Psychiatric assessments: how much is too much?

This anonymous author offers valuable insights into how psychosis is best managed in emergencies

For now, you and I differ about what we think this building, this institution, is for

Imagine groping your way up the shallow steps of a cinema auditorium, full of a powerful film that will colour the rest of your weekend. Make it a film that has got your heart pumping, made you sweat or startle. For good measure, an auditorium light has just exploded, showering one row of the audience with pieces of glass. Multiply all this 10-fold, and it's roughly how I'm feeling now.

I am thinking fast; new fears flood in at the speed of perception. I'm noticing some things you—the interviewing doctors—do not. Yes, hallucinations, some of them; fight or flight is also heightening my senses. I don't know that there is any future. I am already passing through "stages of grief"—much as someone would if awaiting execution. This figures—I believe I am facing execution. The thought, "I'm experiencing psychosis"—terrifying when it comes—is unavailable; it's all too new for that. It follows that all talk of "doctors," "treatment," or "admission" feels like so much persecution and deceit.

You find my thinking jumbled and confused, the quantity and register of my speech are fluctuating wildly. But I'm also hyper-attentive to language, as anyone would be in a high stakes situation (was that "talk to" or "torture"?). In an idle moment a nurse at the foot of my bed has concluded an anecdote with a hearty "I could have killed him," and perhaps she thought that was inaudible, but I heard it. And I thought you wanted me to hear it, that it was in the script. Because for now, you and I differ about what we think this building, this institution, is for.

In such a state, someone like me may seem beyond reassurance. But you can help—there are ways you can avoid reinforcing my fears.

First of all: duration. Sleep deprivation plus questioning is a powerful combination. Someone with an onset of acute psychosis is probably already experiencing the effects of sleep deprivation. So it doesn't help if doctors are over-cautious in making a decision, because a deferred decision equals more questioning, which means more sleeplessness and growing desperation.

In my case I was looked at by two emergency departments within 36 hours. I was interviewed by more than a dozen doctors, most of these over a 15 hour period.

A second, related factor: noise. I was told to sleep but the nurses carried out a full volume conversation among themselves throughout the night. Inevitably a particular word here or there would set off fresh panic. In my room on the ward I was woken by strip lighting every 15 minutes, by that point after many days without any real sleep. Maybe this is an unavoidable part of suicide watch, I don't know. But exposure to another patient's music (by day) surely isn't. The staff could see it was comforting for someone to play a single song on repeat. What they couldn't understand was the sinister meaning that had for me.

Third factor: the style of interviewing. If a doctor asks questions in a blank, detached way, it feels very frightening. A person with psychosis has not lost all ability to recognise and respond to social cues: intrusion, blankness, kindness. For example, if a doctor sits behind a desk, making eye contact but using deliberate silence to elicit my next move—and my normal civil rights hang in the balance—I will find this threatening, disorienting. It wouldn't look out of place in a police drama.

In my experience, the best communicators use calm and clarity; they treat the distressed person as a person. Paramedics are often good at this. For example, an ambulance crew applying the seat belts made this much more bearable for me by talking through the procedure—as if demonstrating it to someone in training. But the tone of the consulting room—quasi-conversational curiosity yet unmistakable authority—is alarming. By contrast, a psychologist who gave me cognitive behavioural therapy for psychosis (both as an inpatient and after discharge) often explained the process behind what she was doing—transparency is therapeutic.

Finally: posture and body language. A pharmacist once explained to me that my drugs, which I had spent days resisting and which had then given me uncomfortable side effects for weeks, had had little therapeutic effect and needed to be switched. He knelt down to my level while delivering this news and spoke with tangible sincerity. He was communicating that it was worth trying again with a different drug and, in effect, that he wasn't out to harm me. I felt cared for by the fact that he had taken the time to come and explain this on the ward, rather than over the counter at the external dispensary. I persisted with that new drug.

THE BOTTOM LINE

- Consider how to make any "wait and see" in the emergency department less of an ordeal. Consider how the patient can be best insulated from noise, sights (processions of ill or injured people), and smells. Make the decision (or, if you must, postpone it), but avoid multiple interviews covering the same ground
- Remain human. Inauthentic curiosity or blank expression may come across as over or under confidence (dominance, weakness, callousness) or as a calculated refusal to communicate. It may provoke understandable hostility. These first impressions last and may affect a patient's recovery and willingness to engage with treatment
- Find the person who calms things down. Ask others, including nurses and paramedics, how they communicate effectively with highly distressed people. If a patient seems to find an individual staff member worrying, consider what scope there might be for swapping places with another person in the team to see if that helps

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PRACTICE POINTER

Superficial thrombophlebitis (superficial venous thrombosis)

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Superficial thrombophlebitis (increasingly being called superficial venous thrombosis) is inflammation of the superficial veins associated with venous thrombosis. Traditionally, it has been considered a benign, self limiting disease of the lower extremity. However, it can affect most superficial venous systems in the body and importantly can be associated with deep vein thrombosis and pulmonary embolism. Treatment is aimed at symptomatic control and prevention of these serious and potentially fatal complications. Treatment options are variable and controversial.

