CASE REPORT

Upper extremity deep vein thrombosis following a humeral fracture: a case report and literature review

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Accepted 30 October 2004

Case report

A 72-year-old female accidentally fell while on holiday in Madeira sustaining an undisplaced simple fracture to the surgical neck of her left humerus. She attended the local clinic where her shoulder was strapped tightly and immobilized by the local medical team to protect it for her journey home.

On arrival back in the UK she attended the hospital fracture clinic where non-operative management was continued but the tight strapping was changed to a collar and cuff sling.

Five days later, she was referred to A + E by her GP with symptoms of pain and swelling in her left antecubital fossa. On examination, she had bruising at the level of the fracture and swelling at the elbow, mostly anteriorly. Elbow flexion and extension were also limited. She had palpable thrombosis in her superficial left forearm veins. An urgent ultrasound scan was requested, which confirmed thrombosis in the superficial and deep venous systems of her left upper arm.

She was admitted to the ward and commenced on treatment-dose low molecular weight heparin while being warfarinised. The hospital haematologists recommended warfarin treatment for 6 months. After 3 months, her fracture had united with almost full recovery of shoulder function following physiotherapy.

Literature review

Introduction

Upper extremity deep vein thrombosis (UEDVT) is an increasingly important clinical entity with potential for considerable mortality. Once considered rare, UEDVT has become more common over the past few decades while forearm DVT remains rare. This is directly related to the increased use of central venous catheters and comprises about 2% of all deep venous limb thromboses.

Although many risk factors for UEDVT have been identified in the literature, bony trauma has not been mentioned but has been recognised as a triggering factor for lower extremity DVT for several years.
Humeral fractures are common, particularly in the osteoporotic or elderly patient. They can cause significant disability and carry a relatively high rate of non-union (5—10%).

UEDVT is most commonly found in the axillary or subclavian veins. It can be classified as primary or secondary on the basis of pathogenesis.

Primary UEDVT is a rare disorder (2 per 100,000 persons per year) and refers to either effort-induced thrombosis (Paget-Schroetter syndrome) or idiopathic origin. In one study, however, 25% of patients presenting with “idiopathic” UEDVT were diagnosed with cancer—most commonly lung cancer or lymphomas—within 1 year of follow-up. As many as 20% of patients present with apparently spontaneous episodes and the prevalence of inherited coagulation disorders in patients with this disease ranges from 10 to 26%.

Secondary thrombosis accounts for most cases of UEDVT and develops in patients with central venous catheters, pacemakers or cancer. Other risk factors include concomitant or previous lower limb DVT, hypercoagulable states including pregnancy and contraceptive use, thoracic outlet syndrome, and anatomic anomalies, and systemic illnesses such as renal failure. Thus UEDVT typically occurs in patients with a systemic illness who have a central venous catheter.

UEDVT is often completely asymptomatic in presentation, highlighting the need for awareness in the critically ill patient. More often, though, patients complain of vague neck or shoulder discomfort, arm swelling, discolouration or pain. Obstruction of the superior vena cava can cause facial oedema, blurred vision, vertigo, or dyspnoea. Examination may reveal a low-grade fever, sinus tachycardia (if venous return to the heart is reduced as in SVC syndrome), mild cyanosis of the involved extremity, a tender cord, arm or hand oedema, supraclavicular fullness, or venous distension (jugular, chest, or distal). If a central venous catheter is present, one or more ports may be occluded.

Although clinically suspicious, less than 50% of these symptomatic patients will have imaging evidence of UEDVT. Likewise, up to 50% of DVTs are asymptomatic. It is, therefore, essential to have evidence of UEDVT by diagnostic imaging before anticoagulation. Blood tests (e.g. ‘D-dimers’—a measure of clot breakdown products) can be helpful if negative but a positive result still requires conclusive imaging.

Ultrasound scanning is the initial diagnostic test of choice but others include contrast venography, contrast CT scanning, and magnetic resonance angiography. These are compared in Table 1.

