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Deep Venous Thrombosis of the Axillary and Subclavian Vein After Osteosynthesis of a Midshaft Clavicular Fracture

A Case Report

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Fracture of the clavicle, accounting for 2.6% to 5% of all fractures, is not uncommon and affects mostly young, active people.[16,17] In sports-related fractures, it is even the most frequent fracture.[6] Midshaft fractures account for approximately 80% of all clavicular fractures.[17] Nonoperative treatment with a figure-of-8 splint or a sling has been the historic gold standard, even with substantially displaced fractures. A recent multicenter, randomized clinical trial conducted by the Canadian Orthopaedic Trauma Society compared nonoperative treatment with plate fixation of displaced midshaft clavicular fractures.[5] An improvement of the functional outcome and a lower rate of malunion and nonunion were seen in the operative group, thus favoring rapid operative treatment of these fractures. Absolute indications for surgery are open fractures, fractures with neurologic or vascular complications, dislocation or a fracture with risk for skin perforation, and a floating shoulder. In our experience, open reduction and internal plate fixation as a treatment of displaced clavicular fractures in professional or semiprofessional athletes can significantly shorten the rehabilitation period. The major complications in operative treatment include deep infection, plate breakage, nonunion, and refracture after plate removal, but those complications are rather rare.[4]

Nonoperative treatment can give rise to late complications; delayed union, nonunion, or malunion; chronic pain; residual paresthesia; partial loss of shoulder motion; thoracic outlet syndrome; and issues with cosmetics are all reported.[11] Direct trauma to the venous plexus can cause upper extremity deep venous thrombosis (UEDVT), but only 4 cases after a clavicular fracture are reported in the literature.[2,12-14] Upper extremity deep venous thrombosis after osteosynthesis of a clavicular fracture has never been reported to our knowledge. We present a case of a professional motocross racer who developed a thrombosis in the subclavian and axillary vein after plate fixation of a midshaft fracture of the clavicle.

CASE PRESENTATION

A 20-year-old top-level motocross racer crashed during the qualifying session of a Grand Prix in the middle of the season and sustained a left midshaft clavicular fracture. The fracture was angulated, but there was no shortening of the bone (Figure 1). After the diagnosis, the patient returned on a 2-hour flight to our hospital in his home country.

Aiming for a fast return to competition, we decided on an operative treatment the day after the accident. Operative details include beach-chair positioning, general anesthesia, and standard surgical procedure by means of open reduction and internal plate fixation with uneventful course. Patient characteristics showed a tall morphotype with a body mass index of 22.4 and an arm length of 76 cm. There was no medical or surgical history concerning the involved upper limb. Postoperatively, he received standard analgesic treatment (paracetamol intravenously) combined with 24-hour antibiotic prophylaxis (cefazolin). The day after the surgery, the patient complained of vague neck pain, which was treated with analgesics (paracetamol and diclofenac) and mild physiotherapy with good result. He was discharged from the hospital 2 days after surgery. The patient was allowed to mobilize actively and passively as tolerated. He wore a sling for about 1 week for support and comfort. Two weeks later during a routine consult, his

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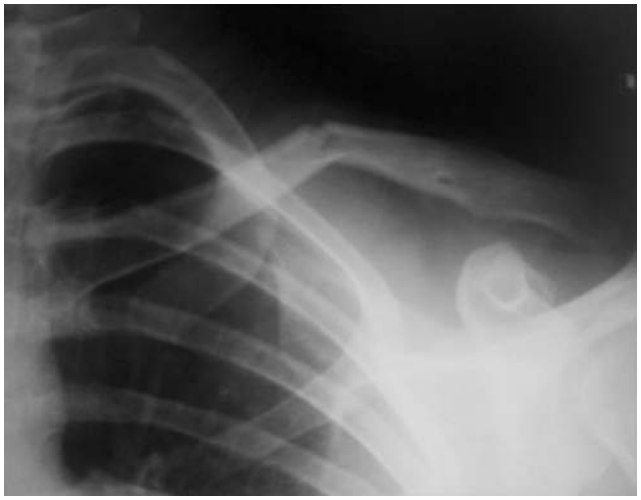

Figure 1. Radiograph of the left clavicle: midshaft fracture with angulation, no gross displacement.

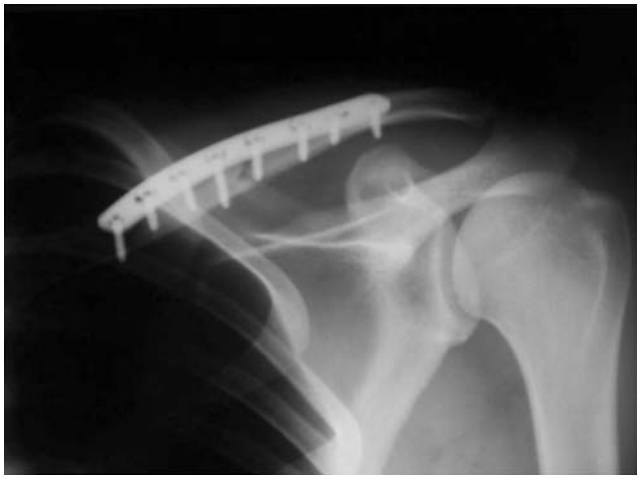

Figure 2. Radiograph after osteosynthesis shows the reduced fracture with an adequate position of the plate and screws.

left arm hurt and was highly sensitive after minor pressure. Clinical examination showed a completely swollen arm with clearly expanded veins. The skin of the upper arm was red-colored and warm. Passive mobility of the shoulder was normal.