How common is superficial thrombophlebitis?

The incidence of superficial thrombophlebitis remains unclear but is thought to be higher than that of deep vein thrombosis, which is estimated at about one per 1000.[1] Although age is not an independent risk factor, the incidences of other risk factors increase with age, making superficial thrombophlebitis more common in older people, and more common in women (50–70%).[2-4] However, complications are less likely in those over 60 years old.[5]

How is it diagnosed?

Superficial thrombophlebitis is a clinical diagnosis. Patients usually present with pain and discoloration (redness in the acute phase progressing to a brown, haemosiderin based pigmentation over days to weeks) over the affected superficial veins (fig 1). On palpation, the vein is tender and hard. Extensive limb swelling should raise the suspicion of deep vein thrombosis rather than superficial thrombophlebitis.

Infection and thrombophlebitis can appear similar. Pain may feature in either condition. Infection as a cause of superficial thrombophlebitis or an alternative diagnosis should be suspected if there is evidence of local skin trauma. However, infection usually involves a diffuse area of skin, unlike the localised signs in superficial thrombophlebitis alone. Fever, swelling, and pus drainage can be present in 44%, 37%, and 9% respectively of patients with infection.[6]

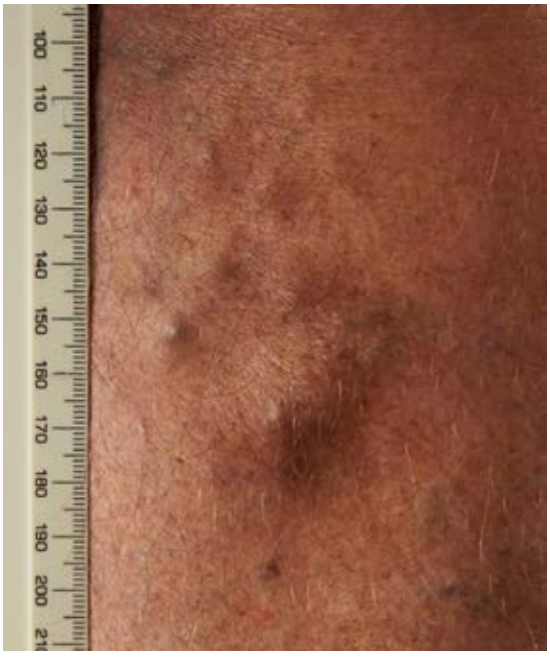

Fig 1 | Typical superficial thrombophlebitis within a calf varicosity. Note the brown haemosiderin discoloration and lumpiness

THE BOTTOM LINE

- Superficial thrombophlebitis is usually a benign, self limiting disease, but consideration should be given to specialist referral for duplex ultrasound imaging and further management
- Patients with limited (below knee) superficial thrombophlebitis without evidence of deep vein thrombosis can be safely managed in primary care with non-steroidal anti-inflammatory drugs and compression
- Patients with thrombosis near the saphenofemoral or saphenopopliteal junction should be considered for surgical ligation or anticoagulation
- Surgery should be considered in those with persistent symptoms or evidence of thrombus propagation despite medical treatment

What are the causes of superficial thrombophlebitis?

Superficial thrombophlebitis shares the same aetiological factors as other thrombotic disorders; it can also develop "de novo." Prolonged immobility, a hypercoagulable state, or trauma to a vessel wall (such as with intravenous cannulation) may predispose to thrombophlebitis. Previous episodes of superficial thrombophlebitis with or without deep vein thrombosis predispose to subsequent episodes, but varicose veins remain the most important clinically identifiable risk factor. Other risk factors include prolonged travel, recent surgery, pregnancy, oestrogen based hormone therapy, and malignancy (5–20%).[7] Varicose veins are by far the most common aetiological factor, in up to 88% of cases.[7]

Superficial thrombophlebitis and varicose veins

The prevalence of superficial thrombophlebitis in patients with varicose veins ranges from 4% to 59%[2 8 9] and is more common in the great saphenous system rather than the small saphenous system. This relationship between superficial thrombophlebitis and varicose veins means that superficial thrombophlebitis is an indication for consideration of varicose vein treatment; primary care practitioners should refer such patients to a specialist (such as a vascular surgeon) for an opinion regarding treatment of varicose veins.[10]

What are the types of superficial thrombophlebitis?

