Deep venous thrombosis in a subject with agenesis of inferior vena cava

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Abstract

Agenesis of inferior vena cava (IVC) is a rare anomaly associated with idiopathic deep venous thrombosis, particularly in young people. We present the case of a 30 year old male, admitted with fever, low back pain irradiating to both thighs and gait difficulty. Abdominal CT revealed agenesis of pre-renal part of IVC as well as retroperitoneal collaterals, associated with deep venous thrombosis. Plasma protein C and S, factor V, anti-thrombin III, anti-phospholipids’ and anti-cardiolipin antibodies were within normal values. He was treated with heparin and antibiotics and symptomatology was resolved within few weeks. Life-long treatment with oral anticoagulants was recommended.

Introduction

The etiologic factors of deep venous thrombosis in young people are frequently associated with congenital defects, congenital coagulation abnormalities, or acquired co-morbidities such as immunologic diseases and cancer. The Virchow's triad: stasis, injury to the vascular endothelium and abnormalities in the blood coagulation, are usually the main cause of the deep venous thrombosis. IVC aberrancy has been reported to occur in 5% of young patients presenting with deep venous thrombosis (1). IVC has 3 segments of different embryologic origin, sourcing during the period between the 6th to 8th week of embryonic development, namely the pre-renal, renal, and post-renal portions of IVC. This is a result of the fusion and partial re-absorption of 3 pairs of vessels that come out from the posterior cardinal veins of the embryo. This complicated process can give rise to anatomic malformations that might impede venous drainage, and thus provoke the development of thrombosis. It is found that 16.2% of patients with iliac vein thrombosis, aging less than 50 years old, had some degree of inferior vena cava malformation (2). The authors didn't find significant differences between sexes, and the first event of thrombosis was generally reported before the fourth decade of life. IVC malformation alone can provoke deep venous thrombosis especially during effort stress, because of inadequate blood flow. In fact, asymptomatic lifelong cases with this malformation exist, suggesting for the presence of other congenital risk or acquired factors (3).

Case Report(s)

A 30 years old male was admitted in the Service of Infectious Diseases, because of fever 39°C, low back pain radiating into scrotal region and thighs, and pain-related gait difficulty. Two weeks before admission he was treated for three days with antibiotics for a dermal infection in the right leg after an insect bite. Ten days prior to hospitalization he felt back pain after a football game and was treated with non-steroid anti-inflammatory drugs for his symptomatology, without any result. The clinical situation became worse, the pain became more intense and three days before hospitalization he became febrile. The patient had no other co-morbidities.

The physical examination at admission showed a deep malaise, tenderness on palpation in the upper part of the legs and redness on the both thighs. The patient had a fever 39°C; upon admission his blood pressure was 100/70 mmHg, with a heart rate of 120/min and a respiratory frequency of 28/min. He had also a count of white blood cells of 16.000 (laboratory normal range less than 10.000 per field). The red cell sedimentation rate was 55 mm for the first hour (normal values 3-15 mm/1st hour). His C-reactive protein was 92 mg/l (normal range < 10 mg/L).

Plasma protein C and S, factor V, anti-thrombin III, anti-phospholipids’ and anti-cardiolipin antibodies were normal. The microbiological and serological examinations were all negative.

An abdominal CT showed agenesis of the pre-renal segment of the IVC (except for its distal 1 cm portion) and developed retroperitoneal collaterals (Figure 1). Meanwhile, thrombosis was seen in this cava distal segment and on both sides in common iliac vein, external iliac veins, femoral veins and deep veins too. No lesions in other abdominal organs or enlargement of lymph nodes were seen. Neurologic assessment for the low back pain and sciatica syndrome didn't reveal any neurological problem.
The patient was consulted with the vascular surgeon and treatment was started with heparin infusion for a week and later on with an oral anticoagulant (acenocoumarin), antibiotics (Ciprofloxacin 200 mg twice daily intravenously and Amikacin 0.5 grams twice daily intravenously) for a probable septic condition. Ten days from the start of this therapy, the fever subsided. Also, three weeks later, the lower limb swelling resolved. Life-long treatment with oral anticoagulant therapy and regular consultations were recommended.

Discussion

The agenesis of IVC is a rare anomaly that is associated with deep venous thrombosis. This anomaly has been reported to occur in 5% of young patients presenting with deep venous thrombosis and its prevalence is 0.3% - 0.6% in the general population (1, 4).

The case we presented was of a male 30 years old with fever, back pain and difficulty in walking for more than a week before hospitalization. The patient complaints started few hours after a football game. The physical exertion as a precipitating factor, can explain the mechanism of the deep venous thrombosis in a patient with vena cava aberrancy, because the pre-existing collaterals may be insufficient, while blood flow is immediately increased. The collaterals may be developed before thrombosis, because of an IVC anomaly or partial absence of the latter (5).

Our patient two weeks before hospitalization was treated for a dermal infection after an insect bite. Sepsis could be a dramatic example of the link between inflammation and thrombosis (6). Toxins stimulate the production of tissue factors and also augment the production of plasminogen activator inhibitor which will inhibit the fibrinolysis, and thus predispose to thrombosis. The patient was treated with anti-inflammatory drugs for his back pain, but the clinical situation became more serious. Sciatica had been described in numerous publications as a rare cause of neural and low-back pain and might be the first clinical manifestation of this rare venous anomaly, as in our present case (7).

The laboratory examinations showed positive inflammatory tests, but no specific data were found in the microbiological and serological tests to explain the origin of the fever. The abdominal CT scan showed pre-renal IVC agenesis, collateral venous circulation and also venous thrombosis. The treatment started with antibiotics for a probable septic condition, heparin for a week and later on with oral anticoagulants. An aberrant IVC associated with deep venous thrombosis, without other predisposing factors, needs a treatment with anticoagulants.

The IVC agenesis is considered as surgically uncorrectable. Because of the permanent risk for venous stasis and thrombosis, the anticoagulant treatment should be a lifelongone, and the patient must be under strict supervision from a vascular surgeon (5, 8). On the other hand, elastic stockings are as well advised from authors, who do not conclude unanimously regarding the precise duration of the anticoagulation (9).

As a conclusion, agenesis of IVC can provoke deep venous thrombosis. Eventually, risk factors for the deep thrombosis seem to be the excessive physical activity, and the septic condition.

References

Illustrations

Illustration 1

Coronal reformatted CT image showing agenesis of pre-renal inferior vena cava (arrow).
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