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Portal Vein Thrombosis After Splenic and Pancreatic Surgery

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Abstract

The portal vein is formed by the confluence of the splenic and superior mesenteric veins, which drain the spleen and small intestine respectively. Occlusion of the portal vein by thrombus typically occurs in patients with cirrhosis and/or prothrombotic disorders. However, portal vein thrombosis (PVT) can also happen after determined surgeries. Moreover, PVT can have serious consequences depending on the location and extent of the thrombosis, including hepatic ischemia, intestinal ischemia, portal hypertension... In this chapter, we will review the incidence, management and prophylaxis of PVT after splenectomy, pancreas transplantation, pancreatic surgery and in the setting of acute and chronic pancreatitis.

Keywords

Portal vein thrombosis • Splenic surgery • Pancreatic surgery • Laparoscopy • Prophylaxis • Pancreatic neoplasms

1 Introduction

Portal vein thrombosis is a multifactorial disorder predisposed by certain risk factors, which can be broadly divided into acquired and inherited conditions [1]. Local intra-abdominal inflammatory processes (e.g., pancreatitis, inflammatory bowel disease), or trauma (e.g., splenectomy), increase the risk for portal vein thrombosis and tend to affect the larger veins. Heritable and acquired thrombophilias (e.g., prothrombin G 20210 mutation) and hypercoagulable states related to systemic disorders (e.g., nephrotic syndrome, malignancy) are more likely to affect the smaller veins [2]. However, this is not always the rule.

In this chapter we will review two of the main local causes of portal vein thrombosis, derived from surgical acts: splenic and pancreatic surgery.

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2 Portal Vein Thrombosis After Splenic Surgery

2.1 Epidemiology

Portal vein thrombosis is a complication of splenectomy less usual than bleeding or infection, but it can be potentially deadly. It is considered as an unfrequent event, although the recent increasing use of image techniques suggest that its appearance could be more frequent than previously suspected [3–6]. Several studies estimate its incidence in open surgery between 1.6 and 11 % after splenectomy [7–10].

In the last decade, laparoscopic splenectomy has become very popular for elective splenic surgery. Laparoscopic approach has shortened considerably the hospital stay and thrombosis may appear once the patient has left the hospital, conditioning a delayed diagnosis. Because symptoms are often mild and non-specific, being sometimes even an asymptomatic process, the diagnosis can be missed, achieving it when chronic complications appear, generally related with portal hypertension syndrome [11]. Therefore, the estimated incidence of portal vein thrombosis after laparoscopic splenectomy is completely different among the different published series, ranging between 8 and 54 % [3, 12–15]. Ikeda et al. [14] reported up to date the highest incidence of portal vein thrombosis after laparoscopic splenectomy. However, 67 % of their cases were asymptomatic and diagnosed after a screening with contrast enhanced CT scan. Since many patients remain asymptomatic, the real incidence of this complication is probably underestimated.

Moreover, some of the reported studies in literature are transversal studies, determining the prevalence of portal vein thrombosis rather than the real postoperative incidence. Some cases of thrombosis resolution without treatment have been reported: Loring et al. [16] describe two cases of complete resolution and one of partial one and Skarsgard et al. [17] two other of complete resolution. Thus, it cannot be discarded that in many transversal series there would have been more cases of portal vein thrombosis, that they were asymptomatic and that the thrombosis has completely disappeared along the time.

Anyway, though after open splenectomy it has not been investigated about the real incidence of portal vein thrombosis, as much as after laparoscopic approach, the incidence of portal vein thrombosis seems to be higher after a laparoscopic surgery.

2.2 Risk Factors

There are some factors described that increase the risk of developing portal vein thrombosis. Splenomegaly over 1 kg rises the risk of all post-splenectomy complications, but specially the risk of portal vein thrombosis, that is 14 times more frequent in those patients. A possible explanation of this phenomenon would be a sudden reduction of the splenic vein flow, originating a thrombus that migrates proximally towards portal vein. Apart from this, there is a bounce in the platelet number proportional to the extirpated splenic volume [3, 18].

