Superficial venous thrombosis

Abstract
Superficial venous thrombosis (SVT) on the left external jugular vein is atypical. We examine this common condition and its treatment modalities which are not yet uniform.

Keywords: superficial venous thrombosis; treatment; jugular

Preface
Venous thromboses, superficial or deep, are the hospital daily practice. The superficial venous thrombosis (SVT) is characterized by a thrombotic coagulation of superficial veins with a minimal inflammatory component. If the attitude for deep vein thrombosis (DVT), regardless of its location, seems well codified, it is not the same for superficial venous thrombosis. These are extremely common and often considered as benign and require poor attention.

Comment
FS, 61 years, with no particular pathological history, consults the emergency room for a left cervical discomfort, without pain, occurred three days earlier. This is accompanied by a locoregional edema. From systematic history, we learn that the patient is G1P1 after a vaginal delivery without incident. She has started oral contraceptive three days earlier. This is accompanied by a locoregional edema.

Clinical examination reveals a palpable venous “cord” in the left cervical region (Figure 1). The rest of the physical examination is common, especially at the lower limbs where legs are supple, painless and with a negative Homans’ sign. Venous doppler ultrasound of the neck shows a thrombosed vein with a large closet, thrombi measuring five millimeters wide and ten millimeters in thickness and occupies the vein’s lumen with a very low residual flow. These are observed in the left jugular, rising above the collarbone and down to the in nominate vein in the direction of the vena cava. The left subclavian vein is also thrombosed. We conclude with a left subclavian vein thrombosis.

These are observed in the left jugular, rising above the collarbone and down to the in nominate vein in the direction of the vena cava. The left subclavian vein is also thrombosed. We conclude with a left external jugular vein thrombosis with a deep vein thrombosis of the left subclavian. The patient, with a weight of 60 kg, will be treated, in absence of renal failure, by nadroparin calcium at the daily dose of 11400 IU anti-Xa (0.6 ml), relayed by vitamin K antagonist for a minimum of three months. Hormone replacement therapy will be stopped and replaced by a treatment with transdermal estrogen. An ultrasound will be performed on the tenth day of treatment to ensure the regression of the clot. No thrombophilia investigation will be realized, given the first thrombotic episode at more than 55 years, in the absence of recurrence and with no family history. Contributing factors of SVT who have led to a secondary DVT have been taking hormone replacement therapy and fall on the left side with a possible contact and trauma of the neck due to the impact.

Discussion
SVT at the upper limb is a common condition after puncture or venous catheterization. In the lower limbs, SVT is mainly localized to the great saphenous vein. Varicose disease is the most common etiology for SVT. The definitive diagnosis of SVT is ultrasound. Nevertheless, we can observe by the patient varicose, subcutaneous in duration sensitive, looking “worm” accompanied by local erythema and painful swelling located. In patients without varicose veins, palpation of a “cord” venous red, hot and painful is to search for.

Predisposing factors (similar to those of DVT) should be given special attention especially when a patient does not carry varicases: thrombophilia (factor V Leiden mutation, deficiency of protein C or S, antithrombin deficiency, other defects prothrombotic, etc), systemic disease (Buerger’s disease, Behcet syndrome, antiphospholipid syndrome, etc), malignancy and trauma direct or indirect. Doppler ultrasound will specify the extent of thrombosis and the presence of deep venous thrombosis associated. Treatment will be initiated by low molecular weight heparin (LMWH) in the curative dose of 100 IU anti-Xa/kg/12 h or 150-200 IU anti-Xa/kg/24 h. The intermediate dose is 100 IU anti-Xa/kg/24 h. It will be delayed later, and for practical reasons, by antagonists of vitamin K targeting an INR between 2 and 3. The treatment recommendations are still unclear. Thus, according to the recommendations of “Thrombosis Guidelines Group of the
Belgian Society on Thrombosis and Haemostasis & the Belgian Working Group on Angiology” a SVT limited to a varicose vein accessory will only be processed locally, until symptoms disappear. SVT undertaking a main vein will be treated with curative doses of LMWH for a period of ten days followed by intermediate doses (called prophylaxis) for a period of twenty days. The SVT associated with DVT is treated as a DVT with curative doses of LMWH with simultaneous introduction of oral anticoagulation for a minimum of three months (except for advanced cancer requiring the maintenance of LMWH). It is necessary to have a local treatment (HIRUDOID cream etc), analgesic/anti-inflammatory drugs (paracetamol/NSAIDs) as well as compression stockings and early mobilization will use for all types of venous thrombosis.1–4

Conclusion

Superficial venous thrombosis is a condition very common in our hospital practice. The diagnostic approach is primarily based on clinical and echographic localization of SVT and this, to answer two specific questions: defining the extension of the thrombus and the presence of deep venous thrombosis associated. Treatment recommendations are specific according to 3 possible categories: SVT limited; SVT entering a main vein; SVT associated with DVT. The cause (varicose veins, trauma) as well as the search for predisposing factors will be important in management.

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Conflict of interest

The author declares no conflict of interest.

References