- Sterile superficial thrombophlebitis—Accounts for most presentations.
- Traumatic superficial thrombophlebitis—Occurs after limb injury and may be associated with ecchymosis of the surrounding tissue. Intravenous cannulation and infusions of irritant products can be causative, including the chemical thrombophlebitis produced by sclerotherapy during treatment of varicose veins.
- Infective thrombophlebitis—Mostly caused by prolonged intravenous cannulation causing infection and thrombosis. Appropriate antibiotics should be used for treatment.
- Migratory thrombophlebitis—Recurrent superficial thrombophlebitis at various separate sites without an identifiable local cause. This can be associated with an underlying malignancy, particularly carcinoma of the pancreas.[11]

What are its complications?

- Venous thromboembolism—Superficial thrombophlebitis may coexist with deep vein thrombosis in 5-53% of patients.[12-14] When superficial thrombophlebitis involves veins near the junction with the deep venous system, the risk of deep vein thrombosis and pulmonary embolism can be as high as 18%.[12-14]
- Skin hyperpigmentation over the affected vein.
- Infection and abscess formation.

How is it investigated?

Accepting that superficial thrombophlebitis is a clinical diagnosis, it remains important to identify any possible cause and determine the extent of thrombosis, its relation to the junctions between superficial and deep venous systems and whether any coexisting deep vein thrombosis is present.

Venous duplex ultrasound scanning

Duplex ultrasound is considered the optimal venous imaging modality and is recommended to confirm the diagnosis (fig 2), exclude deep vein thrombosis, and define disease extent. Clinical examination alone will underestimate the extent of superficial thrombophlebitis in up to 77% of cases.[5] Additionally, an associated deep vein thrombosis is reported in 6–53% of cases.[8] As such, consider all patients with suspected superficial thrombophlebitis for specialist referral, duplex imaging as necessary, and ongoing management.

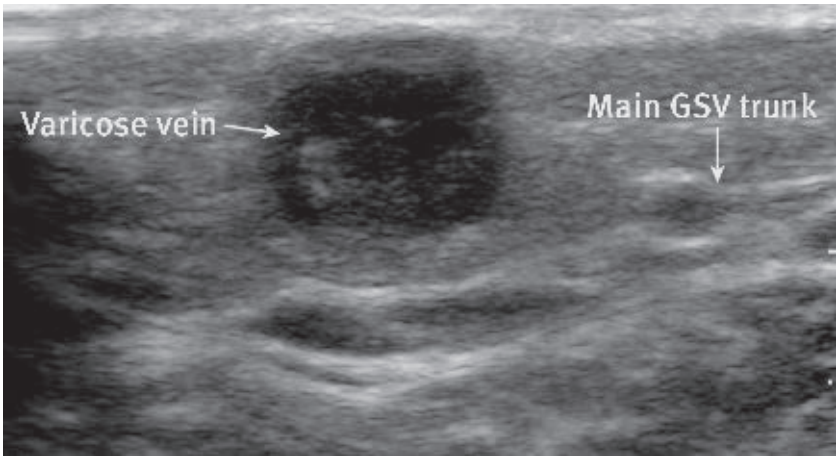

Fig 2 | Typical duplex ultrasound appearance of superficial phlebitis involving a varicosity arising from the great saphenous vein (GSV). Note the echogenic material within the varicose vein

Hypercoagulability

In the absence of a local cause, an underlying occult condition may be present. There are no conclusive studies showing a causative relationship between hypercoagulable states and superficial thrombophlebitis. However, there is a relationship between superficial thrombophlebitis and the prevalence of hypercoagulability. Some argue that, in the absence of an obvious causative factor (such as varicose veins, trauma, cancer), all patients presenting with extensive superficial thrombophlebitis should undergo screening for coagulation anomalies.[8] The main coagulation abnormalities associated with superficial thrombophlebitis are factor V Leiden mutation (16%), prothrombin 20210 mutation (10%), and deficiencies in antithrombin III, protein C, or protein S (10%).[15]

D-dimer

D-dimer is a fibrin degradation product; its concentration is often elevated in deep vein thrombosis and pulmonary embolism and may be increased in cases of superficial thrombophlebitis. As such, it is of little value in differentiating superficial thrombophlebitis from deep vein thrombosis and does not help in diagnosing superficial thrombophlebitis.[16]

How is it treated?

