Extensive portal vein thrombosis related to abdominal trauma

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SUMMARY

Abdominal trauma is a classic but very rare cause of portal vein thrombosis. We report the case of a patient with portal vein thrombosis and cavernoma associated with portal hypertension. Anamnesis identified a serious thoraco-abdominal trauma related to a bicycle accident 6 months before. Biological screening identified an inherited heterozygous G20210A factor II gene mutation which supports a recent notion that portal vein thrombosis most often occurs when both local and systemic aetiological factors are combined.

RÉSUMÉ

Thrombose portale extensive après un traumatisme abdominal

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Un traumatisme abdominal est une cause classique mais rare de thrombose portale. Nous rapportons l’observation d’un malade présentant une thrombose avec cavernome portée associée à une hypertension portale mise en évidence lors de la consultation d’un syndrome douloureux abdominal. L’anamnèse identifiait la notion d’un violent accident de la voie publique avec traumatisme thoraco-abdominal 6 mois auparavant. Le bilan de thrombophilie mettait en évidence une mutation hétérozygote G20210A dans le gène du facteur II. Cette observation originale conforte le concept de la conjonction de facteurs locaux et généraux dans la genèse de la thrombose porte.

Introduction

Abdominal trauma is a classic but very rare cause of portal vein thrombosis. We report the case of a patient with portal vein thrombosis and cavernoma associated with portal hypertension. Anamnesis identified a serious thoraco-abdominal trauma related to a bicycle accident 6 months before. Biological screening identified an inherited heterozygous G20210A factor II gene mutation which supports a recent notion that portal vein thrombosis most often occurs when both local and systemic aetiological factors are combined [1].

Case report

In January 2002, a 41-year-old man was referred to our institution for acute abdominal pain. There was no tobacco or alcohol abuse. The patient did not have any past medical history except for a bicycle accident with thoraco-abdominal trauma six months earlier in July 2001. That fall was complicated by right flail chest, right pneumothorax, right acromio-clavicular dislocation and abdominal contusion. Emergency abdominal ultrasonography was normal. The patient required draining of the pneumothorax and one week of hospitalisation.

Upon admission, the patient complained of right upper quadrant pain with general malaise. He was apyretic and haemodynamics were normal. Laboratory studies gave the following results: normal blood cell counts, prothrombin index 92% (N > 80), factor II 142% (N < 120%), aspartate aminotransferase 33 IU/L (N < 37), alanine aminotransferase 69 IU/L (N < 41), serum alkaline phosphatase 314 IU/L (N < 270), gamma glutamyltranspeptidase 109 IU/L (N < 50) and total serum bilirubin 7 mmol/L (N < 12). Lipase and C-reactive protein were normal. Serum tests for hepatitis B and hepatitis C virus were all negative. Abdominal CT scan showed thrombosis of the portal system extending from the spleno-mesenteric confluence to the portal vein bifurcation reaching the right and left branches (figure 1). This thrombus appeared hypodense and was associated with the development of a portal cavernoma (figure 2). No other intra-abdominal lesions were identified. Endoscopic examination of the upper gastrointestinal tract showed grade II oesophageal varices with portal hypertensive gastropathy. Exhaustive search of an underlying thrombophilic state was performed to explain the occurrence of extensive portal thrombosis in our patient. Specific tests for paroxysmal nocturnal haemoglobinuria, lupus anticoagulant and anti-phospholipid antibodies, deficiency in protein C or S, antithrombin III or plasminogen, factor V Leiden and MTHFR gene mutations, were all negative. The serum homocysteine concentration was also normal. In vitro culture of bone marrow progenitor cells without the addition of erythropoietin did not show spontaneous formation of erythroid colonies. Finally, a heterozygous G20210A factor II gene mutation was diagnosed.

Coumarin anticoagulants were introduced (Fluindione) after a hypercoagulable state was performed, 20 mg a day to obtain an INR between 2 and 3. Beta-adrenergic-antagonists (propanolol, 20 mg, three times a day) were begun to prevent gastrointestinal bleeding. The clinical course was favourable: abdominal pain and hepatic biological abnormalities disappeared within a few days. An abdominal CT scan follow-up six months later showed stabilization of the thrombus. In light of the underlying thrombophilic state of the patient, oral anticoagulant therapy was continued and the inherited thrombogenic disorder was searched in his family. The patient was asymptomatic when last seen in August 2004.

Discussion

We report a case of a 41-year-old man who presented with extensive thrombosis of the portal system after a violent thoraco-abdominal trauma that led to identification of an underlying inherited prothrombotic disorder (heterozygous G20210A factor II gene mutation). To our knowledge, no association

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Between portal vein thrombosis, heterozygous G20210A prothrombin gene mutation and abdominal trauma has been previously published.

