosis, or skin disorders and there is a risk of rupture of the aneurysm. Severe stenosis can be treated with angioplasty. If necrosis or the risk of aneurysmal rupture appears, a surgical review is necessary.[2]

Pseudoaneurysms are ruptures contained by the soft tissue that occur primarily in puncture sites. Pseudoaneurysms of PTFE (polytetrafluoroethylene) prostheses can also be treated using percutaneous or surgical approaches.[1] In the absence of infection, a local repair can ensue by suturing the graft defect or by graft interposition.[1,8] To conclude, we would like to point out that aneurysmal dilations are complications that can jeopardise both the viability of the vascular access and the life of the patient. As such, it is essential to make a correct differential diagnosis between aneurysms (arterial and venous) and pseudoaneurysms for proper treatment planning, since the appropriate treatment varies with each case.

Conflicts of interest
The authors affirm that they have no conflicts of interest related to the content of this article.

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Nephrogenic ascites: a thing of the past?

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To the Editor,
Nephrogenic ascites is a refractory type of ascites that affects patients with chronic kidney disease on haemodialysis.[1] Although the pathogenesis of this condition is not completely clear, it appears that these patients with hypoalbuminaemia could have altered permeability of the peritoneal membrane and deficient lymph drainage.[2] The diagnosis is made by exclusion,[3] after ruling out other causes such as infection, liver disease, and heart failure. The best available option for treatment is daily haemodialysis treatment, the best alternatives for which are peritoneal dialysis and kidney transplantation.[4] There have been documented cases of complete remission of ascites following kidney transplantation.[5] Without treatment, the prognosis for nephrogenic ascites is very poor.[4]

Here we present the case of a 66 year-old patient with no toxic habits and a history of arterial hypertension, atrial fibrillation, stroke in the left middle cerebral artery with residual right hemiparesis, aphasia, and dysarthria along with acute myocardial infarction. The patient started haemodialysis treatment in January 2005 due to renal failure secondary to post-streptococcal glomerulonephritis. The patient sought treatment in November 2010 with a progressive increase of the abdominal perimeter, with a physical examination indicative of ascites. We performed an

Table 1. Characteristics of the ascites fluid

Glucose	103mg/dl
Triglycerides	42mg/dl
Total protein	4.1g/dl
Albumin	3.4g/dl
LDH	121IU/l
Amylase	43IU/l
Adenosine deaminase	23.6IU/l
Cell count	
- Leukocytes	100/mm^3
PMN	2%
MN	98%
- Blood cells	Abundant
Culture	Sterile
Histology	Cytology negative for tumour cells

LDH: lactate dehydrogenase;
MN: mononuclear;
PMN: polymorphonuclear neutrophils.

Table 2. Blood test results

Leukocytes	2.68x10^3/ml
- Neutrophils	65.0
- Lymphocytes	20.3
- Monocytes	7.9
- Eosinophils	3.7
- Basophils	0.8
Blood cells	3.56x10^6/ml
Haemoglobin	11.3g/dl
Haematocrit	37.0%
- MCV	103.9fl
- MCH	31.8pg
- MCHC	30.6g/dl
- RDW	14.7%
Platelets	100x10^3/ml
Iron	38g/dl
Transferrin	169mg/dl
Iron transfer capacity	215g/dl
Transferrin saturation index	18%
Ferritin	589ng/ml
Sodium	135mmol/l
Potassium	4.9mmol/l
Chlorine	99mmol/l
Glucose	75mg/dl
Total cholesterol	98mg/dl
Total protein	5.2mg/d
Albumin	3.9mg/dl
Total calcium	10.6mg/dl
Phosphate	4.3mg/dl
LDH	163IU/l
GOT	13IU/l
GPT	10IU/l
GGT	35IU/l
Alkaline phosphatase	90IU/l
Cholinesterase	4726IU/l
Amylase	81 IU/l
Lipase	30IU/l
Total bilirubin	0.85mg/dl
Creatinine	4.5mg/dl
Urea	84mg/dl
Uric acid	3.6mg/dl

MCHC: mean corpuscular haemoglobin concentration; GGT: gamma-glutamyl transferase; GOT: glutamic oxaloacetic transaminase; GPT: glutamic pyruvic transaminase; MCH: mean corpuscular haemoglobin; LDH: lactate dehydrogenase; RDW: red blood cell distribution width; MCV: mean corpuscular volume.

abdominal ultrasound and observed abundant fluid in the abdominal cavity and a possible lesion of the head/body of the pancreas. Under the suspicion of tumour-based ascites, we decided to hospitalise the patient for analysis. We removed 5 litres of serosanguineous fluid by paracentesis and sent samples for testing. Given the cell counts (Table 1) and biochemical properties of the peritoneal fluid, we ruled out the possibility of infection; cultures also resulted sterile. A histological analysis ruled out the possibility of malignant tumour cells. We also confirmed negative serology tests for hepatitis C and B and HIV. Blood analyses (Table 2) did not reveal any significant abnormalities. We performed an abdominal axial computed tomography and magnetic resonance cholangiography, in which we observed a slightly over-sized liver, and the head of the pancreas was not visible. We asked the gastrointestinal department to perform an endoscopy in order to confirm the existence of a lesion on the pancreas as well as to look for evidence of possible oesophageal varices that would indicate portal hypertension from cirrhosis, in light of the liver disease demonstrated by the imaging tests. However, both conditions were ruled out and the patient was discharged.