Diverse works indicate that when the postoperative recount overcomes the million of platelets (1000×10^9/L), thrombosis risk also rises considerably [1]. However, Griesshammer et al. [19] point out that primary thrombocytosis is the one that increases thrombosis risk, not secondary thrombocytosis, concluding that thrombocytosis after splenectomy does not associate a higher risk of portal vein thrombosis. Other recent studies point out that only qualitative platelet disorders increase the risk of portal vein thrombosis [20]. Anyway, the use of antiplatelets is universally accepted when the recount overcomes the million of platelets [19].

Hypercoagulative disorders are also a risk factor for portal vein thrombosis. Qi et al. [21] supported that inherited antithrombin, protein C, and protein S deficiencies significantly increased the risk of portal vein thrombosis, although they were rarely observed in these patients. Similarly, the same authors also reported that the Factor V Leyden and prothrombin G20210A mutations are

associated with an increased risk of portal vein thrombosis without cirrhosis, although they were also rarely observed in such patients.

Regarding their results, Ikeda et al. [14] conclude that the laparoscopic approach implies a higher risk of portal vein thrombosis than the open procedure. Experimental studies [22] suggest that CO_2-pneumoperitoneum associated to anti-Trendelemburg position during laparoscopic surgery reduces the portal and splenic blood flow, being the flow reduction proportional to the intra-abdominal pressure [22]. The vessels clipage of the splenic hilium has also been involved to the blood flow reduction around the bounded area and to an increase venous ecstasy. These findings might suggest that laparoscopic approach could be the start-point of portal vein thrombosis, although its appearance weeks or months after surgery would indicate that it is a multifactorial process. Some cases of portal vein thrombosis have been described appearing up to 3 years after surgery [3].

2.3 Clinical Manifestations and Diagnosis

As previously mentioned, symptoms are often mild and non-specific, being sometimes even an asymptomatic process. Therefore, the diagnosis can be missed, achieving it when chronic complications appear, generally related with portal hypertension syndrome [11]. The progression of the thrombus, occluding portal and mesenteric veins, may cause acute hypertension in splacnic circulation and intestinal infarct, or develop long term portal extrahepatic hypertension, conditioning hepatic failure or the appearance of oesophageal varixes and portal cavernoma. Early diagnosis is therefore crucial, since the complete reabsorption of the thrombus can be achieved with adequate treatment [5, 6, 11].

Ultrasonography has been classically considered the gold standard for the diagnosis of portal vein thrombosis, because of its sensibility, accessibility, low costs and non-invasiveness [20]. However, at the moment it has been broadly overcome in terms of sensibility and specificity by contrast enhanced CT-scan, that allows the detection of portal segmentary and distal splenic vein thrombosis, difficult to observe at US-Doppler, because of the interference of intraabdominal gas [14]. Nowadays, contrast enhanced CT-scan should be maybe considered the test of choice to carry out when portal vein thrombosis is suspected [23].

2.4 Prophylaxis

Preventive measures to avoid portal vein thrombosis (**primary prophylaxis**) include perioperative use of anticoagulant, thrombolytic and antiplatelet treatments [20]. Prophylactic anticoagulation with low dosis of Low Molecular Weight Heparin perioperatively do not avoid completely the appearance of portal vein, but it probably reduces the risk of deep vein thrombosis or pulmonary thromboembolism [9, 23]. Ikeda et al. [14] do not use antithrombotic prophylaxis in their patients, what could probably have increased their thrombosis incidence; although Chaffanjon et al. [9] describe a thrombosis incidence of 6.7 % in spite of heparinic prophylaxis, while Skarsgard et al. [17] describe an incidence of 6.3 % without any anticoagulant treatment. Considering the proposed etiopathogenic way for portal vein thrombosis and valuing that most thrombotic cases appear in the first week after surgery, some authors think that it would be necessary to consider these patients as high risk subjects and anticoagulant prophylaxis should be prolonged up to 1 month after surgery. Probably, this would not avoid thrombosis, but could reduce the number of cases, always individualizing the risk of postoperative bleeding in each patient [10].