According to a recent hypothesis, thrombosis of the portal system most often occurs when a local and a systemic risk factor are associated [1, 2]. The main local factors include disorders leading to a decrease in portal flow, such as cirrhosis, hepatobiliary malignancies and inflammations of the abdomen, or endothelial lesions which initiate thrombus formation, such as trauma or surgical injury [3]. Systemic risk factors mainly include hypercoagulability states such as hereditary and acquired coagulation disorders, haematologic diseases including occult myeloproliferative syndrome, and oral contraceptive use [1].

In this case, we suggest that the occurrence of extensive portal vein thrombosis was related to the external trauma, which was the local precipitating factor. Indeed, as previously suggested, trauma may shear the portal vein and induce an endothelial lesion which initiates thrombus formation. The underlying thrombogenic state promotes progressive thrombus extension [4]. The severity of trauma and chronological features of our case support this finding. Indeed, thrombosis formation followed the trauma, as shown by the normal ultrasound performed a few hours after the bicycle accident. Thrombosis developed early after the accident because in the abdominal CT scan performed six months later portal vein thrombosis appeared to have been present for a certain time (hypodense character of the thrombus and development of a portal cavernoma). Extension of the thrombosis could probably explain the recent acute abdominal pain. Another argument supporting the post-traumatic origin of this portal system thrombosis is the absence of other apparent local causes, such as cirrhosis, abdominal neoplasia, or inflammatory and infectious abdominal diseases.

The post-traumatic origin for portal vein thrombosis in this case report represents exceptional pathological circumstances, which are poorly documented. To our knowledge, nine cases have been reported in the literature [5-11] and only five have been described in detail [5-7, 9, 11]. The rarity of this cause is explained by the rarity of lesions to the portal system in abdominal traumas. In a study including 2000 patients who experienced abdominal trauma requiring surgical treatment, portal vein injury was diagnosed in only 1% of cases (22 patients). In addition, in 20 of the 22 observed cases, abdominal trauma was penetrating and then, only two cases of portal vein injury were associated with external abdominal trauma [12]. Among the few previously described cases of post-traumatic portal vein thrombosis, two cases were revealed by abdominal pain, one case by hepatic biological abnormalities, and two cases by hematemesis (portal hypertension). The rarity of the post-traumatic origin requires exhaustive aetiological investigation. Among these five cases, proof of the trauma in the occurrence of thrombosis is only well established in two cases [5, 11]. In the other cases, biological evaluations did not exclude occult myeloproliferative syndrome. An underlying prothrombotic systemic state was found in the present case, in the form of a heterozygous G20210A factor II gene mutation. Factor II (prothrombin) is the precursor of the serine protease thrombin, a key enzyme in the hemostatic and thrombotic processes [13]. The presence of the G20210A factor II gene mutation has been found in 2.29% in controls and 8.01% in patients with confirmed venous thrombosis. Carriers of this hereditary abnormality have about a 4-fold increased risk of venous thrombosis and a 1.4-fold increased risk of portal vein thrombosis [14, 15]. This risk of portal vein thrombosis remains relatively low compared to other inherited thrombophilic factors such as factor V Leiden mutation or hereditary protein C deficiency (odds ratios were respectively 2.7 and 4.6). In addition, factor II gene mutation is associated with higher plasma prothrombin levels as in our case (factor II 142%, normal values: 60-120%). In two thirds of cases, this prothrombogenic abnormality is isolated.

An association between local and systemic aetiological factors of portal vein thrombosis has been found in about one fourth of patients [15]. When a local risk factor is involved, the search for associated prothrombotic systemic disorders is positive in 70% of cases [4]. This case report shows that an underlying prothrombotic disorder should be looked for in patients with portal vein thrombosis, even in those with an apparently obvious cause.

β-adrenergic blocking agents were used in our patient for the prevention of bleeding of ruptured oesophageal varices. This preventive treatment, which has been validated in the case of cirrhotic portal hypertension, might also be beneficial in the treatment of portal hypertension secondary to portal vein thrombosis [16, 17]. For curative and preventive management of portal vein thrombosis, the patient began coumadin anticoagulants which should help avoid extension of the disease without increasing the risk or severity of gastrointestinal bleeding, as long as prophylactic treatment of oesophageal variceal rupture by β-blockers or endoscopic therapy is simultaneously begun [16]. As previously recommended, anticoagulant therapy was given for 6 months and is being continued because of the underlying thrombophilia [18].
REFERENCES