



ORIGINAL ARTICLE / *Cardiovascular imaging*

Computed tomography follow-up of acute portal vein thrombosis



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KEYWORDS

Portal system;
Venous thrombosis;
Tomography;
Spiral computed

Abstract

Purpose: To assess the evolution of acute portal vein thrombosis by computed tomography (CT).
Patients and methods: Retrospective single-centre study (2005–2011) including 23 patients who had an initial CT scan and a CT scan during the first year. The analysis compared the last CT scan available with that of the initial CT scan. Neoplastic thrombosis, extrinsic compressions and cavernomas were excluded. All patients received anticoagulant treatment.

Results: The causes included: cirrhotoses ($n=6$), blood disorders ($n=4$), locoregional inflammations and infections ($n=8$), abdominal surgery ($n=1$). The thrombosis was idiopathic in 4 cases. After a mean follow-up of 7.7 months, 7 patients (30%) benefited from a *restitutio ad integrum* of the portal system, a stable or partially regressive thrombosis was noted in 12 patients (52%) and an aggravation of the thrombosis was noted in 4 patients (18%). In the sub-group of portal vein thrombosis, repermeabilisation was noted in 37.5% of the patients (6/16) and 6 cavernomas developed.

Conclusion: CT monitoring helps follow the evolution of an acute portal vein thrombosis and demonstrates complete repermeabilisation of the portal vein in 30% of the patients.

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Abbreviations: CT, computed tomography; PACS, Picture Archiving and Communication System; PT, Portal trunk; SMV, Superior mesenteric vein; SMTerr, Splenomesenteric territory.

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Introduction

Portal vein thrombosis is a rare, although serious disease that requires specialised emergency care [1,2]. The mechanisms of obstruction group the fibrinocruoric thrombus, tumoral thrombi and extrinsic compressions. The formation of a fibrinocruoric thrombus is often multi-factorial. A general prothrombotic cause or a combination of prothrombotic disorders is found in 70% of the cases (malignant blood disease, thrombophilia), a locoregional cause in 30% of the cases (cirrhosis, locoregional inflammation and infection, locoregional surgery) [3,4]. Sometimes the cause is not identified and the thrombosis is considered to be idiopathic [3]. The occurrence of a thrombus in the portal system increases the venous pressure and the immediate risk of evolution, according to the thrombosed territory, is the occurrence of mesenteric infarction, splenic infarction and more rarely colonic infarction [5,6]. The risk of evolution depends on the disorder responsible for the thrombosis, any possible recurrence and the occurrence of portal vein hypertension. The treatment is based on effective anticoagulation. Without lysis of the thrombus, the patient is exposed to the occurrence of a portal cavernoma, portal hypertension, recurrent thromboses and obstructive biliopathy [7,8]. The initial clinical picture is non-specific and the diagnosis is based on an abdominal-pelvic CT scan. The anatomical-radiological outcome is poorly known. A recent study [9] showed that repermeabilisation of the portal system was possible. However, patients with cirrhosis were excluded and the medical imaging technique was not specified.

The main purpose of this study is to assess the outcome of thrombosis of the portal system by CT. The secondary goal is to describe the evolution of the portal vein thrombosis according to the cause.

Patients and methods

Patients

Between October 2005 and October 2011, 61 patients were admitted to our teaching hospital for the treatment of an obstruction of the portal system. The patient files were prospectively colligated during the "Portal Vein Thrombosis" multi-disciplinary meetings including the Hepatogastroenterology, Vascular Medicine and Biological Haematology departments.

The criteria for inclusion were: thrombosis of the portal system diagnosed by an abdominal-pelvic CT scan during the acute phase and at least one CT scan obtained during the first year of follow-up. A maximum period of one week between the admission and the initial CT scan was accepted. The criteria for exclusion were: tumoral thrombus (12 patients with hepatocellular carcinoma), the presence of a cavernoma on the initial CT scan (16 patients) and an extrinsic compression by pseudo-cyst of the pancreas (1 patient).

Patients without a CT scan in the initial phase or follow-up by CT were not included (9 patients).

A total of 23 patients were included. The clinical characteristics of the patients (Table 1) and the cause were

Table 1 Description of the population.

Parameters	Population (n = 23)
Age (mean, standard deviation)	57 ± 16 years
Sex [n (%)]	
Women	12 (52)
Men	11 (48)
Smokers [n (%)]	
No	16 (70)
Current	4 (17)
Former	3 (13)
Aetiological context [n (%)]	
Cirrhosis	6 (26)
Locoregional inflammation or infection	8 (35)
Locoregional surgery	1 (5)
Blood diseases	4 (17)
Idiopathic	4 (17)
Constitutional thrombophilia (n = 15 patients tested)	5 (33) ^a

^a In the 5 patients, the constitutional thrombophilia was associated with another aetiology.

colligated retrospectively. Six patients had cirrhosis of alcoholic (n = 2) or metabolic (n = 4) origin. Eight patients had a locoregional inflammation or infection: diverticulitis (n = 4), acute gastroenteritis (n = 1), acute pancreatitis (n = 1), haemorrhagic rectocolitis (n = 1), mesenteric panniculitis (n = 1). One patient had pancreatic surgery. Four patients had a blood disease: polycythemia (n = 1), thrombocythemia (n = 2) or monoclonal gammopathy of undetermined significance (n = 1).

Thrombophilia was searched for in 15 of the 23 patients. Five patients (22%) had constitutional thrombophilia (one combined protein C and protein S deficiency, three antithrombin deficiencies, and one combined antithrombin and protein C deficiency). The constitutional prothrombotic factors were, in this study, systematically associated with another cause:

- the combined protein C and protein S deficiency was associated with metabolic cirrhosis;
- the antithrombin deficiencies were associated with alcoholic cirrhosis, haemorrhagic rectocolitis and essential thrombocythemia;
- the combined antithrombin and protein C deficiency was associated with alcoholic cirrhosis.

No cases of thrombophilia were identified as the exclusive cause in the study.

All patients were anticoagulated with an effective dose as soon as the diagnosis was made, initially by heparin therapy and then an early relay by vitamin K antagonists (INR 2–3). All patients were anticoagulated with an effective dose between the first CT scan and the final follow-up CT scan.

The specific care of the causal disease was systematic.

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