A recently conducted meta-analysis to explore the role of pharmacologic prophylaxis of PVST after splenectomy, concluded that pharmacologic prophylaxis might decrease the incidence of PVST after splenectomy in patients with portal hypertension and did not increase the risk of bleeding. However, the effect of pharmacologic prophylaxis of PVST in patients with

hematological diseases remained questioned. It has to be considered that the indication for splenectomy is different between Asiatic and Western countries. In Western countries, most of patients underwent splenectomy due to the hematological diseases, while in China and Japan most of patients underwent splenectomy due to the portal hypertension. Thus, the difference in the indications for splenectomy might lead to the discrepancy in the role of pharmacologic prophylaxis of PVST after splenectomy [23].

It has also been suggested that heparin combined with antiplatelet agents or Vitamin K antagonists could be indicated in high risk splenectomized patients, although their management appear to be difficult because of the risk of postoperative bleeding. Most published works do not recommend their employment with prophylactic aims [20].

Some authors recommend screening with US-Doppler or CT-scan as the best prevention method (**Secondary prophylaxis**), mainly in high risk patients (big spleen, mielodysplasic syndrome and thrombocytosis), that allow an early diagnosis [16, 24]. In our opinion, a contrast enhanced CT-scan should be performed when any suspicious clinical manifestation take place; US-Doppler can present numerous misdiagnosis. It is still unclear the value of a contrast enhanced CT-scan screening after laparoscopic splenectomy in high risk thromboembolic patients, considering the clinical importance of this entity and its consequences, but the variable incidence reported among the different studies reported in literature.

To avoid the appearance of complications secondary to portal vein thrombosis, anticoagulant treatment should be started (**Tertiary prophylaxis**). A complete disappearance of the thrombus after anticoagulant treatment between 2 and 6 months after its setting-up has been reported in around 75 % of the cases, with clinical improvement in the remaining 25 % [14]. In our experience, in those cases diagnosed diagnosed in acute phase and treated with Acenocumarol during 6 months, contrast-enhanced CT-scan carried out after having finished treatment, showed a complete disappearance of the thrombus. Agreeing with literature, we also defend that anticoagulation seems to be the most effective treatment, achieving resolution of the process in most cases [23].

3 Portal Vein Thrombosis After Pancreatic Surgery and Pancreatic Diseases

3.1 Pancreas Transplantation

Nowadays, results of Pancreas Transplantation (PT) have significantly improved [25]. More efficient immunosuppressive agents, better postoperative care and more refined surgical technique have improved overall survival and decreased postoperative complications after portal thrombosis (PT). Even so, surgical complications and technical failures keep on being a severe problem after PT, associated with increased morbidity and graft loss [26]. Most frequent events are vascular complications, pancreatitis, anastomotic leaks and intraabdominal infection.

Incidence of vascular complications after PT is around 10–20 %. These are divided in thrombosis, haemorrhagia, pseudo-aneurisms, anastomosis stricture and arteriovenous fistulas [27].

3.1.1 Thrombosis

Incidence of thrombosis is around 8.8–35 % (venous in 60 % of the cases and arterial in the rest 40 %) [28]. An early diagnosis is essential, but normally, and instead of an early surgical treatment, this complication is associated with a 50 % of graft loss [29]. There are two types of thrombosis:

(a) Early thrombosis: Most part of the cases (70 %). Normally during the first week after transplantation.
(b) Late thrombosis: More rare and associated to chronic failure of the graft. The mechanisms are not well-known.

Analysis of ***risk factors*** implicated in thrombosis of the graft is so much complex, with multiple variables in its pathophysiology. Nowadays,

Table 1 Risk factors for thrombosis

Risk factors	
Donor	Receptor
Older 45 years old and cardiovascular disease as cause of death	Pancreaticoduodenojejunal anastomosis
Asystolia donor	Acute failure of graft
Unstable haemodynamically	Peritoneal dialysis
Use of desmopressin	Hypercoagulability
Obesity with a BMI >30 kg/m^2	Re-transplantation
Traumatic extraction of pancreas with an excess of fluid preservation	Partial and segmentary pancreas transplantation
	Arterial reconstruction with Carrel "Patch"
Time of preservation >24 h	Excessive length of portal vein or use of an interposition of a portal venous graft
Preservation injury and pancreatitis of the graft	Implantation of the graft in left iliac fossa
	Post-transplant pancreatitis
	Immunosuppressive drugs (cyclosporine, tacrolimus)

it has been described multiple risk factors dependant on the donor and receptor, that are involved in this specific complication [30, 31] (Table 1).

