
Case report

Deep vein thrombosis as the result of extension of a retroperitoneal haematoma in a patient on warfarin therapy: an interesting case report.

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Abstract

A known complication of anticoagulation is bleeding. Cases have been reported of retroperitoneal haematomas in patients on warfarin therapy. This is a unique case of a 75 year old man, who had a retroperitoneal haematoma, which extended to his thigh, and caused deep vein thrombosis. The patient was treated appropriately and adequately, which gave him a favourable outcome.

Key words: deep vein thrombosis, haematoma

Introduction

The Virchow triad, as first formulated (i.e., venous stasis, vessel wall injury, hypercoagulable state), is still the primary mechanism for the development of venous thrombosis.(DVT) The formation, propagation, and dissolution of venous thrombi represent a balance between thrombogenesis and the body's protective mechanisms, specifically the circulating inhibitors of coagulation and the fibrinolytic system.

Coumarins inhibit hepatic synthesis of the Vitamin K–dependent coagulation factors II, VII, IX, and X and the anticoagulant proteins C and S. Typically, Vitamin K is a cofactor in the post-ribosomal synthesis of the clotting factors mentioned above. The Vitamin K–dependent step involves carboxylation of glutamic acid residues and requires regeneration of Vitamin K to its reduced form.

History and treatment

A 75 year old man, presented to the Medical Admissions Unit, with history and symptoms suggestive of left leg cellulitis. He was started on IV antibiotics for the cellulitis. While he was admitted in the ward, it was observed that he had not opened his bowels for 5 days. He was given laxatives, with no favorable result. His bowels were palpable on abdominal examination. An erect abdominal X-ray revealed dilated bowel loops, with faecal impaction. His penis and scrotum were bruised. On surgical review, a diagnosis of a retroperitoneal haematoma with pseudo obstruction was made. His INR was 8.0 on admission. He was on regular warfarin medication for previous two

episodes of pulmonary embolisms. The ultrasound venogram of his left leg demonstrated a deep venous thrombosis of his left common femoral vein which extended proximally into at least the external iliac vein. CT abdomen confirmed the diagnosis of a large haematoma in the left iliopsoas muscle. A flatus tube and nasogastric tube were inserted. Warfarin was stopped and the patient was started on Tinzaparin. He opened his bowels after 3 more days, following regular laxatives and enemas. He was then admitted to our unit for further review and management.

On further questioning, the patient had no previous history of DVT, and no family history of any clotting disorders. It was indeed very unusual to find a patient on regular warfarin to have an INR of 8.0, with a DVT. A closer look at his full blood count had revealed an increased MCV. His liver function tests revealed serum alkaline phosphatase of 132 IU/L, Albumin of 30 g/l and bilirubin of 28mg/l. On further questioning, a history of chronic alcoholism was elicited. He consumed around 26 units of alcohol per week, which was above the recommended normal limit. He also gave past history of arthroscopy of left knee, after which his mobility had decreased due to pain. And since the previous few weeks he had not mobilized as he was in pain. He also had the general feeling of being unwell, while the retroperitoneal haematoma developed. He was asked to discontinue alcohol, with all its ill effects explained, and

was treated with Tinzaparin 20,000iu/ml. He made speedy recovery and with adequate physiotherapy was able to walk.

Discussion

Warfarin is metabolized by hepatic cytochrome P-450 (CYP) isoenzymes predominately to inactive hydroxylated metabolites, which are excreted in the bile. It also is metabolized by reductases to reduced metabolites, which are excreted by the kidneys. Warfarin metabolism may be altered in the presence of hepatic dysfunction or advanced age but is not affected by renal impairment. Drug interactions are numerous and include agents from a variety of pharmaceutical classes, such as antibacterials, antimycobacterials, antifungals, antiarrhythmics, anticonvulsants, antihyperlipidemics, antineoplastics, nonsteroidal anti-inflammatory agents, H₂-receptor antagonists, immunosuppressive agents, and many others. Excessive anticoagulation may also occur because of accidental or intentional overdose.

In this patient, alcohol intake caused inhibition of microsomal enzymes, leading to the increase in warfarin level, while simultaneously due to chronic hepatic failure, his vitamin K dependant factors were not present in adequate amounts, the additive effect which explained the high INR

The main adverse effect of all anticoagulants is haemorrhage. Checking the INR and omitting doses when appropriate is essential; if the anticoagulant

is stopped but not reversed, the INR should be measured 2 – 3 days later to ensure that it is falling.

The following recommendations are based on the result of the INR and whether there is major or minor bleeding; the recommendations apply to patients taking warfarin:

- Major bleeding – Stop warfarin, give phytomenadione (Vitamin K) 5 – 10mgs, by slow IV injection; give prothrombin complex concentrate (Factors II, VII, IX, X) 30 – 50 units/kg or if no concentrate is available fresh frozen plasma 15 ml/kg.
- INR > 8.0, no bleeding or minor bleeding – Stop warfarin, restart when INR < 5.0; if there are other risk factors for bleeding give phytomenadione 500 micrograms by slow IV injection or 5mg by

mouth. Repeat dose of phytomenadione if INR still too high after 24 hours.

Conclusion:

It is of paramount importance to regularly monitor patients on Anticoagulant treatment. What was unique about the case was its presentation. Recognition of anticoagulant induced complications, along with prompt management would be life saving. The current recommendations of the 'British Society of Haematology' does give an insight into management of this problem.

Acknowledgements

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