Abstract

Recently, there has been an increase in the number of cases of superior vena cava (SVC) syndrome associated with chronic indwelling central venous catheters. Fibrinolytic therapy and endovascular treatment are currently achieving good results. We present a case history of a patient with SVC with a catheter used for chemotherapy, which was successfully treated with catheter-directed (intraclot) infusion thrombolytic therapy with urokinase.

Key words

Central venous catheter • Superior vena cava • Fibrinolysis

Introduction

The first time that superior vena cava (SVC) syndrome was described in 1757 by William Hunter, in a patient with syphilitic aneurysm, it was characterised by the classic trio constituted by oedema of the face, neck and upper trunk, cyanosis in face and upper extremities, and brachial thoracic collateral circulation associated with migraine, breathing difficulty and occasional loss of consciousness [1]. The aetiology of this syndrome is variable, and although in 85% of cases it appears in the environment of an advanced mediastinal neoplasia [2], lately an increase of benign causes has been observed [3], long-term central venous catheters employed in oncology causing 20–30% of thromboses in SVC [4]. The treatment of SVC syndrome is based on radiotherapy, and medical, surgical [5] and endovascular [6] treatment.

We present the case of a patient with SVC syndrome associated with a central venous catheter for chemotherapy, and treated successfully with fibrinolysis.

Clinical case

A 50 year old woman with mammary carcinoma and antecedents of lymphoedema in her left arm (2001), treated by means of lumpectomy, chemotherapy (teflon catheter for right subclavian vein) and radiotherapy was admitted to our hospital at the end of March of the 2005 with intense cyanosis and oedema of the upper thorax and eyelids. She had been treated at home by a primary physician for the first time 15 days before, due to inflammation in face and neck, and dyspnoea, with a dose of Urbason 20 mg; and for the second time, 3 days later, for significant cyanosis in face, neck and upper extremities, and dyspnoea, with Urbason 20 mg and O2.

When she did not improve, she went to hospital, and the following parameters were found: haemogram of 13 510 WBC (80.2 neutrophils, 16 lymphocytes and 2.8 monocytes) and glycaemia of 222 mg/dl. O2 saturation (resting) was 97%. Chest X-ray showed a central venous catheter. The computed tomography (CT) scan of thorax with contrast revealed thrombosis of the SVC and an absence of solid tumours and mediastinal adenopathies. A bolus of 5000 IU of solution of heparin followed by 1000 IU was administered per hour in continuous solutions. She was moved to our Central Hospital with no dyspnoea, but with cyanosis and oedema of the face, neck and upper trunk.

The cavography (Fig. 1) demonstrated an occlusion of the SVC immediately below the confluence of the innominate veins, which were permeable, with a thrombus encasing the distal portion of the catheter. In the Intensive Care Unit local fibrinolysis began with a bolus of 4400 U/kg of urokinase for 15 min, continuing with continuous infusion of 4400 U/kg/h for 24 h. The fibrinogen levels and partial thromboplastin times were measured before the treatment and every 6 h. On the cavography the following day, the occlusion of the SVC persisted, but the azygos major vein became permeable. After
a further 24 h of thrombolysis with the catheter in the thrombus, cavography (Fig. 2) showed good flow toward right atrium, with recanalisation of the SVC (diameter of 8 mm at level of the pericardial reflection and 21 and 13 mm above and under it, respectively). The stent was not placed because of the technical difficulty caused by the anatomical gradient. The central venous catheter was removed. The cyanosis and the oedema diminished respectively at 24 and 48 h of the fibrinolysis.

The dicumarin therapy was indicated for 6 months [4]; a new cavography did not show rethrombosis, and the transverse diameter in the pericardial reflection was 11 mm, and the same above and under it. It was stabilised, respectively at 15 and 12 mm.

Discussion

There are different alternatives for the treatment of SVC syndrome. Radiotherapy is useful in cases with a neoplastic cause. Medical treatment is of limited utility and is used as a last resort when all alternatives have failed. It consists of rest, elevation of the head, systemic anticoagulation and diuretics. Surgical treatment is based on autologous bypass of the saphenous vein, between the internal jugular vein or the brachiocephalic trunk and the right auricle [5], and is used mainly for patients with benign illness. Endovascular treatment [6] is carried out with stents. Although surgery is used mainly for patients with benign illness, it is used less and less, because of the aggressiveness of this technique, involving sternotomy, and because it cannot be justified when a minimally invasive technique such as the endovascular technique is available. This is why surgery would be restricted to patients in whom it was not possible to place a venous stent or in whom this had failed.

The use of vascular endoprosthesis gives good results in SVC syndrome, including improvement of symptoms, high permeability to medium term and low mortality. The cause of its failure is invasion of the vena cava by malignant tumours [1].

In our case the thrombosis of the SVC could be attributed to the catheter, as those made of polyvinyl, polyethylene and teflon are more thrombogenic than those made of silicone [7], and the hypercoagulability study was normal.

Fibrinolysis with urokinase was indicated, because it is not antigenic or pyrogenic, and its results are superior to those of streptokinase and tissue plasminogen activator [8]. We did not carry out transluminal percutaneous angioplasty, because the residual stenosis was not significant (<50%) [9], and we did not carry out endovascular treatment [6], because the dilation of the SVC above the pericardial reflection was 21 mm, against 13 mm in the inferior part. This caused such a significant anatomical gradient that it would have been very difficult to fix the stent while avoiding its migration [10].

There are two factors in the success of the thrombolytic treatment of thrombosis secondary to SVC associated with a central catheter. The first one is related to the time lapse between the onset of symptoms and the beginning of the thrombolysis. So the older the thrombus, the lower the possibility of smoothing it; however, it is advisable to attempt thrombolytic therapy, even if it is late, as in our patient [11]. The second factor is related to the incorporation of the central venous catheter in the thrombus, because it is a favourable factor for