

## CASE FOR DIAGNOSIS

# An opacity in the chest following a subclavian catheterization

L. Portel, G. Hilbert, D. Gruson, G. Gbikpi-Benissan, J.P. Cardinaud

### Case history

A 70 yr old male was admitted to a nephrology department with an acute anuric renal failure. In his past history, one could note a hypertensive cardiomyopathy with an atrial fibrillation, treated with amiodarone, digoxine, flecainide, trinitrine and fluindione, and a noninsulin dependent diabetes treated with metformine. One month before admission, the patient had been treated for an ischaemic cerebrovascular stroke, which was complicated by a rhabdomyolysis responsible for renal failure with a creatinine clearance measured at  $26 \text{ mL}\cdot\text{min}^{-1}$ .

On admission, clinical examination revealed an overall cardiac failure and fine crackles were heard over the pulmonary bases. Oedema of the lower limbs was also found. Biological analysis revealed a worsening of the pre-existing renal failure with a creatinine clearance of  $9 \text{ mL}\cdot\text{min}^{-1}$  without any associated ionic disorder. Diuretic treatment was initiated, enabling a transitory improvement before a rapid deterioration of the renal function occurred. The patient developed anuria, so that dialysis became necessary. The anticoagulant treatment was interrupted and treatment with heparin injections ( $250 \text{ IU}\cdot\text{kg}^{-1}\cdot\text{day}^{-1}$  in three injections) was initiated, 4 days before a dialysis catheter was set up. Injections immediately before and immediately after the venous catheterization were omitted. The prothrombin time was then measured at 96% and the activated

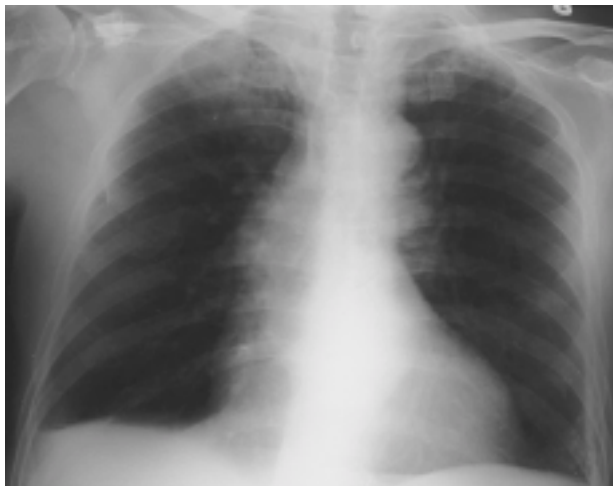


Fig. 1. - Pulmonary radiograph of right subclavian catheterization control.

Service de Réanimation Médicale B, Hôpital Pellegrin, Bordeaux Cedex, France.

Correspondence: L. Portel, Service de Réanimation Médicale B, Hôpital Pellegrin, Place Amélie Raba-Léon, F-33076 Bordeaux Cedex, France. Fax: 33 556796122.

a)



b)



Fig. 2. - Pulmonary radiographs performed 4 days after catheterization. a) front view; b) lateral view.

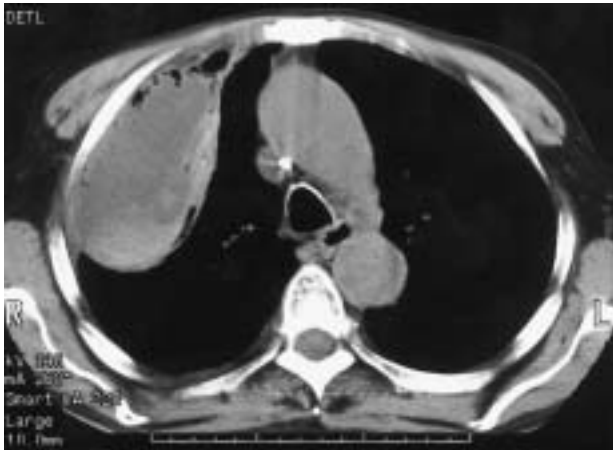


Fig. 3. – Thoracic computed tomography scan performed 8 days after catheterization.

partial thromboplastin time was 1.2 times the upper limit of normal for the laboratory. The platelet count, as well as the fibrinogen rate, were normal.