Diagnosis

Clinical manifestations are variable [27], with acute abdominal pain in the location of the pancreatic graft (normally right iliac fossa), acute and not suspected hyperglycemia, haemoperitoneum (especially in venous thrombosis), thrombocytopenia, leucocytosis, gross haematuria (in venous thrombosis) and suddenly decreasing of the amylase levels in urine. Occasionally, a deep venous thrombosis of the ipsilateral iliofemoral system could be identified, due the retrograde progression of the portal venous thrombus.

However, in other cases, partial venous thrombosis can develop asymptomatically, and be detected in a routine Doppler ultrasound.

A Doppler ultrasound is mandatory in case of a suspected thrombosis to analyze arterial and venous flow. The absence of arterial or venous flow is suggested of vascular thrombosis; however there are episodes of graft loss and pancreatitis that could develop a diminution of the flow. A gammagraphy of the pancreatic graft and a CT angiography could be also performed. Anyway, and in cases of doubts, the definitive test is the arteriography.

Treatment of Venous Thrombosis

In cases of total venous thrombosis (TVT), urgent revascularization of the venous system is vital. This thrombolysis or thrombectomy can be performed by interventional radiologists, or by surgeons with an early re-laparotomy [32]. Total venous thrombosis has a poor prognosis, and in most part of the cases, a re-transplantation is required. In the series of Fernandez- Cruz et al. [33], reporting 20 cases of TVT, a transplantectomy was performed in 14 cases and a surgical thrombectomy with postoperative anticoagulation for 3–6 months in 6 cases. Four of these six cases could be recovered with a good posterior functional result.

In cases of partial venous thrombosis confirmed by Doppler, a thrombolysis or thrombectomy performed by interventional radiologists or high doses of anticoagulation could be used.

Most pancreas transplant centers utilize some form of anticoagulation following transplantation to prevent these complications. Moreover, aspirin is highly recommended. Unfractionated or low-molecular-weight heparin is often administered, but some centers use heparin selectively and typically at low dose to avoid postoperative bleeding. Warfarin is less frequently given and its use should probably be limited to patients with thrombophilia [28].

3.2 Pancreatoduodenectomy (Whipple Procedure)

Pancreatoduodenectomy (PD) is a complex procedure that brings a not insignificant number of postoperative complications. Of these, the most common complications can be divided in four groups: surgical site infections (SSI), delayed gastric emptying, bleeding and anastomotic leakage [34]. Nevertheless, other complications, much less frequent, exist. Their identification in early postoperative course is complicated and if left untreated, they may lead to the death of the patient.

Among such unfrequent complications, it must be included superior mesenteric vein (SMV) thrombosis with subsequent ischemia of the tributary area. Clinical symptoms of SMV thrombosis are untypical, obscure and characterized by slow progress, all this covered by early postoperative period [35]. Because of these obscure symptoms, it was not until 1935, that thrombosis of SMV was identified as a nosology entity [36, 37].

Although PD offers the only chance of cure for patients with adenocarcinoma of the pancreas, questions have arisen regarding the indication, safety and outcomes of patients undergoing extended resections for locally advanced disease [38]. While previous studies demonstrated an overall survival benefit after pancreatic resection without an increase of morbidity and mortality rates [39–41], high mortality rate was reported for patients with PVT after PD [42–45]. In the last years, venous resection and reconstruction is becoming more common during PD. There are multiple options for reconstruction of the mesenteric venous system ranging from primary repair to grafting with autologous or synthetic material [38]. Anyway, if *en bloc* resection with involved vein has been performed and the initial postoperative care of the patient was not uneventful, it must be kept in mind that a PVT could be established.