### Table 1

<table>
<thead>
<tr>
<th>Test</th>
<th>Advantages</th>
<th>Disadvantages</th>
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</thead>
<tbody>
<tr>
<td>Ultrasound</td>
<td>Inexpensive</td>
<td>Poor detection of central thrombus</td>
</tr>
<tr>
<td></td>
<td>Non-invasive</td>
<td>Clavicular acoustic shadowing</td>
</tr>
<tr>
<td></td>
<td>Reproducible</td>
<td></td>
</tr>
<tr>
<td>Contrast venography</td>
<td>Inexpensive</td>
<td>Contrast sensitivity</td>
</tr>
<tr>
<td></td>
<td>Good anatomy</td>
<td>Phlebitis worsening thrombus</td>
</tr>
<tr>
<td></td>
<td>Assessment of treatments</td>
<td>Contraindicated in pregnancy</td>
</tr>
<tr>
<td>Contrast CT</td>
<td>Central thrombus detection</td>
<td>Contrast (as above)</td>
</tr>
<tr>
<td></td>
<td>May detect extrinsic compression</td>
<td>Contraindicated in pregnancy</td>
</tr>
<tr>
<td>Magnetic resonance</td>
<td>Accurate detection</td>
<td>Expensive</td>
</tr>
<tr>
<td></td>
<td>Excellent anatomy, including</td>
<td>Limited availability</td>
</tr>
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|                           | collaterals and flow                | Patient exclusion, e.g.:
|                           |                                     | • claustrophobia                                      |
|                           |                                     | • metallic implants                                   |

Treatment

Treatment begins symptomatically with elevation and analgesia. Compression garments are a matter of controversy. Anticoagulation is the cornerstone of therapy and typically begins with ‘high dose’ unfractionated or low molecular weight heparin (LMWH) administered sub-cutaneously while oral warfarin is commenced. The heparin is discontinued when the warfarin reaches a therapeutic level—typically an INR of 2.5 in first-event deep vein thrombosis. It is recommended that warfarin be continued for a minimum of 6 weeks in post-operative calf DVT without other persistent risk factors. This increases to 3 months in non-surgical patients and 6 months in those with proximal vein thrombosis or pulmonary embolus.
Complications

Pulmonary embolus complicates UEDVT in up to 36% of patients and may be the presenting feature. Venous catheter removal is also a risk factor as fibrinous coatings may break loose and embolise.

Post-thrombotic syndrome is caused by venous hypertension secondary to outflow obstruction and valvular injury. Symptoms vary from mild to severe oedema with chronic pain, and skin ulceration of the limb. It may occur in over half of patients and can present up to 10 years later. Its incidence may be reduced by the use of elastic compression stockings in the initial period.

Recurrence usually occurs in those with underlying medical problems or secondary UEDVT. Hence, the short-term mortality rates are very high compared with patients who have DVT of the lower extremity.

Other complications include infection, SVC obstruction, thoracic duct obstruction, and loss of venous access. The latter is of great significance in critical care for the administration of nutrition and medication as well as patient monitoring.

Prevention

Primary UEDVT is difficult to predict. Screening for thoracic outlet obstruction or coagulation disorders has never been shown to be cost-effective. Long-term anti-coagulant prophylaxis is normally reserved for the prevention of secondary UEDVT in high-risk patients.

Monreal et al. have shown that once-daily subcutaneous administration of LMWH in cancer patients with central venous catheters greatly reduces the frequency of UEDVT, ensuring that the first dose is given 2 h prior to catheterisation. This has fewer bleeding complications, even in those with poor nutrition or liver disease, than the previously suggested ‘mini-dose’ (1 mg) of warfarin.

Discussion

This patient had a secondary UEDVT caused by the precipitating factors of trauma, tight strapping and possibly air travel.

We have already discussed trauma as a known cause of lower limb DVT, but in the literature, humeral fracture is not a recognised risk factor for UEDVT.

There is no published evidence that quotes tight strapping as a cause for DVT but we know that compression or obstruction of the venous drainage of the upper limb can lead to venous stasis and hence thrombosis.

Air travel is a known risk factor for lower limb DVT but only a flight time of more than 3 h is quoted as significant. One small study has looked at the association with UEDVT but this was inconclusive. Other than the patient’s age (72) we could find no other obvious risk factors.

In summary, there were many factors that may have contributed to this lady’s UEDVT but we propose that it might not have occurred if the arm had not been tightly strapped. We also recommend that humeral fracture is added to the list of risk factors for UEDVT in the same way that lower limb fractures are well known to cause lower extremity DVT.

Learning points

Avoid tight strapping of injured limbs for sustained periods.

Have a low index of suspicion for UEDVT in critically ill patients or those with venous catheters and have a low threshold for imaging.

Consider the use of LMWH in patients with long-term venous catheters, particularly in the presence of malignancy.

Consider UEDVT in the presence of underlying injury.

References


Further reading
