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CASE REPORT

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Iatrogenic fatal outcome of traumatic axillary aneurysm

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Abstract Five months after dislocation of the left shoulder a 66-year-old patient noted a swelling in the left axilla. After CT-scan a malignant tumour was suspected and an incisional biopsy was performed. A week later the patient died due to hemorrhaging from the biopsy wound. The autopsy revealed a false aneurysm of the axillary artery. The incision had damaged the external wall of the aneurysm leading to consecutive rupture. Post-mortem findings are presented.

Key words Traumatic axillary aneurysm · Improper anamnesis · Incisional biopsy · Fatal diagnostic treatment

Zusammenfassung Es wird über eine tödliche Komplikation bei nicht erkanntem traumatisch bedingtem Aneurysma der A. axillaris berichtet. Fünf Monate nach einer Schulterluxation bemerkte eine 66jährige Frau einen wachsenden Tumor in der linken Achselhöhle. Ein Computertomogramm zeigte einen Tumor, der Radiologe äußerte den Verdacht auf Malignität. Eine Woche nach der durchgeführten diagnostischen Punktion des Tumors verblutete die Frau akut aus der Biopsiewunde. Bei der Autopsie erwies sich der Tumor als falsches Aneurysma der A. axillaris. Die Biopsie hatte offensichtlich eine Wandschwäche und die Ruptur des Aneurysmas bedingt. Die Obduktionsbefunde werden vorgestellt und diskutiert.

Schlüsselwörter Traumatisches Axillarisaneurysma Ungenügende Anamneseerhebung · Offene Probeexzision Todesursächliche diagnostische Maßnahme

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Introduction

Damage to blood vessels after blunt trauma may cause serious diagnostic problems especially if complications arise at a later time. Failure to diagnose vascular complications can lead to death from internal hemorrhage or loss of the affected limb. Therefore only the inclusion of such complications into a diagnostic investigation can prevent such false conclusions (Böhler 1954; Köhl and Bassey 1991).

Case report

A 66-year-old woman who had a history of previous dislocation fell and suffered a dislocation of the left shoulder. Following self-reduction she was taken to the hospital. On examination there was a large hematoma of the shoulder and the upper arm, accompanied by pain and restricted movement. The patient received no treatment and was discharged with a residual perception disorder of the 4th and 5th finger.

Five months later a rapidly enlarging swelling appeared in the left axilla and a CT-scan revealed a massive tumour suspected to be malignant (Fig. 1). A biopsy was recommended which was carried out one month later. The pathologist diagnosed a conglomerate of necrotic detritus. Six days after the incision the patient suddenly noted a "bubbling" in the axilla and a short time afterwards she collapsed. The emergency physician found the patient uncon-

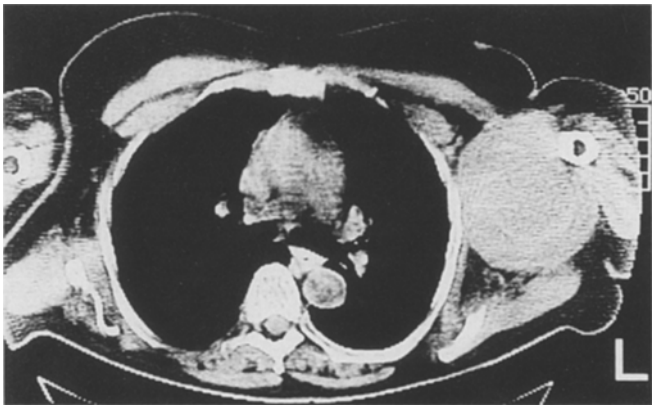

Fig. 1

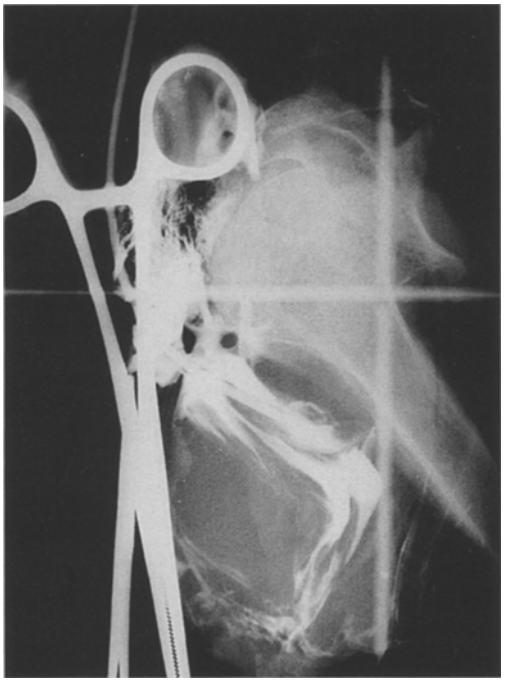

Fig. 2

Fig. 3

scious with copious hemorrhaging from the biopsy wound. Resuscitation remained unsuccessful.