Radiography of the left clavicle showed a well-reduced fracture with hardware position unchanged (Figure 2). Echo Doppler examination and adjacent venography indicated a thrombosis of the axillary and the subclavian vein with an extensive collateral circulation (Figure 3). Treatment with therapeutic doses of nadroparin 0.8 mL, a low molecular-weight heparin (LMWH), was started immediately combined with a compressive custom-made bandage from hand to shoulder, figure-of-8 bandage, and rest. After 10 days, no resolution was noticed and the

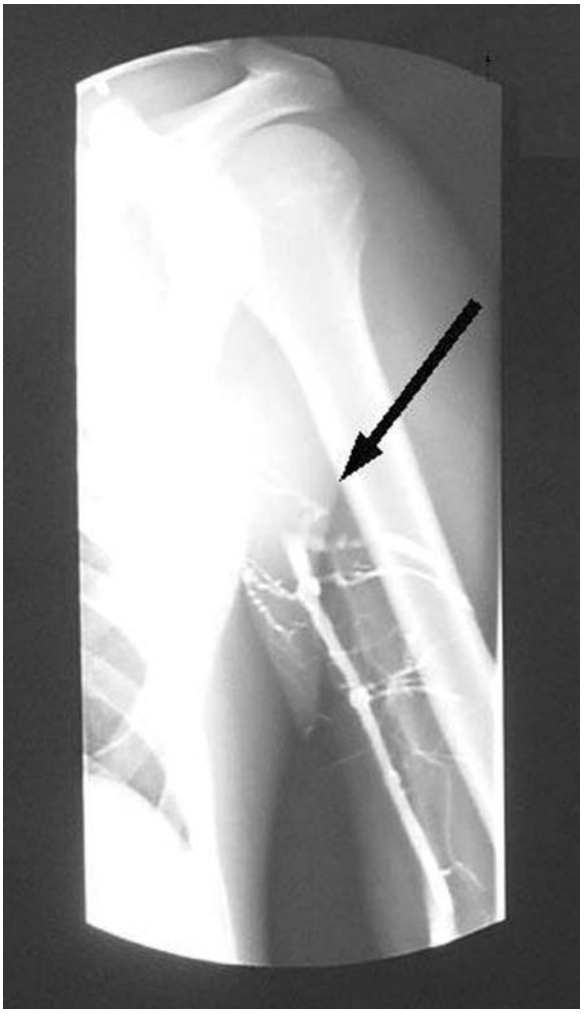

Figure 3. Venography: occlusion of the axillary vein.

patient was referred to a university hospital for local catheter-guided thrombolysis with urokinasis injected in the axillary vein. The axillary vein could be opened, but the subclavian vein remained occluded after 2 attempts. Nonoperative treatment using LMWH and the bandage was continued. Over time the collateral venous circulation started to expand significantly.

After 2 months, a new venography revealed a subclavian vein that was still not completely open but was bridged by recanalization of several well-developed distal collaterals that drained into the subclavian vein. In addition, swelling resolved and LMWH therapy was stopped. At that moment the patient was allowed to begin sport activities as tolerated. Six weeks after the surgery, the fracture had healed completely on radiography (Figure 4) and another 4 weeks later, the patient was able to resume his sports activities. He started racing again at 14 weeks postoperatively. At the latest visit, 2 years postoperatively, the patient had no remaining symptoms and only some venous collaterals were clinically visible.

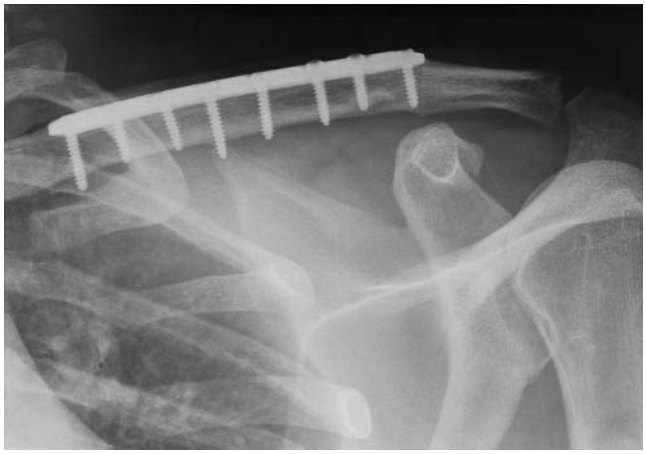

Figure 4. Radiograph showing a healed fracture.

