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Acute Portal Vein Thrombosis a Case Report

Mountassir Moujahid[1, *], Hicham Iraqui[1], Mohamed Rhari[2], Khalid Chekoura[3], Issam Serghini[3], Moulay Hassan Tahiri[1]

[1]Departement of Surgery, University of Medicine and Pharmacy of Rabat, 5th Military Hospital, Guelmim, Morocco
[2]Departement of Radiology, University of Medicine and Pharmacy of Rabat, 5th Military Hospital, Guelmim, Morocco
[3]Departement of Anesthesia Resuscitation, University of Medicine and Pharmacy of Rabat, 5th Military Hospital, Guelmim, Morocco

Email address

m.moujahid@gmx.fr (M. Moujahid)

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Abstract

The portal thrombosis vein is the most frequent cause of not cirrhotic high blood pressure portal. Its can be asymptomatic of fortuitous discovery or on the occasion of an abdominal pain during the acute phase or after appearance of a collateral venous circulation and formation of the portal cavernoma. The clinical diagnosis of portal thrombosis is difficult; it was made easy with the not invasive medical imaging. The spontaneous evolution is grave. The treatment is especially medical basing on anticoagulants allowing mostly a repermeabilisation of the thrombosis vessel. We report a case of acute portal thrombosis collaged in the 5th military hospital.

Keywords

Portal Thrombosis, Portal Vein, Portal Cavernoma, Diagnostic and Treatment, Anticoagulant Therapy

1. Introduction

It is a thrombosis of the portal vein situated between the hepatic hile and the confluence lower mesenteric vein and splenic vein. The clinical diagnosis of portal thrombosis is difficult, it rests essentially on the medical imaging. The spontaneous evolution is sometimes mortal .The treatment is especially medical basing itself on anticoagulants allowing most of the time a repermeabilisation of the thrombosis vessel.

2. Observation

34-year-old patient without histories admitted by means of emergencies in a symptom) of acute abdominal pain with vomitings evolving in a feverish context à38°C and this for 24 hours . The abdominal examination showed a pain of the right hypochondria and epigastralgic with scapular and dorsal irradiation, there has no confusion of transit. The rest of the somatic examination is without any peculiarity. A realized biological balance sheet showed a hyperleuKocytosis in 15000 element / mm3 and an inflammatory syndrome with CRP at 65mg / l. The hepatic balance sheet was normal.

An abdominal scanner with injection of the contrast agent showed a thrombosis of the trunk carry and of his right intra hepatic branches, a permeability of the superiors and subordinates mesenteric venous branches, veins knew hepatic and of the lower vena cava (figure 1 and 2). A medical treatment established by a typical anticoagulant of low molecular weight balanced by dosages repeated by the activated cephalin (TCA) aiming at a "patient" report TCA / TCA "witness" between 1,8 and 2,5, a resting of the digestive tract with the pose of a naso gastric sonde, a parenteral nutrition, and a broad-spectrum antibiotic treatment adapted afterward by the antibiogrammes of the realized hemocultures. We completed our biological balance sheet by dosage of the protein C, and of the anti thrombine III as well as by the search for factor V Leiden and for transformation of the factor II of the coagulation. The balance sheet was everything made normal. No thrombectomy or thrombolyse were realized. The relay by ant ivitamin K was established 20 days later. This treatment will be extended by duration of six months in the absence of affection thrombotique pro documented, of personal histories of deep venous thrombosis,

intestinal infarct or familiy history of deep venous thrombosis recurrence. The evolution was favorable under treatment, no sign of second offense on a backward movement of two years.