At autopsy the left axillary artery was found to be stretched over a tumour measuring 12 × 12 × 10 cm. Filling with radiopaque medium revealed a slit-shaped flow throughout the tumour down to the incisional wound (Fig. 2). A cross section revealed a false aneurysm with fluid blood in the centre, with a radially increasing coagulation and organization (Fig. 3). The arterial wall lying next to the tumour was partially ruptured and a communicating hematoma had developed. The bioptic incision had obviously weakened the aneurysmal wall which finally ruptured due to blood pressure.

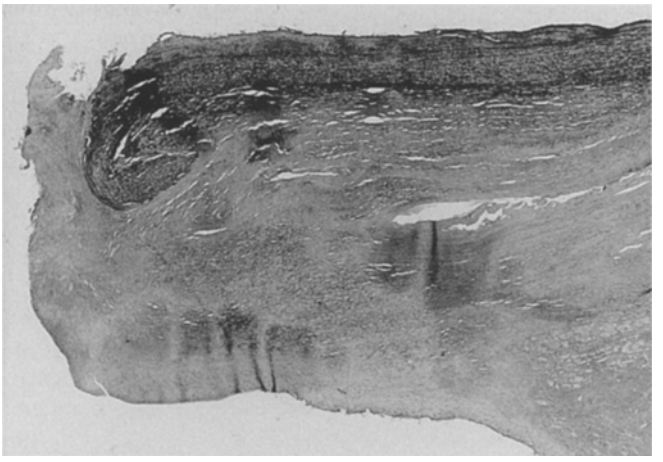

Fig. 4

The histological investigation revealed mild intimal sclerosis of the axillary artery and moderate to severe generalised atherosclerotic changes. At the site of the vascular rupture there was no dissection of the arterial wall but the borders of the laceration were surrounded by massive scar tissue (Fig. 4). In addition there was focal necrosis of the media with loss of nuclei and hyalinic homogenisation. There was no siderosis, no formation of cysts or mucoid substances. The aneurysm itself displayed a tough fibrous wall, focal granulation tissue and serous fluid.

Discussion

The delay between the injury to the shoulder and the vascular complication in this case is striking. In most cases of shoulder trauma, arterial damage occurs at an early stage. A few reports mention the occurrence of a false aneurysm up to 6 months after the accident (Fitzgerald and Keates 1975; Majeed 1985; Bhamra et al. 1989). As a rule this development is caused by a primarily ruptured intimal layer and subsequent forming of the aneurysm.

Brinkmann (1975) postulated that this development could be anticipated in the elderly and in atherosclerotic vessel degeneration as the tensile strength of the adventitial layer relatively exceeds that of the intimal-medial layer in these particular cases. Moreover traumatic damages to the vasa vasorum have to be taken into account as a pathological mechanism resulting in possible malnutrition of the vessel wall, subsequent ischemic necrosis, secondary rupture, and development of a false aneurysm (Brinkmann 1974).

In our particular case, no histological evidence was found supporting such pathogenetic conclusions. The cicatricial tissue at the arterial laceration merely indicated a period of at least several weeks between laceration and autopsy. The focal loss of nuclei in the media could be

caused by malnutrition (Brinkmann 1974), but it has been pointed out by previous investigators, that these changes may also be present in normal aging vessels (Cellina 1931; Schlattmann and Becker 1977). Cystic changes with pools of mucopolysaccharides, as is typically seen in cystic medial necrosis of the aorta as well as in non-traumatic dissecting aneurysms of the subclavian artery or muscular arteries (Persaud 1968; Leu 1993), could not be seen. Several hypotheses have been proposed for the mechanism of injury to the axillary artery. Henson (1956) and Drury and Scullion (1980) suggested that the artery may be fixed by the origins of the subscapular and circumflex humeral arteries and would be inflexible in cases of dislocation of the humerus. Brown and Navigato (1968) described the pectoralis minor muscle as a fulcrum over which the artery rides when the arm is abducted and forcibly rotated externally. Jardon et al. (1973), Stock et al. (1988), and Mustonen et al. (1990) suggested the concept of scar tissue formed due to previous dislocation of the shoulder fixing the artery and resulting in rupture. In addition to which scar tissue might reduce the amount of hemorrhage and strenghten the capsule of the aneurysm.

In general, when investigating cases of traumatic arterial rupture one may not expect uniform causality (Bratzke and Wojahn 1977). Most authors regard arteriosclerotic lesions as an important risk factor.

We think that our findings support the following hypothesis: shoulder dislocation led to a partial rupture of the axillary artery and hematoma compressing the brachial plexus. Previous dislocations may have induced the formation of scar tissue which limited hemorrhage and delayed the development of a large spurious aneurysm. Finally the incisional biopsy damaged the fibrous wall of the aneurysm resulting in rupture and fatal hemorrhage.

As to the failure to diagnose the aneurysm the following considerations should be taken into account: an aneurysm usually resembles a painless expanding pulsating tumour, however the intensity of pulsation depends on the wall of the aneurysm. The angiography showed that only relatively minor contrast flowed from the blood vessel to the site of puncture and so a pulsating tumour may have been missing. But it could be regarded as malpractice, that the physician failed to make a proper diagnosis. The lack of anamnestic information about the history of previous dislocation of the shoulder has obviously hindered a correct diagnosis and led to contraindicated diagnostic treatment with fatal outcome.

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