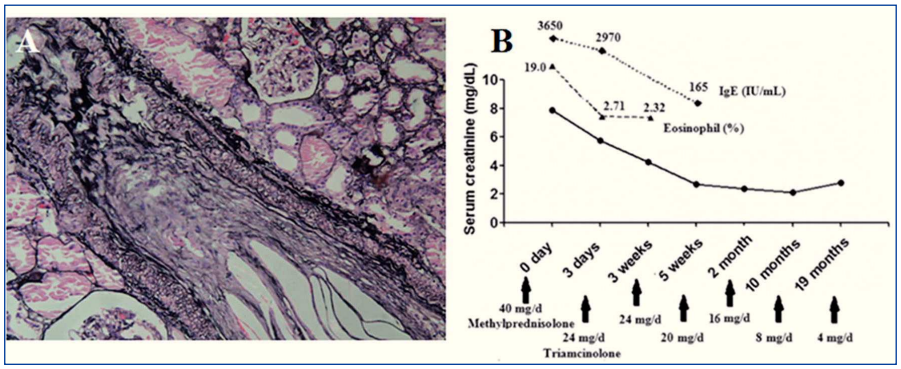

Figure 1. The kidney biopsy and clinical course of the patient.
(A) Kidney biopsy showed cholesterol clefts in the artery (PAM stain, x400). Empty clefts were seen within the obliterated lumen of small arteries in the kidney (PASM stain x 200). (B) The clinical course of the patient.

trigger an inflammatory reaction.[5] Although the role of immune system in the pathogenesis of CCE was only considered to be an inflammatory response after embolism, opposite proofs have also existed. Recently, there were two important studies which showed a significant correlation between allergic disease and atherosclerosis. In the Bruneck study of 826 middle aged and elderly Italian subjects, the risk for atherosclerosis development and progression increased significantly for 32 subjects with allergic disorders. Furthermore, serum IgE levels were significantly raised in subjects in whom atherosclerosis had developed or progressed.[2] In the ARMY study which took 141 male Austrian subjects aged 17 and 18 years, the vascular intima-media thickness (IMT) measured by ultrasonography of the 34 subjects diagnosed as having allergic disease was higher than the healthy subjects.[2, 3] Although the exact mechanism is still not fully understood, components of the allergic process such as leukotrienes and mast cells may be involved in atherogenesis.[2] Furthermore, IgE has been proved to be able to promote atherogenesis in Apoe$^{-/-}$ mice.[4]

There has not been a definite consensus of the treatment of CCE by now.[6,7] Although the use of corticosteroid is still controversial, our patient received only corticosteroid treatment without other above drugs and satisfactory results were obtained. Previous reports indicated that Anticoagulant therapy, which is generally essential in hemodialysis, was harmful to patients with CCE because it was a potent risk factor for plaque rupture and cholesterol embolism.[8] Since the present patient was cured successfully with corticosteroid, hemodialysis was avoided and the renal function kept stable to date. So we think treatment with corticosteroid should be a recommended alternative for patients with CCE, especially for patients who had obvious allergic conditions.

Conflict of interest

The authors declare that there is no conflict of interest associated with this manuscript.

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Percutaneous closure of arteriovenous fistula for haemodialysis due to venous hypertension secondary to subclavian vein occlusion

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To the Editor:

Central venous disease is a common issue in patients on haemodialysis following the creation of an arteriovenous fistula (AVF). The primary treatment of stenosis/occlusion of the central vein consists of recannalising the vein using

endovascular techniques: percutaneous trans-luminal angioplasty (PTA) with or without stent placement. However, the venous patency produced by these procedures is limited, and on occasions, the fistula must be surgically closed due to symptoms of venous hypertension. We propose a simpler and less aggressive approach for closure, through ultrasound-guided thrombin injection.

CASE REPORT 1

Our first patient was a 78-year old female on haemodialysis since 2009 with a left humerocephalic AVF. The patient developed oedema in the arm with the AVF, which produced progressively worsening pain and functional impairment. The patient was diagnosed with occlusion of the subclavian vein, and several attempts at PTA resulted in early recurrence. We decided to place a right central venous catheter and to close the AVF due to poor functioning. The AVF was annulled through ultrasound-guided injection of thrombin (Figure 1) into the cephalic vein, producing thrombosis. Ten days later, the oedema had disappeared and the artery was again patent with no symptoms.

