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.

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 stable. Physical examination was normal. Body mass index was at 27 kg/m². 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 antiphospholipid 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 anti-coagulants were introduced (Rinidone) after a hypercoagulable state search was performed, 20 mg a day to obtain an INR between 2 and 3. Beta-adrenergic-antagonists (propranolol, 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 anti-coagulant therapy was continued and the inherited throbogenic 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...
between portal vein thrombosis, heterozygous G20210A pro-
thrombin gene mutation and abdominal trauma has been pre-
viously 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 disor-
ders leading to a decrease in portal flow, such as cirrhosis, hepatobi-
lary malignancies and inflammations of the abdomen, or endo-
thelial lesions which initiate thrombus formation, such as
trauma or surgical injury [3]. Systemic risk factors mainly include
hypercoagulability states such as hereditary and acquired coag-
ulation disorders, haematologic diseases including occult myelo-
proliferative syndrome, and oral contraceptive use [1].

In this case, we suggest that the occurrence of extensive por-
tal vein thrombosis was related to the external trauma, which
was the local precipitating factor. Indeed, as previously sug-
gested, trauma may shear the portal vein and induce an
endothelial lesion which initiates thrombus formation. The under-
lying thrombogenic state promotes progressive thrombus exten-
sion [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 inflam-
matory 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 abdo-
nal 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 addi-
tion, 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 throm-
basis, 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 etiological 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, biologi-
cal evaluations did not exclude occult myeloproliferative syn-
drome. An underlying prothrombotic systemic state was found in
the present case, in the form of a heterozygous G20210A factor II
genotype. 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 defi-
ciency (odds ratios were respectively 2.7 and 4.6). In addition,
factor II gene mutation is associated with higher plasma pro-
thrombin levels as in our case (factor II 142%, normal values:
60-120%). In two thirds of cases, this prothrombogenic abnor-
mality is isolated.

An association between local and systemic aetiologi-
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 pro-
thrombotic 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 cir-
rhotic portal hypertension, might also be beneficial in the treat-
ment 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
is being continued because of the underlying thrombophilia
[18].
REFERENCES