

Special Study Module in Accident and Emergency Medicine

Axillary / Subclavian Vein Thrombosis

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Venous Thrombosis

A thrombus is a plug or solid mass formed within the heart or any of the peripheral vascular system. Thrombosis is the process of thrombus formation and involves activation of both platelets and the clotting cascade. Thrombi consist of platelets, red blood cells, fibrin strands and moderate numbers of leucocytes. Rudolf Virchow recognized three groups of factors (Virchow's triad) that are likely to promote thrombus formation:

1. *Changes in the intimal surface of the vessel* (Smoking, MI, CHF, immobilization (>3 days), fractures, ulcerative colitis, DVT in past, age, obesity, burns)
2. *Changes in the pattern of blood flow* (major trauma, indwelling catheter, surgery (especially hip or knee surgery), drug abuse, chemotherapy, long plane or car trips)
3. *Changes in the constituents of the blood* (protein C, S deficiency, antithrombin III deficiency, pregnancy, blood type A, heparin thrombocytopenia, lupus anticoagulant, AIDS or medications (oral contraceptives, warfarin))

Once a thrombus is made there are three things that may occur:

1. Lysis
2. Organization and recanalization
3. Embolization

Clearly lysis is the most favourable outcome and is the aim of treatment in a clinical setting. It occurs in fewer than 10% of patients even with anticoagulation. Organization and recanalization usually starts 5-10 days post thrombus formation. Until this process is established fully the thrombus may propagate and/or embolize. Embolization is the most dangerous consequence of thrombosis and can lead to life threatening disease.

Venous thrombosis is divided into superficial and deep vein thrombosis. Deep venous thrombosis can also be subdivided into axillary / subclavian vein thrombosis and deep venous thrombosis of the deep veins of the leg. I intend to focus on axillary / subclavian vein thrombosis.

Superficial phlebothrombosis / thrombophlebitis

Superficial phlebothrombosis and thrombophlebitis commonly involves the saphenous veins and is often associated with varicosities. In thrombophlebitis there is local superficial inflammation of the vein wall with a secondary fibrosis. Thrombophlebitis commonly affects the superficial veins. Clinically there is often a painful, tender cord-like structure which is associated with redness and swelling. Treatment is symptomatic with accompanying rest, elevation of the limb and NSAIDs. Embolization does not occur from superficial veins.

Deep Venous Thrombosis (DVT) of the Legs

Deep venous thrombosis of the legs is common with approximately 600000 cases in the USA annually. Risk factors for DVT of the legs have been well documented in the past few years and all relate to Virchow's triad (see above). DVTs are more common in the elderly and increasing age is a definite risk factor.

Signs and symptoms of DVT of the legs include unilateral oedema (>3 cm is significant), pain or tenderness in leg, warmth, erythema, dilated superficial veins, low grade fever or symptoms and signs of pulmonary embolism. Clinically a DVT can be detected if D-dimer is raised and ultrasonography shows the formation of a thrombus. Ultrasonography is unreliable in detecting calf vein thrombi.

Treatment is anticoagulation and compression stockings. The main aim of treatment is to prevent serious complications such as pulmonary embolism and post-thrombotic syndrome

Axillary / Subclavian Vein Thrombosis (ASVT)

Sir James Paget first described thrombosis in the subclavian veins in 1875. He described that the thrombosis is accompanied by pain and swelling of the affected extremity. In 1884, von Schrötter theorized that this syndrome was due to occlusive thrombosis of the axillary and subclavian veins. Hence, Paget-von Schrötter syndrome was coined to describe axillary / subclavian vein thrombosis (ASVT) with an effort induced cause. Axillary vein thrombosis when secondary to other causes tends to present in older and generally less healthy individuals.

ASVT accounts for approximately 1-2%, of all cases of deep vein thrombosis. Incidence is likely to have risen due to increased use of the subclavian vein for central venous access. In effort induced thrombosis with subclavian vein stenosis, the thrombosis occurs in the dominant arm of the patient in 80% of cases.

Primary ASVT or Paget-von Schrötter syndrome generally has a traumatic pathogenesis but can be due to excessive or repetitive use of the arm(s) resulting in minor trauma to the axillary / subclavian vein. Movements likely to cause minor trauma are hyperabduction and external rotation of the arm or backward and downward rotation of the arm and shoulder. Examples of causative activities include playing cricket, tennis, basketball weight lifting, gymnastics or chopping wood. Trauma is not a prerequisite since 13% to 32% of patients have reported no particular activity before their thrombosis.

Thoracic outlet compression (hypertrophied anterior scalenus muscle, first rib, clavicle or cervical rib, congenital fibromuscular bands) may all predispose patients to having a primary ASVT due to changes in both the vessel wall and in the pattern of blood flow.

