pulse were eventually restored. Although limb infusion had been adequate in previous vascular surgical reviews, it poses a difficulty in wound healing.

Late-stage vascular access thromboses are of multifactorial etiology, with predisposing factors such as hypotension, haemoconcentration and hypercoagulable states.[1,2]

For treatment of vascular access thromboses, clinical practice guidelines recommend surgical thrombectomy or mechanical or pharmacomechanical thrombolysis, depending on experience, with good results.[3,4] None of the guidelines either recommend or advise against massage as an immediate treatment of thrombosis of an arteriovenous fistula. However, in practice this technique is applied with a view to performing a fistulography within subsequent hours.

There are no publications in the scientific literature that approach this subject. Only a case of embolisation following massage of a PTFE graft is described. Our case is similar, only with an autologous fistula. Both cases demonstrate one of the potential negative consequences of practising massage: distal embolisation of the extremity, for which an early diagnosis of acute ischaemia is necessary. Other possible consequences, such as pulmonary embolism, also cannot be ruled out. Unfortunately, there is no evidence to quantify the favourable results of internal fistula massage in correcting a thrombosis, meaning we are unable to establish a balance between its risks and benefits. It is possible that the benefit is greater in radiocephalic fistulae, owing to their distal location and presence of the palmar arch. Despite this, to the extent that established therapeutic options exist for the correction of a vascular access thrombosis (surgical thrombectomy and thrombolysis), we believe that the practice of massage should be advised against due to its potential complications.

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Late pleuroperitoneal leak in a peritoneal dialysis patient

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Dear Editor:
Mechanical complications in peritoneal dialysis (PD) are common, particularly the appearance of abdominal herniae dependent on a sustained increase of intra-abdominal pressure.[1] The presence of anomalous pleuroperitoneal communication results in the appearance of massive pleural effusion.[2]

Diagnosis is usually clinical, although there are various available imaging techniques for confirming this.[3] In our centre we have available the Tc-99 scintigraphy, and have used it as a simple and innocuous method for confirming clinical suspicions.

We present the case of a 52 year old patient who began a CAPD programme in April 2008 following five years in haemodialysis. No pathological history of interest, except nephroangiosclerosis as cause of renal failure.

The PD catheter was fitted using a laparoscopic technique (Techknoff swan neck, rectum) without complication. At two months the patient was following a routine of APD with no problems. The course was completed on a humid day at low volume due to abdominal discomfort.

Four months after fitting the catheter the patient suddenly complained of general discomfort and intense epigastric pain with no vomiting, depositional alterations or fever. Orthopnoea and pain in the right ribcage with a dry cough. Increase in weight of 2kg during previous days, apparently without loss of UF.

On examination, global hypoventilation of the right lung was apparent. There was no oedema and the abdomen was normal.

A chest x-ray showed a massive right pleural effusion.

A thoracentesis was performed and one litre of liquid was obtained which had properties similar to dialysis liquid, with high glucose content. Negative cultures.

With a diagnosis of pleural drainage, we proceeded to peritoneal rest.

A scintigraphy was carried out, which showed the presence of a right pleuroperitoneal leak (figure 1). The patient remained on haemodialysis for three months, then resumed APD without relapse. The scintigraphy was

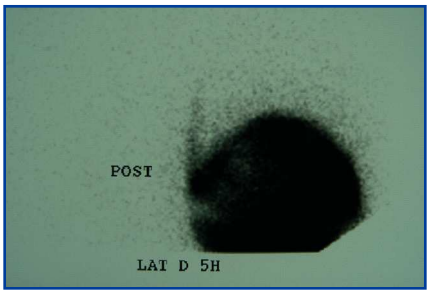

Figura 1.

repeated and showed no leak. During the following two months, the patient continued on APD until receiving a transplant.

The presence of dyspnoea compels us to rule out the most common pathology in our patients (hydrosaline retention, heart failure, etc.) but we must not forget about mechanical complications.[4]

The diagnosis will primarily be clinical, and on analysing the effusion we can test the properties of the dialysis liquid.

We stress the Tc-99 scintigraphy as a simple and safe technique for confirming a diagnosis.

It is carried out by means of a manual exchange with 2mCi Tc-99. The first reading is taken after 10 to 15 minutes, and after 3-4 hours delayed images are taken in different positions. Finally, all liquid is drained and destroyed according to nuclear medicine department protocol.

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Treatment with thymoglobulin as the cause of acute demyelinating polyneuropathy in a renal transplant patient

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Dear Editor:
Guillain-Barré syndrome (GBS), or acute inflammatory demyelinating polyneuropathy (AIDP) is a severe pathology, often fulminating, which in more than two thirds of cases presents as an antecedent infection usually caused by a virus (cytomegalovirus [CMV] or Epstein-Barr virus). We present the case of a 48 year old male, recipient of a renal transplant in January 2007; D/R CMV serology positive; immunosuppression with steroids, mycophenolate and tacrolimus; delayed graft function during initial progress but good subsequent progress (creatinine on discharge, 1.5mg/dL).

During evolution, the patient presented with an acute Banff grade IIB cellular rejection, treated with thymoglobulin, which caused an allergic reaction, and bicytopenia. Treatment was withdrawn, but renal function improved. Four days after receiving thymoglobulin the patient presented with arthromyalgias and febricula, rapidly progressing to weakness of the lower extremities and 4/5 paresis in all four extremities, and severe dysphonia and dysphagia. He remained afebrile and without respiratory compromise. Laboratory tests showed a deterioration of renal function with 2.5mg/dL creatinine. Supplementary tests: cranial CT showed no alterations. Negative cultures. CRP for CMV negative. EMG: alterations compatible with acute motor demyelinating polyneuropathy. On assessment by Neurology, the patient was diagnosed as having symptoms compatible with GBS, starting treatment with polyclonal immunoglobulin IV at a dose of 2g/kg and high doses of steroids. The symptomatology disappeared 24 hours after treatment, although a slight motor deficit persisted for several weeks. Renal function on discharge: Cr: 1.7mg/dL

Discussion

In an immunodepressed population, it is logical that the most frequently reported issue has been CMV2-4. Immunosuppression in itself constitutes an alteration of immunological equilibrium. This alteration, in keeping T suppressor lymphocytes inhibited, allows lymphocyte clones which are capable of generating an autoaggressive response to remain free. No habitual risk factor associated with the development of this entity was found in our patient. Circumstances exist in which abnormal circulating proteins have been associated with neuropathy (Waldenström's disease, multiple myeloma, POEMS syndrome). It is possible that the administration of thymoglobulin causes a condition similar to dysproteinemia, through the formation of immune complexes (serum sickness). T cells and neurons possess similar glycolipids in the membrane, with the associated chalcogens against GBS GM1. We have found only one reference[5] in the literature to a possible relationship with antilymphocyte polyclonal antibodies. In this instance, the temporal ratio and the symptoms displayed by the patient on administering the ATG, as well as an absence of other causes, cause us to think that there is a possible association between thymoglobulin treatment and subsequent AIDP, possibly related to serum sickness.

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