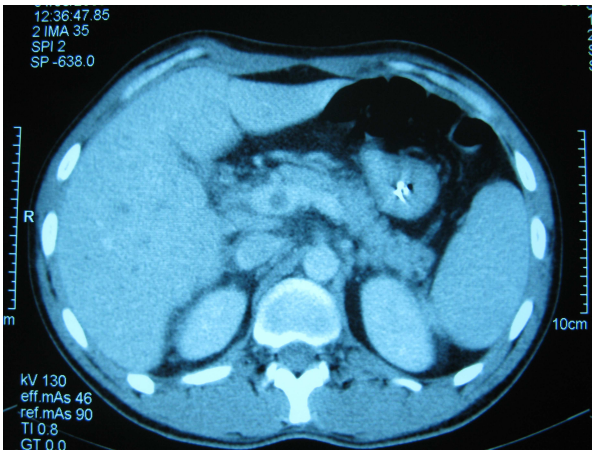

Figure 1. *Abdominal scan with injection showing a portal vein thrombosis*

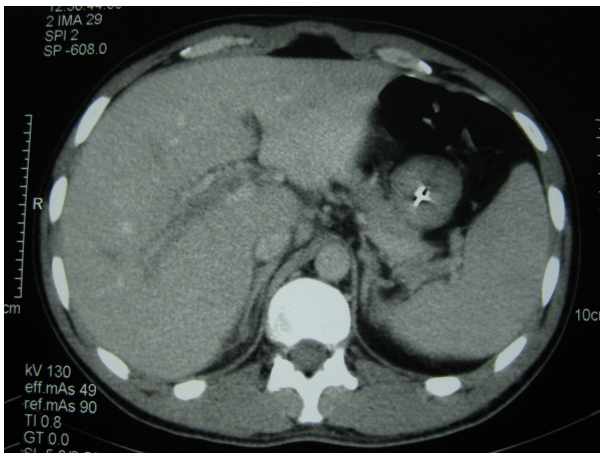

Figure 2. *Abdominal scan showing a portal trunk thrombosis and right intra hepatic brunches*

3. Discussion

The thrombosis of the portal vein (TVP) can be separated in two entities: the acute thrombosis and the thrombosis chronicles diagnosed at a late stage after the arisen of the portal cavernoma (1). We are going to limit our clarification to the acute portal thrombosis. The thrombosis of the trunk carries can extend downstream in the right and left branches and in the segmentary hepatic branches intra or upstream in the spleen vein in the superior or subordinate mesenteric vein. She can finally correspond to the extension downstream to a mesenteric or splenic thrombosis. The occlusion can be complete or partial (2).

The acute portal thrombosis can be due to a sterile thrombus cruorique, or to an infected thrombus, responsible for the presence of material purulent in the portal vein (3). The consequences of the thrombosis of the portal vein have no same gravity downstream and upstream to the thrombus:

- Upstream to the thrombus, there is not much effect on the bowel as long as the mesenteric venous arches remain permeable these arches indeed work as a collateral circulation draining the intestinal blood flow towards the neighboring territories not thrombosis. The ischaemia results from the extension of the thrombus towards mesenteric veins and the venous arches (4). When she goes on beyond several days, an intestinal infarct can arise. In 20 in 50 % of the cases, the intestinal infarct is responsible for the death because of a peritonitis and of a multivisceral failure. The intestinal resection following a venous mesenterico-portal thrombosis is one of talk of the short syndrome of hail (5).
- Downstream to the thrombosis of the portal vein, the hepatic consequences are little marked. Clinically, the signs of hepatic infringement are absent or passing (6). The good hepatic tolerance in the interruption of the portal flow is connected to two compensatory mechanisms, the immediate vasodilatation of the hepatic artery in answer to a decrease of the venous portal flow , and the fast appearance of collateral veins Portal, visible a few days after the thrombosis. These collateral veins develop in the periphery or within the structures neighboring to the portion blocked by the portal vein: principal biliary way, gall-bladder, pancreas, gastric antre, duodenum. When the obstruction of the trunk of the portal vein persists, the collateral veins gradually increase in volume and establish a portal cavernoma (7).

The circumstances of discovery of the acute thrombosis of the portal vein are usually abdominal pains, most of the time in posterior irradiations, continuous on several days, very intense. A high fever can be present, even in the absence of infection. The physical examination is normal, the belly is supple, contrasting with the intensity of the pain. There is sometimes a passing and moderate increase of transaminases, but most of the time the hepatic balance sheet is normal or almost normal. The clinical symptom does not allow to evoke straightaway the thrombosis of the portal vein but the characteristics of the pain incite to practice quickly an examination of abdominal imaging which allows to carry the diagnosis (8).

