CASE REPORT

Portal vein thrombosis Uncommon clinical picture

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Abstract

The authors described a case of the patient of the idiopatic group of PVT. Due to only a few symptoms, the diagnosis was established late. This was confirmed by the fact, that the patient was not hospitalized. So it was an atypical case of chronic PVT. Portal vein thrombosis belongs to rare clinical conditions. Early diagnosis based on modern technique is possible only if we keep this condition in mind *(Tab. 1, Fig. 1, Ref. 23)*.

Key words: portal vein thrombosis, hypertension, CT examination.

I have seen this clinical picture at the end of my clinical praxis. I believe it is a rare condition. While studying available literature, several related studies and case reports were found. We decide to review the topic and publish this case.

Anatomical notes

Portal vein collects blood from single abdominal organs (stomach, lien, small intestine and large intestine). Blood in portal vein is not so low in oxygen as other veins. This fact may explain why hepatic artery is so thin compared to the weight of liver. Portal vein is 6–8 cm long with a diameter about 18 mm.

Portal vein begins by confluence of three veins: splenic vein, superior mesenteric vein and inferior mesenteric vein at the back of pancreas. Portal vein is divided into left and right branch. Right branch is shorter and thicker, dedicated to right hepatic lobe. Left branch is longer and thinner, dedicated to left hepatic lobe, quadrate lobe and caudate lobe. Left branch is connected with chorda ductus venosi as a residue after embryonical umbilical vein. Blood pressure inside portal vein is estimated to be 10–15 mmHg (2).

Theoretical background

Etiology of portal vein thrombosis (PVT) is very variable (13). The main cause is a pre-sinusoidal portal hypertension associated with three mechanisms (18, 21, 22).

1) "Intrinsic-factor" stands for pathological changes in early or late cirrhosis or in tumors of liver and gallbladder.

2) "Extrinsic-factor" stands for mechanical obstruction in tumors of surrounding organs, lymphadenopathy and in inflammatory processes in abdominal cavity (pancreatitis, cholecystitis, diverticulitis, appendicitis etc.).

3) "Spontaneous thrombosis" stands for a thrombosis development without mechanical causes. It includes all haematological conditions considered as risk factors (Tab. 1) (22).

According to literature data, the first group of PVT causes is responsible for 50 % of all cases, the second group for 25–30 % and the third group for only 10 % of cases. Combinations of cases are possible and frequently published.

In pathogenesis of PVT, one rare group of causes is "idiopathic", responsible for only 8–15 % in literature (20). We were interested in this group of causes, as it was our case.

Case report

Patient, a 71-year old retiree, is living in Bratislava. His family history is without singularities. Patient suffers from hypertension about 25 years, regularly takes medicaments. In 1986 he has suffered from right bronchopneumonia, in 1987 from cholecystectomy due to gall stones, he has never been icteric. He has experienced frequent headaches, taking painkillers. The patient

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Tab. 1. Prevalence of etiological factors simultaneously investigated in 36 patients with portal vein thrombosis (adapted to 17).

Etiological factor	%	95% confidence interval
Primary myeloproliferative disorder	22	9 – 36
Prothrombotic coagulation disorder	42	26 – 58
Primary myeloproliferative disorder plus prothrombotic coagulation disorder	8	0.7 – 17
Specific coagulation disorder		
Antiphospholipid syndrome	4	0.8 – 21
G1691 factor V gene mutation	3	0 – 8
G20210A factor II gene mutation	14	3 – 25
C677T MTHFR gene mutation	11	0.8 – 21
Protein S deficiency	30	11 – 49
Protein C deficiency	0	–
Antithrombin deficiency	4	0 – 8