At this point, recommendations for anticoagulation following major venous reconstruction for malignancy are not clearly established, because it has been showed that the systematic administration of anticoagulation does not protect against venous thrombosis [46]. In a study of the durability of 64 PV reconstructions by Smoot et al. [38], no significant difference in thrombosis rate was observed between those who did and those did not receive anticoagulation. Most patients remained patent without the use of warfarin or aspirin, and that anticoagulation therapy did not seem to influence outcomes. A possible explanation is that, because of the high flow and the absence of valves in the portomesenteric vein, the risk for thrombosis seems to be low.

Diagnosis of this entity is hard by the fact that clinical symptoms are non-specific and covered by postoperative paralytic ileum and modified pain reaction secondary to analgesics [47]. Nausea, vomiting abdominal pain and distention with no other signs of obstruction appear to be the initial presentation in most patients. No plasma biomarkers for intestinal ischemia exist, and only D-dimers is used as a marker of [48]. This postoperative complication should be considered in a patient requiring unusually large amounts of fluids to maintain homeostasis.

Although angiography remains the standard diagnostic modality, CT scan of the abdomen may shows reduced contrast enhancement in the SMV with or without PV thrombosis, dilated intestinal loop with wall thickening and the presence of peritoneal fluid.

Therapy of thrombosis of SMV is divided into conservative, endovascular, and surgical. Basis of the *conservative management* was stated by Barrit and Jordan in 1960 [49]. Treatment of the thrombosis of SMV (heparinization) does not differ from the treatment of the thrombosis in any other localization.

The basis on the *endovascular treatment* is thrombolysis, either administered systemically or locally. First option is via transfemoral approach with the direct introduction of thrombolytic agents into the superior mesenteric artery (chemical thrombolysis); and the second alternative, is by direct aspiration thrombectomy from SMV without use of thrombolysis (mechanical thrombolysis) [50].

When the patient is clinically deteriorated, with a suspicious thrombosis of the SMV, with signs of peritonitis or bowel paralysis of unclear origin, a *laparotomy* is mandatory [51]. Goal of laparotomy is facilitation of venous outflow (usually by thrombectomy) and resection of the necrotic parts of the bowel. Considering complicated assessment of the bowel vitality in venous congestion, recommended practice is planned re-laparotomy 24–48 h after revision [52].

Mechanical injury to VSM during surgery can be considered as the most common cause of postoperative thrombosis of SMV, ant this occurred in patients with extreme inflammatory and fibrotic surrounding tissue around the pancreas (severe acute and/or chronic pancreatitis, huge tumors that involve PV...).

Prognosis of the patient depends on the clinical state, early identification and aggressive treatment. Management of the patient is multidisciplinary (surgeon, anesthesiologist, internal medicine specialist, radiologist...) but the mortality rate even after aggressive surgery is high. Due to the possibility of different surgical revisions, the use of open abdomen with negative pressure wound therapy could be indicated, not only to avoid the developing of a compartment syndrome, but also to evacuate fluids and contaminate collections [46].

3.3 Distal Pancreatectomy

Although incidence of PVT following pancreatic transplantation and pancreatoduodenectomy has been previously described [53] and it is well accepted, there is a paucity of data in the literature on PVT in patients undergoing distal pancreatectomy (DP) [54]. Recently, the Mayo Clinic [55] has published a study with nearly 1000 patients undergoing DP with or without splenectomy, and has showed an overall incidence of PVT of 2.1 % (21 patients). However, in this study, patients who had a portal venorrhaphy, portal venous reconstruction, pre-operative PVT or chronic pancreatitis were excluded.

Although, it is well-known that pancreatic cancer has a major risk of venous thromboembolism and that it is the most common indication for DP, surprisingly, PVT occurred infrequently in this population.

Clinical presentation of PVT was variable and depended on the extent and location of PVT. The median time from DP to diagnosis of PVT was 16 days. Non-specific abdominal pain was the most common symptom (52 %), clinical suspect for pancreatic leak or intraabdominal infection (24 %) and during the follow-up surveillance in the rest 24 %. Anyway, authors concluded that the true incidence of PVT after DP is difficult to assess, because some patients could develop asymptomatic PVT that was not diagnosed.