Over a 6-month period, we administered 4 paracentesis sessions and sent samples for microbiological, histological, and laboratory analyses, with no changes from the aforementioned results. Considering the possibility of a cardiological origin for the ascites, we performed an echocardiogram, observing severe biventricular dysfunction with an ejection fraction of 22% and degenerative mitral and aortic disease with no haemodynamic repercussions. Despite a pathological echocardiogram, the patient never showed signs of heart failure, with no oedema or dyspnoea. Since a serum albumin-ascites gradient

greater than 1.1 g/dL is indicative of portal hypertension with a 97% accuracy, we performed 2 tests, which resulted in values <1.1% and ruled out both liver disease and heart failure. Even so, we continued screening for a liver disease, ruling out viral, alcoholic, and other possible causes of an autoimmune liver disease. We also ruled out infections and peritoneal carcinomatosis.

Given the findings from numerous tests, the diagnosis appears to be compatible with nephrogenic ascites. Given the patient's situation and inability for self-care, peritoneal dialysis is not an option. Kidney transplant is not an option either due to the associated comorbidities and the patient's important bilateral iliac atherosclerosis. As recommended by the gastrointestinal department, evacuation paracentesis continues to be administered upon demand. We intensified the dialysis treatment and added intra-dialysis parenteral nutrition, with progressive improvements in the patient's nutritional parameters and a complete disappearance of the ascites. Currently the patient is asymptomatic.

Conflicts of interest

The authors affirm that they have no conflicts of interest related to the content of this article.

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Postpartum hemolytic uremic syndrome with multiple organ involvement in a severe case

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To the Editor,

Postpartum hemolytic uremic syndrome (PHUS), first described in 1968, is defined as a thrombotic microangiopathy (TMA) typically following a normal delivery after a symptom-free interval (mean 26.6±35 days).[1] It usually occurs in primigravida with the mean age of 27.0±6 years and preeclampsia is historically associated with the disease.[1,2] The involvement of extrarenal vascular beds in PHUS has been less reported. Here we report for the first time a severe case of PHUS complicated by pancreatic necrosis, bilateral visual loss due to central retinal artery occlusion (CRAO) and disseminated intravascular coagulation (DIC).

A 20-year-old primigravid was admitted for edema and headache when she was 34 weeks pregnant. On presentation her blood pressure (BP) was 180/115mmHg and moderate edema on face was noted. Initial investigations showed 3+ proteinuria and normal serum creatinine (Scr) concentration. The diagnosis of preeclampsia was established and a cesarean section was performed in the 35th week of gestation.

Nine days later, the patient complained of oliguria, nausea with BP of 175/105 mmHg. Laboratory tests revealed hemolytic anemia, with hemoglobin of 81g/L, serum haptoglobin <0.2 g/L, and schistocytes shown in peripheral blood smear. Platelets (Plt) were markedly reduced at $41x10^9$/L and an acute rise of Scr to 463.2 µmol/L showed acute renal failure. The immunologic studies revealed negative anti nuclear antibody and Coomb's tests. Under suspicion of PHUS, antihypertensives, aspirin and furosemide were commenced on the 1st day of presentation and renal biopsy was performed on day 2.

The patient complained of left-upper abdominal pain after renal biopsy and developed a sudden bilateral painless visual loss. The subcutaneous bleeding over her upper arms was noted and she rapidly developed anuria, dyspnea, confusion, hypotension with BP of 70/50 mmHg. The ultrasound scan excluded the existence of perinephric / subcapsular hematoma caused by renal biopsy. The fundus exam revealed bilateral CRAO. Laboratory tests on day 3 showed elevated serum amylase, lipase and Scr up to 625 µmol/L, Plt down to $12.2x10^9$/L. The level of fibrinogen decreased to 3.82 µmol/L with delaying activated partial thromboplastin time and positive D-dimer. Computed tomography scan confirmed pancreatic necrosis. Renal pathology showed thickened glomerular capillary walls with subendothelial edematous expansion that forming double contouring and renal arteriolar intimal expansion with fibrin exudation on the arteriolar wall (Figure 1). Based on these findings, the diagnosis of PHUS complicated by pancreatic necrosis, CRAO and DIC was established.

She was treated with pulse methylprednisolone 500 mg/d and intravenous immunoglobulin (IVIG) 20 g/d for 3 days. Meanwhile, plasma exchange (PE) with fresh frozen plasma (FFP) infusion and CRRT were initiated. Anticoagulant therapy for DIC and CRAO were also carried out. On day 15, she was improved significantly and the uri-