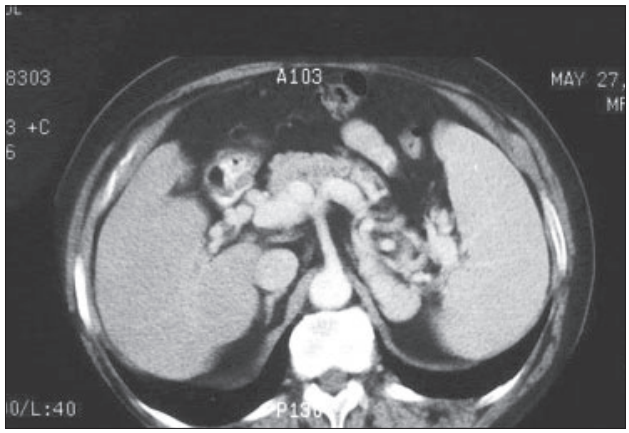

Fig. 1. CT scan of the portal vein thrombosis.

almost daily works in the garden, consumes alcohol only occasionally.

In March 2003, while working in the garden, the patient experienced a pain in lower part of right chest. He had no fever, nor cough. General practitioner has suggested intercostals neuritis and prescribed painkillers. His clinical status was not improved in 3 days; he was feeling weary, weak, without appetite. Surgical examination has excluded an acute abdomen. Neurological examination was negative – patient was taking Voltaren and myorelaxing agents with mild improvement. Beginning May, after an ultrasound examination, a CT was recommended.

CT-examination (27.5.2003)

Caudate lobe is enlarged, bile ducts are not dilated, status after cholecystectomy, pancreas is not changed, lien is enlarged with multiple calcifications. In portal vein area and along pancreas dilated vessels are seen, like in area of right and left gastric vein. Portal vein is not visible, lien vein is also dilated (Fig. 1).

Summary: Status after portal vein thrombosis, marked collaterals, enlarged caudate lobe, calcifications inside lien.

After this examination a complete haematologic examination including sternal punction was performed. Beside mild anemia, the result was negative. Patient refused hospitalization and anticoagulation therapy. Patient was administered B-12 vitamin inj. 20 times i.m. and Legalon tablets. His clinical status was gradually improving, pain dissolved, appetite was improved and currently the patient has no problems.

Discussion

The biggest group called “Intrinsic-factor” (50 %) is clear enough in both pathogenesis and diagnosis. Gomez et al have found increased levels of phospholipids antibodies in 30 patients with liver cirrhosis as a possible cause of PVT (11). In contrast, Mangia and company have serologically proved in 219 patients with liver cirrhosis that hypothesis on deep thrombosis based on congenital or acquired risk factors in non-tumor PVT has no rational background is (16).

Second group “Extrinsic-factor” (25–30 %) is frequently published in literature. Tumor of pancreas can occasionally cause PVT (23). But most frequently, there are inflammatory processes within an abdominal cavity, e.g. cholecystitis (15), abdominal abscesses: out of 197 patients 7 cases of PVT (3), and diverticulitis (9). In literature, one patient with M. Crohn and following PVT was reported (19) and also ona patient with abdominal TBC (5).

Even in childhood it is possible to develop a TPV. Flores and company have reported statistical data in 158 children (10).

Pure haematological causes of PVT are rare. We have found one case in literature, so called myeloproliferative condition, and also one “prothrombic-defect”, with genetic background (13). During pregnancy, one case was reported as “hypercoagulation status during pregnancy” (14).

In a subgroup of idiopatical causes of PVT, a wide discussion is found in literature. In recent years, cases of PVT after prolonged laparoscopic interventions were reported (1, 4). Hassn and company have reported a 10 % PVT after splenectomy. One case of PVT was reported in Budd-Chiari syndrome (8, 17).

Our patient belongs to the idiopatic group of PVT. Due to only a few symptoms, the diagnosis was established late. This was confirmed by the fact, that the patient was not hospitalized. So it was an typical case of chronic PVT. Condat distinguishes acute and chronic course of this condition (6). Patient is taking Anopyrin 100 mg daily. Follow up in one year did not reveal any worsening of his clinical status, patient is doing well, with no problems. The cause of PVT is only hypothetical in this case. It cannot be excluded that the history of cholecystitis and cholelithiasis have lead to mild impairment of liver parenchyma in spite of negative liver tests.

Portal vein thrombosis belongs to rare clinical conditions. Early diagnosis based on modern technique is possible only if we keep this condition in mind.

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