Secondary ASVT can be due to:

- Subclavian vein catheterization, especially in cancer patients.
- Malignancies (especially adenocarcinoma)
- Transvenous pacemakers
- Other risk factors – related to Virchow's triad (see above)

In stark contrast to DVT in the legs, Paget-von Schrötter syndrome usually present in young, active and otherwise healthy individuals. There does not seem to be any associated risk in terms of geography or gender. Interestingly, Eskimos of North West Greenland seem to be at lower risk of thrombosis due to alterations in their plasma concentrations of certain lipids, this is thought to be due to specific fish oils in their diet.

Clinical presentation

The clinical presentation of ASVT may include:

- Swelling of the arm, which is usually the first symptom
- Pain or discomfort in the affected arm usually described as a dull ache.
- Discoloration
- Numbness
- Heaviness

On examination:

- Non-pitting oedema
- Superficial veins tend to be prominent
- Anterior chest and shoulder veins become visible
- Pulses are normal
- Arm tends to have a dusky color and mottling
- May have palpable cords in the axilla or proximal arm
- Tenderness may also be present along the brachial or axillary veins

The symptoms and signs of ASVT tend to all occur within 24 hrs of the event in an acute attack.

Investigations

Blood investigations:

- Full blood count
Check for hyperviscosity, platelet numbers
- Coagulation studies
Check prothrombin time (extrinsic system), and activated partial thromboplastin time (intrinsic system). It is important to send for these before anticoagulation is begun so that a clotting disorder is not missed.
- CRP
Checks for any recent systemic inflammation
- Liver function tests.
Checks liver function which is important in production of coagulation factors and blood proteins.
- D-Dimer
If raised indicates the presence of excess fibrinogen degradation products which are indicative of proximal vein thrombosis but are nonspecific.

Duplex ultrasonography

Has a high specificity and relatively low sensitivity when compared to venography. It is preferred and indicated in all suspected cases as it is non-invasive. If this test is negative and clinical suspicion is high then venography should be employed.

Venography of subclavian vein

A cannula is inserted into a peripheral vein of the arm and a contrast injected into the arm. This carries risks of contrast-induced adverse effects.

CT scanning / MRI scanning

Can be used to detect thoracic outlet compression.

Treatment

Immediate treatment with heparin for 4 to 7 days halts clot propagation. Local catheter-directed tissue-Plasminogen Activator (t-PA) in the acute phase is now the most effective modality for achieving thrombolysis and vein patency. This should be followed by anticoagulation with warfarin. Local thrombolysis is highly successful, success rates varying from 69% to 94%. It is most useful in the first 7 days post ASVT but is effective 10 to 14 days post onset of symptoms.

An alternate regimen for treatment of all deep venous thrombi is low molecular weight heparin (LMWH) at a dose of 1.5mg/kg/24 hr subcutaneously. The advantage of LMWH is that it allows patients to be treated as outpatients so lowering costs.

This again should be followed by 10 mg of warfarin orally. Stop heparin dose when International Normalized Ratio is between 2.0-3.0. Warfarin should be continued for 6 weeks if post-op. 6 months if recurrent or no cause can be found or indefinitely if cause is permanent. Analgesia should also be provided for the patient.

If there is thoracic outlet compression or recurrent thrombosis more invasive therapy is indicated with either a balloon angioplasty or surgery. In subclavian catheter associated thrombosis warfarin prophylaxis is recommended e.g. 1mg of coumadin 3 days before catheter placement.

Complications of AVST are:

- Nonfatal pulmonary embolism – 20-36%
- Recurrence of AVST – 14% have history of prior episode
- Stroke – paradoxical embolism
- Chronic venous insufficiency
- Venous gangrene
- Pulmonary hypertension
- Right sided heart failure

Prognosis

Prognosis in patients with AVST, is good with 80% to 90% of patients treated with thrombolysis followed by surgical decompression returned to a long-term asymptomatic state.

Without treatment case fatality rates may be as high as 10%. The AVST syndrome can lead to reduced exercise capacity and may lead to occupational disability and lower quality of life.

In the future there is likely to be an increased number of ASVT cases due to increased use of subclavian catheterization. There is a need here for a randomized control trials to standardize treatments. Furthermore a retrospective study of ASVT post subclavian vein catheterization is needed to give accurate risk assessment of central venous lines.

Conclusions

This case highlights that young healthy individuals can be suddenly affected by disease and for no apparent reason. The patient was treated appropriately. In light of this patient's biochemistry, it would be interesting to see what is causing an elevated haemoglobin and haematocrit level and whether this relates to ASVT.

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