CASE REPORT 2

Our second patient was a 74-year old female on haemodialysis since 2010 with a right humerocephalic fistula, which developed pain and increased volume of the right arm, producing functional impairment and poor functioning of the AVF (Figure 2). We performed a phlebography of the right arm, observing obstruction of the subclavian vein. We attempted to re-establish patency using endovascular techniques with no success. Given the progressively worsening symptoms, we decided to place a central jugular venous catheter and to close the AVF. We injected thrombin into the cephalic vein under ultrasound guidance approximately 4-5cm from the AVF and confirmed thrombosis. In a follow-up consultation 7 days later, the patient reported improvements in sensations of pain, although oedema remained. A Doppler ultrasound analysis revealed patency along the first few centimetres of the

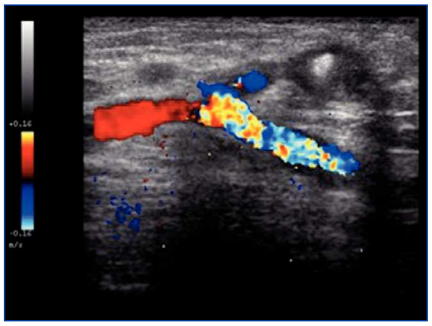

Figure 1. Humerocephalic anastomosis following the injection of thrombin. Thrombus formation in the cephalic vein.

cephalic vein, but the rest of the vein was thrombosed up to the junction with the axillary vein. We performed another proximal injection, obtaining thrombosis of the entire cephalic vein. Seven days later, the oedema had disappeared (Figure 3). A follow-up Doppler ultrasound analysis confirmed occlusion of the cephalic vein and patency of the humeral artery.

DISCUSSION

Between 17% and 40% of patients on haemodialysis will develop central vein stenosis due to multiple cannalisations.[1,2] This can produce incapacitating oedema due to venous hypertension in up to 40%-50% of cases.[2,3] Initial treatment generally involves angioplasty with or without stent placement. However, the duration of this solution is limited, producing primary patency and assisted patency rates of 20%-30% and 60%-70%, respectively, after 12 months.[3,4] In addition, more than 50% of patients will require subsequent interventions.[5] Revascularisation through open surgery involves higher rates of morbidity, and surgical interventions produce a final patency that is similar to that produced by repeated endovascular procedures.[6] Approximately 50% of patients on haemodialysis with central venous stenosis finally require ligation of the AVF, and this is particularly common in patients that produce no or only minimal initial responses to endovascular treatment.[6] Closure of the fistula is commonly performed using surgical techniques, through dissection of the area of the anastomosis and ligation of the fistula under local anaesthetic. This intervention is not without risks, since it involves operating on an arm with oedema in an area that has already undergone multiple interventions due to complications of the AVF. In our case, we closed the AVF by injecting thrombin directly into the arterialised vein under ultrasound control. This provides the advantages of being more comfortable for the patient (avoids the need for subsequent interventions), is less expensive, and produces fewer complications.

For this procedure, we applied compression to the upper arm until flow to the fistula was completely occluded, thus preventing the possibility of migration

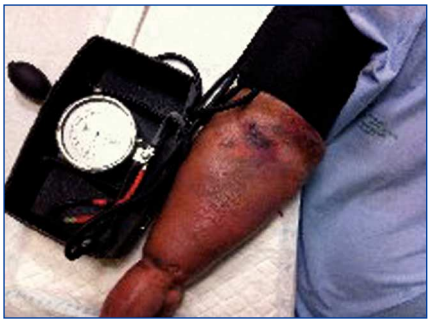

Figure 2. Internal right humerocephalic arteriovenous fistula with severe oedema and erythematous areas.