DISCUSSION

Virchow[19] was the first to describe the major causes of venous thrombosis: injuries in the vascular endothelium, alterations in normal blood flow, and hypercoagulability. This triad still counts as the basis for the etiogenesis of thrombosis. Many orthopaedic procedures create a higher risk of developing a thrombosis. Primary thrombosis in the subclavian/axillary vein is rather rare. It was described by Sir James Paget in 1875 for the first time and later independently by Leopold von Schroetter in 1884. The Paget-Schroetter syndrome is characterized by spontaneous thrombosis of an upper extremity vein, especially in young, active people after repetitive, strenuous activity. This kind of thrombosis is most often referred to as effort thrombosis. In the current case, a preexisting Paget-Schroetter syndrome cannot be excluded because of the heavy loading of the upper extremity during motocross racing. Patients with a secondary thrombosis generally have one or more predetermined factors such as a malignant neoplasm, chemotherapy, neurologic disease with paresis, central venous catheter or pacemaker, varicose veins, and superficial vein thrombosis.[9] Congenital or acquired hypercoagulability is described in 20% to 30% of deep venous thromboses (DVTs).[15] Congenital hypercoagulability is most frequently seen in factor V Leiden, which develops resistance to antithrombotic activity of protein C. Prothrombin mutation, anticardiolipin syndrome, lupus anticoagulant, and hyperhomocysteinemia are other molecular dissociations associated with DVT. Congenital deficiencies of endogen protein C, protein S, and antithrombin III are additional coagulopathies that can lead to thrombosis.[8]

There are some other general and acquired risk factors associated with a higher prevalence of thrombosis: age >40 years, sepsis, duration of hospital stay, air travel, congestive heart failure, nephrotic syndrome, obesity, varicose veins, oral contraception, estrogen therapy, infarction, inflammatory bowel disease, smoking, pregnancy, prolonged anesthesia (>30 minutes), femoral venous catheter, hyperglycemia, hypertension, and hyperlipidemia. History of thromboembolic events, malignancy, and a family history of thrombosis of unknown origin are risk factors for the development of DVT as well. In a systematic review of over 250 articles on the described risk factors, Edmonds et al[7] found a significant correlation between prevalence of postoperative thrombosis and age, obesity, history of thromboembolism, varicose veins, oral contraception, malignancy, factor V Leiden, general anesthesia, and orthopaedic surgery. In this review, however, no evidence for higher risks in developing DVT was found concerning pregnancy, hormonal therapy, gender, ethnicity, chemotherapy, thrombophilia, cardiovascular factors, blood type, and smoking. Our patient had none of these risk factors.

Symptoms of thrombosis of the upper extremity are aspecific, including pain, cramps, edema, and paresthesia of the arm. Edema mostly affects the entire arm. In the case of thrombosis in the axillary vein, a collateral circulation will most often develop from the shoulder to the contralateral neck. When thrombosis is located in the subclavian vein, the collateral veins stretch from the ipsilateral posterior neck or shoulder to the contralateral side of the neck.[1] Some of the patients with upper extremity thrombosis remain asymptomatic.[3] The most important complication is pulmonary embolus, which may result in pulmonary hypertension or even death. Venography is the gold standard to establish the diagnosis, but a color-flow Doppler ultrasonography is probably the least invasive and most readily available test. Other examinations such as CT angiography or MRI angiography are good alternatives but the downsides are cost and availability. Treatment starts symptomatically with elevation and analgesia, followed by anticoagulants. Anticoagulants form the cornerstone of the nonoperative treatment and bring satisfactory results.[18] The goal is to help maintain patency of venous collaterals and to reduce thrombus propagation. There is no difference between the treatment of a UEDVT and DVT of the lower limb.[10] Other more invasive treatments have also proven their success: thrombectomy, systemic or catheter-guided thrombolytic therapy, and balloon angioplasty with stenting. Catheter-guided thrombolysis restores the venous retour more rapidly, minimizes the endothelial damage, and diminishes the risk for complications (eg, postthrombotic syndrome). This is generally recognizable through a chronic edema of the arm and hand. In case an extrinsic venous compression is responsible for the thrombosis, as is seen with thoracic outlet syndrome, an early anatomic correction is an option (rib or clavicular resection).

In our patient, the occurrence of a UEDVT had no influence on fracture healing, as bony healing was observed on radiographs 6 weeks postoperatively.

CONCLUSION

No case reports have been reported in the literature describing an acute postoperative venous thrombosis in the ipsilateral arm after surgical treatment of a clavicular fracture. The thrombosis observed in the described patient is most likely the consequence of the trauma and the subsequent surgery. An underlying Paget-Schroetter syndrome, however, cannot be excluded. The specific cause

of a thrombosis in the upper extremity is in many cases difficult to assess. It is most often clinically apparent in an aspecific way. In the case of operative treatment of clavicular fractures, precaution is needed to avoid and/or detect possible complications of DVT of the upper extremity. We believe that the use of antithrombotic prophylaxis could be considered after operative treatment of clavicular fractures, especially in professional athletes.

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