When the diagnosis or the treatment are late, symptom of intestinal ischaemia can appear, with intense abdominal pains and ileus, or on the contrary diarrhea (1, 2). An intestinal ischemic necrosis must be suspected in front of rectorragia, abdominal defense, peritoneal effusion, metabolic acidosis, renal or respiratory insufficiency (2) .In case of septic phlebitis, the portal thrombosis complicates an intra-abdominal abscess and there is then a high fever and hepatic pains. Hemocultures is generally positive most of the time to Bacteroïdes species (9). The symptoms of the portal thrombosis become confused with those of the intra-abdominal source of the infection.

The immediate Complications of the acute portal thrombosis swallow it of the portal vein are small at the hepatic level. Upstream, there is a risk of mesenteric, spleen,

and epiploic venous infarct (10).

The late complications are represented by the portal cavernoma if a repermeabilisation was not obtained, The digestive bleedings by portal sub-hepatic high blood pressure and the compression of the biliary ways by the veins of the cavernoma (11).

The not invasive imaging techniques allow making the diagnosis of acute thrombosis of the portal vein, to specify the extension of the thrombosis, to distinguish between a old and recent thrombosis and to look for a local cause. The echograph coupled with the Doppler asserts the diagnosis when is highlighted the direct image of the thrombus material echogenic intraluminal (12) .In more than 85 % of the cases, allows a good exploration of all the vessels splanchnic, including the superior mesenteric vein (13,14). The main difficulties concern the visualization of the superior mesenteric vein and the evaluation of the flow in the portal vein. The scanner without injection of contrast can show a thrombus dating less than 10 days under the shape of an image spontaneously hyperdense intra luminal (15). The angio scanner and the angio-MRI can assert the diagnosis portal time, by highlighting the absence of raising of thrombosis vessels, and especially the direct image of the thrombus (hypo density encircled by the contrast agent) (16). The hepatic vascularization seems stressed in the arterial time (because of a compensatory hepatic arterialisation of the portal deperfusion) and decreased in portal time in the portal blocked territory. In case of intestinal venous ischaemia, the scanning shows obvious fact a thickening and an aspect in rosette of the reached digestive handles. The cause of the thrombosis of the portal vein is often multi factorial: a local cause and an affection prothrombotic or a combination of affection. A local cause is highlighted in 30 % of the cases and the general cause in 70 % of the cases.

The causes of obstruction of the portal vein are variables. Among the local factors, the infectious and the cancerous tumors intra abdominal are most often incriminated. The appendicitis, the diverticulitis, the inflammatory diseases of the bowel and the pancreatitis can be responsible for septic or not portal thrombosis (17).

A surgical trauma of the portal vein or its branches can also be the cause of TVP, in particular at the sick reached by portal high blood pressure (in the fall of a splenectomy or a shunt Porto-systemique) (18). In the absence of portal high blood pressure, the splenectomy complicates of TVP essentially in case of hemolytic anemia or of myeloproliférатif syndrom, but much more rarely in case of lymphoma or of spleen trauma (19). Abdominal neoplastic hurts not invading directly the veins of the portal system can be the cause of cruoric thrombosis of the portal vein, as the cancer of the colon. The intra-abdominal adenopathy responsible for TVP is most of the time of tubercular origin. A prothrombotic affection acquired or innate is highlighted at the majority of the sick reached by TVP, that a local factor is present or not (20).

The role of the oral contraception and the pregnancy is less clear than for the venous thrombosis of the other sites. However, it seems reasonable of to indicate the oestrogen-progesterone contraception to the sick infringements of TVP. The only progestatif being associated to a risk of much lower thrombosis, their use could be an alternative. Often, the TVP is the first appearance of a myeloproliferatif syndrom at sick young people.

The osteo-medullary biopsy and the search for spontaneous shoot of erythroblastic colonies allows to assert the diagnosis of myeloproliferatif syndrom to 30 % of the sick reached by TVP (21).

