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-

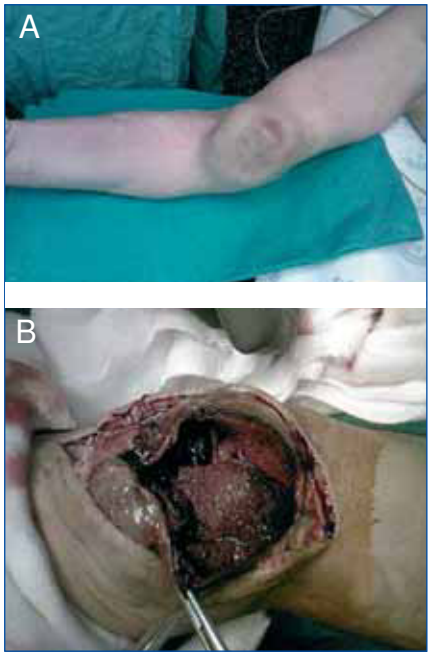

Figure 1. A) The pseudoaneurysm's large pulsatile protrusion, with onset of trophic disorders secondary to maintained local pressure. B) Thrombotic content after opening the pseudoaneurysm, with removal of the content (thrombectomy).

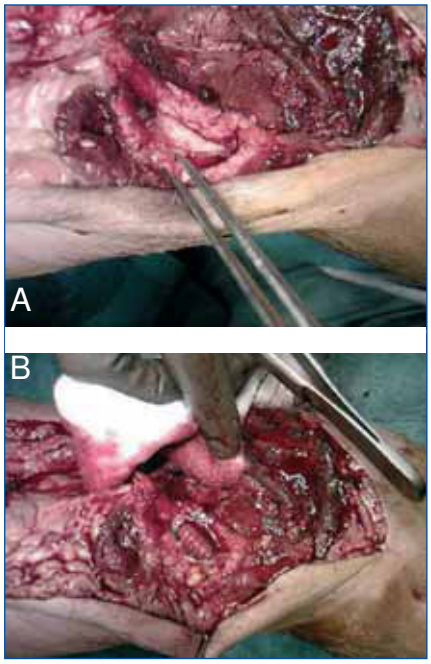

Figure 2. A) Section on which the proximal and distal mouth of the native humeral artery were identified. B) Final result of humero-humeral bypass with prosthetic graft, after performing both end-to-end anastomosis.

lar access's life.[4] In contrast, for cases such as that described, open surgery is the only effective treatment for avoiding events that require urgent action.

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Rhabdomyolysis secondary to hyponatraemia

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To the Editor,

Rhabdomyolysis is a syndrome with multiple causes and whose aetiology is unknown in many cases. Hyponatraemia is a rare cause of rhabdomyolysis and can go unnoticed if not suspected. In this instance, the rhabdomyolysis was related to hyponatraemia secondary to symptoms of nausea and vomiting.

Rhabdomyolysis is characterised by skeletal muscle injury, alterations in cell membrane integrity and release of intracellular products into the blood. This potentially lethal condition becomes worse with acute renal failure (ARF) in 4%-30% of patients, and causes 7%-10% of all ARF cases.[1,2]

There are multiple causes for rhabdomyolysis, which can be grouped as: *1)* direct muscular trauma; *2)* excessive muscular activity; *3)* hereditary enzymatic defects (McArdle's disease); *4)* less evident causes, such as drugs (antipsychotic, antidepressive and lipid-lowering agents, cyclosporin, etc.), toxic agents, infections, autoimmune , endocrines (hypothyroidism, hyperaldosteronism, ketoacidosis) and electrolytic disorders.[2,3] The pathophysiology for many of these processes converge into one final path that compromises the adenosine triphosphate (ATP) synthesis and the functioning of Na^+/K^+ and Na^+/Ca^{++} pumps. It results in an increase of permeability to Na^+ and an increase in intracellular Ca^{++}, which starts an enzymatic activation and cell death process.[3-5]

Rhabdomyolysis is related to hyponatraemia/hypernatraemia, severe hypopotassaemia and hypophosphataemia which affect the membrane homeostasis and cell integrity. We present a clinical case of rhabdomyolysis with severe hyponatraemia.

The patient is a 74-year-old female, with hypertension and dyslipidaemia. She was undergoing treatment with omeprazole, fluvastatin and tramadol. She presented with liquid diarrhoea and intractable vomiting, which had lasted a week. She was treated as an outpatient with oral fluid therapy. Her level of consciousness was reduced and she presented with a tonic-clonic seizure and sphincter relaxation. The emergency