The treatment of superficial thrombophlebitis remains variable and controversial. However, therapeutic strategies must include symptomatic relief, limitation of thrombosis extension, and, very importantly, reduction of the risk of pulmonary embolism.

Currently, there is no single, evidence based therapy.[17] A recent Cochrane review examined a range of treatment modalities including hosiery, fondaparinux, various formulations of heparin, topical and oral non-steroidal anti-inflammatory drugs, and surgery.[1] The review concluded that the available evidence on oral treatments, topical treatment, and surgery was too limited to inform clinical practice about the effects of these treatments on venous thromboembolism or superficial thrombophlebitis extension. With respect to anticoagulants, the review concluded that a prophylactic dose of fondaparinux for 45 days seemed to be a valid therapeutic option.

It is currently accepted in the UK that in cases of limited (below knee) superficial thrombophlebitis without evidence of deep vein thrombosis, compression and non-steroidal anti-inflammatory drugs alone will suffice by providing symptomatic relief. However, if thrombus extends to the saphenofemoral or saphenopopliteal junctions prophylactic use of low molecular weight heparin may be indicated. Surgical intervention is a controversial option if anticoagulation is contraindicated or not tolerated, but it may compound the risk of venous thromboembolism.

Hosiery

Graduated compression helps improve venous flow and can increase local and regional intrinsic fibrinolytic activity. If tolerated, all patients with superficial thrombophlebitis may benefit from compression stockings with or without adjunctive treatments.

Non-steroidal anti-inflammatory drugs (NSAIDS)

The role of NSAIDs in venous thromboembolic disease is ill defined, but they are commonly prescribed in the UK to manage local symptoms. They reduce extension of superficial thrombophlebitis and its recurrence but have no effect on the incidence of venous thromboembolism.[1]

Anticoagulation

The most widely used anticoagulants are unfractionated heparin, low molecular weight heparin, and, more recently, fondaparinux. A randomised controlled trial showed that anticoagulation with unfractionated heparin, low molecular weight heparin, and warfarin were all superior to compression therapy alone in reducing superficial thrombophlebitis extension.[18] More recent studies show that the use of low molecular weight heparin is superior to unfractionated heparin both in prophylactic and therapeutic doses.[19]

Fondaparinux is a newer anticoagulant and has been shown to significantly reduce the symptoms of superficial thrombophlebitis, as well as reduce the risk of superficial thrombophlebitis extension and recurrence when compared with placebo.[1 20] To date, there are no reliable data comparing fondaparinux with low molecular weight heparin. The absence of evidence prevents firm recommendations regarding the duration of treatment and the long term efficacy of anticoagulation in patients with isolated superficial thrombophlebitis.[1]

Antibiotics

Antibiotics have no role in the treatment of thrombophlebitis, except in clear cases of infection.

Surgical intervention

Whether surgery offers any benefit over pharmacological therapy for initial treatment remains controversial, and it should probably be limited to the small number of cases where superficial thrombophlebitis propagates towards the saphenous junctions despite effective medical therapy. Surgical saphenous ligation with compression is superior to compression alone in preventing superficial thrombophlebitis propagation and thromboembolism. However, surgery is itself associated with venous thromboembolism. The evidence is still lacking for a comparison of surgery and anticoagulants.

With respect to recurrence of superficial thrombophlebitis, one small randomised trial suggests that saphenofemoral disconnection is superior to low molecular weight heparin in reducing the risk of recurrence (3.3% *v* 10%), but with higher risk of thromboembolism,[21] although it is not possible to draw firm conclusions from this small study.

When to refer

Consider referring patients with suspected superficial thrombophlebitis for venous duplex scanning, which can then guide treatment and the need for further specialist intervention.[10]

Vascular surgeon—During the acute phase response, patients with thrombus near the junction may be considered for anticoagulation or surgical intervention. Once the acute phase has passed then referral is useful to help guide elective superficial venous intervention to reduce the risk of recurrence.

Haematologist—All patients with identified coagulation abnormality.

ANSWERS TO ENDGAMES, p 35

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CASE REVIEW

A 56 year old woman with syncope, weakness, and refractory hypotension

1 Panhypopituitarism secondary to radiotherapy.
2 Thyrotrophin is unreliable (although typically low relative to thyroid function) in central hypothyroidism, and triiodothyronine or thyroxine should be monitored instead.
3 Glucocorticoids may uncover partial or compensated diabetes insipidus.

STATISTICAL QUESTION

Placebos and sham treatments

Statements *c* and *d* are true, whereas *a* and *b* are false.

SPOT DIAGNOSIS

A painful hand after a fall

Carpometacarpal dislocation.

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