phamide is not associated with pancreatitis, but iphosphamide, another nitrogen mustard, similar to cyclophosphamide is described to produce pancreatitis.[7]

ANCA vasculitis can affect the digestive system. Our patient's cholelithiasis was not complicated; biliary colic could cause analytical alterations similar to those that developed, but in the magnetic resonance cholangiography there were no signs of choledocholithiasis. In this case, there was a clear temporary relationship with cyclophosphamide, which supports the drug's role. According to Naranjo et al's[8] scale, hepatotoxicity and cyclophosphamide are likely to be related in this case.

This case shows that hepatic and pancreatic functions must be monitored during treatment.

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Syndrome of inappropriate antidiuretic hormone hypersecretion associated with Guillain-Barré syndrome

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To the Editor,

Hydroelectrolytic disorders are frequently associated with severe neurological problems, being involved in their pathogenesis or being a consequence of it. Hyponatraemia is the most prevalent hydroelectrolytic disorder in clinical practice and is triggered by several causes. One of them is the syndrome of inappropriate antidiuretic hormone hypersecretion (SIADH) which can also be triggered by several types of neurological diseases.

We present the case of a 63-year-old man who told us that he had fever and diarrhoea, which stopped spontaneously, for two weeks. Later, he started with paraesthesia in his hands and feet, alterations in mobility and instability when walking. He therefore came to the emergency department where his gait instability became worse and stopped him from being able to walk at all. He also presented with paresis in his four limbs and facial muscles. He is hypertensive but is not treated or monitored and drinks 20g of alcohol daily. The neurological examination showed: facial diplegia with hypophonia, absence of patellar and Achilles reflex and decreased biceps and triceps reflex with no loss of strength, absence of the vibration sensitivity and bilateral reduction in tactile and pain sensitivity with ataxic gait.

Complementary tests showed: 10 100 leukocytes with normal formula; haemoglobin: 16g/dl; creatinine: 0.6mg/dl; uric acid: 2.8mg/dl; sodium: 120mmol/l; potassium: 4.3mmol/l; chlorine: 87mmol/l; bicarbonate: 16mEq/l; total cholesterol: 232mg/dl, glucose: 110 mg/dl; and serum osmolarity: 259mOsm/kg. Sodium urine test: 144mmol/l and osmolarity in urine: 719mOsm/kg. Urinary sediment was within normal levels. The cranial tomography did not show any alterations. The cerebrospinal fluid test showed red blood cells: 1/μl; leukocytes: 1/μl; proteins: 141mg/dl; and glucose: 78mg/dl. Lastly, the electromyogram showed a diffuse peripheral and distal demyelinating neuropathy, mainly involving sensitivity. The clinical symptoms and results from the analyses and electromyogram suggested that the patient had Guillain-Barré syndrome (GBS) with SIADH.

Hyponatraemia is often associated with SIADH or salt wasting syndrome in patients with central nervous system disorders. These two conditions are the most important causes of non-iatrogenic hyponatraemia.[1] GBS is a neurological disease in which the immunological system attacks the pe-

ripheral nervous system causing focal and segmental demyelinating foci that provoke an increasing paralysis, which can consequently cause respiratory failure and death. The relationship between GBS and SIADH is not very common and is described through clinical cases. The cause of GBS-related SIADH is not known, but seems to be due to independent vasopressin mechanisms, such as a long-lasting hypo-osmolarity or antidiuretic substances. It could also be caused by the renal tubule becoming more sensitive to vasopressin's action.[2] A recent study has shown a worse GBS prognosis in those patients that develop SIADH.[3] It was already known that the peak of hyponatraemia often corresponds to respiratory failure and the need for mechanical ventilation, given that hyponatraemia favours depression of the respiratory centre.[4] This hyponatraemia must be differentiated from pseudohyponatraemia produced by polyclonal immunoglobulin treatment at high doses, which is used for GBS.

These two processes can be differentiated from one another because there is a difference between the osmolarity calculated by an osmometre and using mathematical formulae in pseudohyponatraemia. In GBS, hydroelectrolytic disorders, especially hyponatraemia, are processes that mark the prognosis and severity of the disease and must be identified early.

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Giant pseudoaneurysm of an autologous arteriovenous fistula in the upper arm: surgical repair

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To the Editor,

Advances in treating end-stage chronic kidney disease have made vascular access one of the most common interventions for vascular surgeons. As with any other surgical procedure, it is not exempt from complications, and patients may present with thrombosis, haemorrhage, infection, steal syndrome, venous hypertension or long-term formation of pseudoaneurysms. The development of this last condition does not only mean that the fistula has a shorter useful life, it also involves greater risk of graft thrombosis, infection, difficulty in access or even rupture.

We present the case of a giant pseudoaneurysm of an upper-arm autologous arteriovenous fistula, which required surgical treatment to be resolved.

A 61-year-old man, diagnosed with chronic renal failure of unknown origin since 1975, when started on regular haemodialysis. Since then, he has received a total of three kidney transplants, having been a recipient for 20 years. Two years ago, he was included once more on a haemodialysis programme (using a central venous catheter). Other interesting aspects of his medical history were: arterial hypertension, insulin-dependent diabetes mellitus, C virus chronic liver disease and surgical repair of pseudoaneurysm developed on an arteriovenous fistula in the contralateral arm (left).

In the last three years, the patient developed a pulsatile tumour in a previous fistula in the right upper arm, compatible with a pseudoaneurysm. After it sharply increased in size during recent months (Figure 1a), a computerised angiotomography (angio-CT) was requested which showed that axillary and subclavian arteries were permeable, the humeral artery was very calcified and fistula in the flexure of the right forearm with a giant pseudoaneurysm of 63x57mm.

Following these observations, surgery was indicated to close the fistula. We used a cuff on the high upper arm to produce ischaemia and control the bleeding during the opening. We performed a longitudinal incision on the pseudoaneurysm (Figure 1) so that the original path of the humeral artery could be located (Figure 2a) and the humero-humeral bypass was performed with a 6-mm Dacron prosthetic graft. The blood flow towards the limb (Figure 2b) was fully re-established.

Vascular access complications are responsible for 15% of haemodialysis patients' hospital admissions.[1] For that reason, a multidisciplinary approach must be used to detect them early.[2] Pseudoaneurysms are relatively uncommon complications, and their incidence is even less in autologous fistulae, compared to those performed with a polytetrafluoroethylene (PTFE) graft.[3] In those cases that reach a small size, endovascular treatment (covered stent or thrombin injection) may be enough to try and prolong the vascu-