The insertion of the central venous catheter *via* the right subclavian vein was achieved after the second puncture attempt, and the control radiograph showed that the subclavian catheter was in adequate position (fig. 1). Three days after the insertion of the catheter, the radiograph appeared to be abnormal (fig. 2) and, at the same time, there was a massive loss of red blood cells with a haemoglobin concentration decreasing from 116 to 77 g·L<sup>-1</sup>. Eight days after the insertion of the catheter, the radiographic opacity worsened with an increase of size from 12 × 8 to 18 × 10 cm. A computed tomodensitometry was then performed (fig. 3), and was completed by an arteriography of the right upper limb (fig. 4).

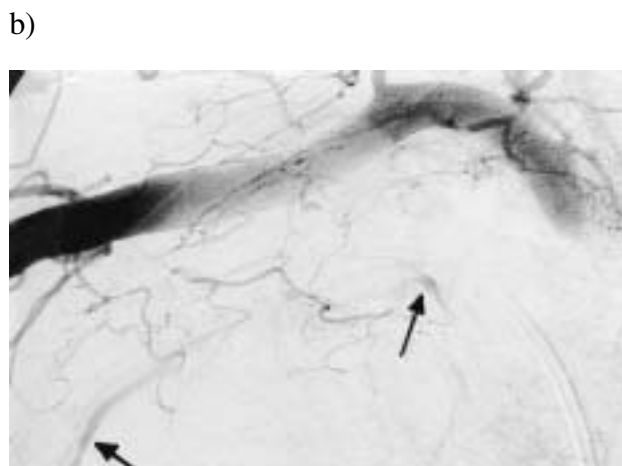
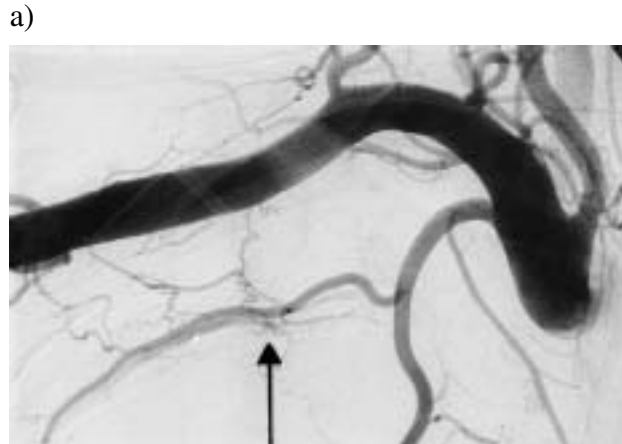


Fig. 4. – Right upper limb arteriography. a) early time; b) late time.

**BEFORE TURNING THE PAGE: INTERPRET THE CHEST RADIOGRAPH, THE COMPUTED TOMOGRAPHY AND THE ANGIOGRAPHY FINDINGS AND SUGGEST DIAGNOSIS**

### Interpretation

On the first chest radiograph (fig. 1), one could note the presence of a right apical opacity, a residual of a previous pulmonary tuberculosis, and the subclavian catheter in adequate position. The chest radiograph performed four days later (fig. 2a) showed a suspended right opacity of hydric tonality without any aeric bronchogram. The contacts with the thoracic wall were acutely angled. On the lateral view (fig. 2b), this opacity appeared to be anterior and suspended. The thoracic computed tomography (CT) scan (fig. 3) revealed a right anterior liquid effusion (7.6 Hounsfield units) in extrapleural location (outside the parietal pleural leaflet whose integrity is respected) and air-pockets pictures of iatrogenic origin.

On the right upper limb arteriography, there was a precocious abnormal venous opacification indicating the presence of an arteriovenous fistula (AVF) which was developed at the expense of an intercostal branch of the right internal mammary artery. On the first view (fig. 4; early time after bolus infusion) the source of the AVF is indicated by an arrow. On the second view (fig. 4b; late time) taken a few seconds later, the artery is flushed and the arrows show the double venous drainage (anterograde (arrow on the right) and retrograde (arrow on the left)).