The transformation V617F of the recently described tyrosine kinase JAK2, a specific marker of the myeloproliferatifs syndroms, can be easily diagnosed thanks to a sequencing of nucleic acids or by PCR (22). Prevalence of the transformation V617F JAK2 at the sick reached by TVP was not studied yet. An association of several prothrombotic affections is not rare at the sick reached by TVP. So, at these sick it is reasonable to look for systematically the affections the most frequent prothrombotic as the deficits in protein C and S, in antithrombine III, the factor V Leiden, the transformation of the gene of the prothrombine, the syndrom of antiphospholipides and the myeloproliferatifs syndroms (23). Indeed, these diagnoses have important consequences for the family screening and the coverage of the sick

At the stage of recent thrombosis, a premature anticoagulation is recommended. Indeed, the anticoagulating treatment allows the repermeabilisation of thrombosis vessels to 75 % of the patients while the spontaneous recanalisation seems rare; furthermore, he(it) also warns the arisen of an intestinal infarct (1,3,24). The repermeabilisation can be progressive and be complete only after 4 in 6 weeks of treatment. The main predictive factor of recanalisation is the initial extension of the thrombosis (25). However, a partial recanalisation can be even obtained in case of extensive thrombosis. The duration recommended by the treatment anticoagulating in case of recent thrombosis of the portal vein is of at least 6 months, by analogy with the other venous thrombosis. There are no data allowing recommending an extension of the treatment anticoagulating beyond 6 months. However, as in case of old thrombosis (26), it adorned us useful to propose a treatment anticoagulating in the long term, in case of affection documented prothrombotic, of personal histories of deep venous thrombosis, intestinal infarct or abdominal pain suggestive of intestinal ischaemia, or familiy history of deep venous thrombosis.

On the contrary, the treatment anticoagulating in the long term is prothrombotic or of extensive thrombosis, especially can be not necessary in the absence of argument in favor of an affection when a local factor which was able to be deleted was clearly highlighted, or when there is a greater risk of bleeding because of an advanced age or of an associated morbid condition. So the decision to stop arrest or to continue the anticoagulating treatment must be individually taken.

The initial anticoagulating treatment is the heparin because of its quick action. The heparins of low molecular weight is

generally preferred because of their lesser risk of bleeding or allergic thrombopenie. A relay by antagonist of the vitamin k can be realized as soon as the invasive examinations are ended. The objective of the treatment is to obtain an INR between 2 and 3 (27).

The arguments in favor of a repermeabilisation are clinical (disappearance of the abdominal pains in one-two weeks, even sometimes in 2 in 3 days, in the absence of intestinal infarct), and ultrasound. The inflammatory syndrom quickly declines.

The indication of an aggressive treatment of the acute portal thrombosis remain little clear. The treatments which were proposed include agents' administration thrombolytic by systematic way or by the mesenteric artery, or by catheterization of the portal vein by trans jugular or percutaneous way. The implementation of a TIPS to maintain the flow in the portal vein was also proposed. There is no datum allowing specifying the report profit risks of these invasive procedures compared with the simple anticoagulating treatment. However, it seems that the thrombolyse in situ often allows a recanalisation, sometimes after failure of the anticoagulating treatment, but is associated with a high rate of grave hemorrhagic complications (28).

The surgical thrombectomy is not indicated except the case or a laparotomy was made necessary because of the suspicion of an intestinal infarct. She must be associated with an anticoagulating treatment to prevent the thrombotic second recurrence (29).

The short and long-term forecast of the sick treaties prematurely by anticoagulants is good. At the sick taken care at the stage of intestinal venous infarct the mortality can reach 50 % (1, 2).

4. Conclusion

The acute thrombosis of the portal vein is situated between the hepatic hile and the confluence vein lower mesenteric and spleen vein. The causes are superposables to those of the cavernoma portal. The diagnosis is easy thanks to the not invasive medical imaging. The main complications of the acute thrombosis of the system door are the venous mesenteric infarct, the later arisen of digestive bleedings due to the high blood pressure portal under person suffering from a liver complaint, and the compression of the biliary ways by the veins of the cavernoma.

The anticoagulating treatment is the treatment of first intention which often allows the repermeabilisation of thrombosis vessels in the acute phase. The thrombolyse in situ determine a high rate of repermeabilisation of the portal vein, but its place is restricted because of the frequency of the severe complications.

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