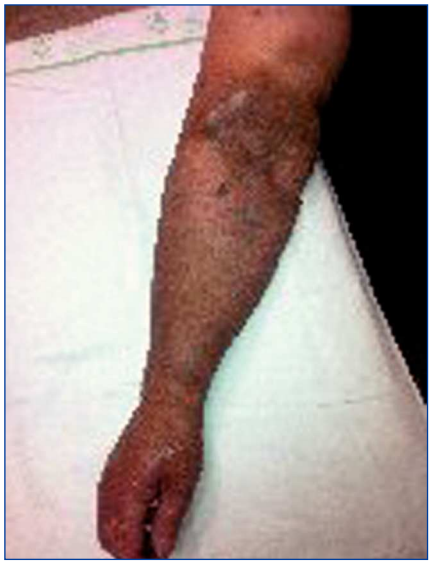

Figure 3. Clear improvement following closure of the arteriovenous fistula.

of the thrombus, although even if this were to occur, it would not produce any clinical repercussions since the subclavian vein was also occluded. We used Doppler ultrasound guidance to locate the fistula and then continued 4-5cm distally along the vein in order to avoid accidental injection of thrombin into the artery. We then placed the needle into the arterialised vein in the direction of blood flow, in order to avoid migration of the thrombin towards the artery, and injected the thrombin until thrombosis was achieved. Finally, after removing compression, we placed a compressive bandage on the arm for 48 hours.

We performed this technique on two obese patients, after several failed attempts at re-establishing patency and with symptoms that incapacitated the patients. In both cases, the AVF was closed with no complications. This is a simple, fast, inexpensive, and comfortable technique for the patient and physician, and should be taken into account as a treatment alternative when planning to close an AVF.

Conflicts of interest

The authors declare that they have no conflicts of interest related to the contents of this article.

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Pregnancy in haemodialysis patient

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To the Editor:

Although it is uncommon for females with chronic renal failure to become pregnant while on haemodialysis, there is a clear increase in the number of such cases published in the medical literature with a notable success rate, possibly due to the improvements made in dialysis techniques and obstetric care. However, we must not underestimate the risks and complications associated with pregnancy in patients on renal replacement therapy.[1-3]

Here we report on our experience with a 32-year old female patient with chronic renal failure on a periodical haemodialysis programme and who intended to be pregnant. The patient's clinical history included: chronic kidney disease secondary to a glomerulopathy that had not been biopsied, arterial hypertension, dyslipidaemia, chronic lymphocytic thyroiditis/sub-centimetre nodular goitre, perennial allergic rhinoconjunctivitis, and secondary renal hyperparathyroidism on treatment with cinacalcet.

We immediately modified the patient's treatment regimen, suspending losartan and atorvastatin and maintaining anti-hypertensive treatment with doxazosin and atenolol. Seven weeks after deciding to become pregnant, the patient developed amenorrhoea, and gestation was confirmed with a positive beta-chorionic gonadotropin blood test. We then proceeded to modify the patient's dialysis regimen, switching to 6 sessions per week of 4 hours each (24h/week) and haemodiafiltration with endogenous reinfusion. The calcium concentration in dialysate was lowered in order to avoid a positive calcium balance, and the potassium concentration was increased in order to avoid hypopotassaemia. We also modified the concentration of bicarbonate in the dialysate solution, initially lowering it to 25mEq/l in order to avoid metabolic acidosis, but then increasing it to 30mEq/l due to post-haemodialysis metabolic acidosis. For intra-dialytic anti-coagulation therapy, we administered 20mg enoxaparin (intravenous) during each session. Ultrafiltration was limited to 500ml/h in order to avoid sharp decreases in blood pressure, which heavily influences placental perfusion. As regards medical treatment, atenolol and doxazosin were replaced by methyldopa, and omeprazole was replaced by almagate; we reduced the dosage of cinacalcet, which was completely suspended after 7 weeks of gestation due to the lack of information regarding its use in pregnant women. We used calcium acetate as a phosphate binder. As regards other medications, the patient received oral iodine, vitamin C and B complex, folic acid, and carnitine 3 times per week. We did not limit the patient's dietary intake except for salt restrictions.[4,5]

During the first 22 weeks of gestation, the patient received dialysis in a peripheral dialysis centre in collaboration with her reference hospital, and went through regular controls in a high-risk pregnancy consultation. Weekly measurements were taken for haemoglobin, leukocytes and pre-haemodialysis platelets and urea, creatinine, total protein, urea nitrogen, sodium, potassium, phosphorous and pre/post-haemodialy-