### Diagnosis

It appears to be a right extrapleural haematoma from an iatrogenic arteriovenous fistula complicating a right subclavian venous catheterization.

### Treatment and clinical course

An attempt to embolize the leaking artery failed, because the selective catheterization of the branch of the internal mammary artery was impossible. Surgical treatment was not indicated due to the absence of any haemodynamic failure and the patient's precarious general state. The haematoma eventually healed spontaneously.

### Discussion

An extrapleural haematoma is a diagnostic trap due to its nonspecific presentation [1]. Several aetiologies can be found: thoracic trauma with costal fractures [2]; aneurysmal rupture of the thoracic aorta or of the superior vena cava [3]; and iatrogenic origin during catheterization of the subclavian vein or of the internal jugular vein, even though vascular lesions are often responsible for mediastinal haematomas [4, 5]. The blood loss can be extensive: MACHIN [6] reported a 500 mL·h<sup>-1</sup> bleeding after an initial 1.5 L discharge of blood in a case of blunt chest trauma.

The extrapleural location cannot be asserted by the thoracic radiograph, whereas the computed tomodensitometry makes the diagnosis possible. The integrity of the parietal pleural border characterizes the extrapleural location of the effusion. The border appears as a hypodense linear picture which corresponds to the subpleural fat that can be clearly seen between the hydric density effusion and the adjacent atelectasis of tissue density.

Although subclavian catheterization is more likely to damage the subclavian artery, one can see on the arterio-

graphy that this artery appears not to be injured. The bleeding arises from a supplementary branch of the internal mammary artery called the lateral costal branch. This branch is quite far from the area normally accessed during central catheterization. However, it belongs to the anterior chest wall and can be reached accidentally. This lateral costal branch of the internal mammary artery may occur in 10% of the population [7].

That an AVF should appear, complicating the insertion of a central venous catheter, has been recognized since JAMES and MYERS [8] first described it in 1973. Only 37 documented cases have been reported up to now [9] and their incidence seems to be near to 0.03% of the subclavian catheterizations [10]. Their location is asymmetrical: 30 of the 37 were located on the right side due to a different anatomical configuration. Their real frequency is likely to be underestimated, because most of them remain asymptomatic and finally heal spontaneously. In a study dealing with 1,500 subclavian venous catheterizations, FELICIANO *et al.* [11] did not describe any cases of AVF. Among the 13,800 cases of subclavian venous catheterizations reported by EEROLA *et al.* [12], there were 113 (0.9%) accidental arterial punctures, but there was no case of an AVF. However, in these two studies, the search for AVF was not systematic, which may explain why no cases were reported. Some authors [9, 10, 13] recommend that the subclavian area must be auscultated during the 2 weeks following any difficult subclavian catheterization, so as to look for any murmur with systolic reinforcement revealing an AVF, which would make it possible to evaluate their frequency more accurately. Three cases of AVF are reported by HANSBROUGH *et al.* [13], two of which occurred following an internal jugular venous catheterization, the other one following a subclavian venous catheterization. Even though a systolic and diastolic bruit was always heard, these three cases were asymptomatic and the evolution was favourable (twice spontaneously and once after surgical ligation).

For many years, the treatment was a surgical one, but now one relies most often on endovascular embolization techniques. In a series of seven patients, RICOLFI *et al.* [9] achieved success in all seven. Only one case needed a combination of surgery and embolization and for the six others endovascular embolization alone was sufficient to close the fistula. It is the authors opinion that the endovascular approach is a reliable and safe method to perform as a first treatment. Surgery is still indicated when the endovascular techniques fails or when a vascular injury needs surgical treatment.

The present observation emphasizes the necessity of a systematic clinical search for an arteriovenous fistulae when a central catheter is inserted, especially when the venous approach is performed on the right side and when insertion is difficult. The use of endovascular embolization is recommended.

**Keywords:** Arteriovenous fistula, complications, extrapleural haematoma, subclavian catheterization.

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