The diagnosis of PVT was confirmed by CT or ultrasonography in all the patients. Thrombus occurred in the main PV in 15 patients (71 %), right portal vein branch in 8 (38 %), left portal vein branch in 3 (14 %), and superior mesenteric vein in 7 (33 %) patients. In 8 patients (38 %) there were multiple segments of the PV involved, and a complete PV occlusion was seen in 9 patients.

The difference in frequency of PVT after DP in patients who underwent laparoscopic or open procedure was not statistically significant (6 % vs. 2.5 %).

Related to treatment, and although anticoagulation does not appear to influence the rate of PVT resolution, authors advice to use anticoagulation until larger and controlled studies define clear advantages and disadvantages. In their series, the duration of the treatment was 6 months, and there was no case of recurrence or progression of PVT. Over a median follow-up of 22 months, complete resolution, defined as recanalization of the portal vein, was observed in only a third of the patients, being these results similar to those obtained in other groups with the anticoagulation treatment for PVT from acute and chronic pancreatitis.

Risk factors for persistence of PVT were anesthesia time >180 min, DM type II, Body mass index (BMI) > 30 kg/m^2, thrombus in an intrahepatic segment of the PV, simultaneous

involvement of multiple segments, a complete occlusion of the PV and presence of thrombus in a sectorial branch of the right portal vein.

Duration of treatment has been largely discussed because of the risk of recurrence and progression of the thrombosis. Current literature on PVT does not support prolonged anticoagulation because of the low rate of recurrence and thrombus progression, and a substantial rate of gastrointestinal bleeding (10–26 %) [53–56].

Because most DP are performed for a malignant disease, and due to the operation itself (it is a pro-coagulant condition), in the absence of thrombus propagation or pro-coagulant condition (e.g., Factor V Leyden, Protein C/S deficiency), authors recommend that the decision for anticoagulation should be made individually, on basis of the extent of PVT and clinical manifestations. Anyway, they advise to provide at least a short-term anticoagulation treatment to patients with PVT followed by repeat imaging study to assess the response of the treatment and decide its duration.

3.4 Pancreatitis

Venous thrombosis (mesenteric, splenic and portal) is a frequent complication that occurs as a sequelae to pancreatitis [57]. All forms of pancreatitis have been implicated as risk factors for thrombosis. Targeted studies report its incidence in hereditary pancreatitis, autoimmune pancreatitis, acute pancreatitis (AP) and chronic pancreatitis (CP). It is considered that this entity is more commonly associated with CP, although a single attack of AP appears sufficient to cause this disorder. The physiopathology of this complication seems to be related to the compression of the vein following inflammation and fibrotic tissue of the pancreas, the injury of the intima secondary to the acute attack and the compression by pseudocysts. Anyway, venous thrombosis may be linked to inherited coagulation disorders, such as deficits of protein C or protein S, or acquired coagulopathies, such as antithrombin III deficiency. Clinical consequences of the venous thrombosis depend on the velocity of instauration, the grade of occlusion and the creation of collateral blood flow [58, 59].

3.4.1 Splenic Thrombosis

In either AP or CP, the incidence of splenic vein abnormalities has ranged from 0.9 to 54 % [60] in surgical series and up to 89 % in radiographic series [61]. Regardless of its etiology, splenic vein thrombosis (SVT) generates a localized form of portal hypertension commonly referred to as “sinistral”, “left-sided” or “linear”. Collateral blood flow develops through the splenoportal or gastroepiploic systems and the resulting localized venous hypertension may produce gastric, esophageal or colonic varices. Historically, patients with SVT most commonly presented clinically with an episode of gastrointestinal (GI) bleeding or abdominal pain. However, nowadays, with the improvement of availability and quality of CT scan, the majority of the patients are asymptomatic. Despite the heterogeneity of available data, the meta-analysis of Butler et al. [58] quantifies an overall SVT incidence of 14.1 % and a bleed rate of 19 %. In relation to operative management, it has been suggested that patients with SVT and a prior history of upper GI tract bleeding or symptomatic hypersplenism may represent a high-risk group and the splenectomy is mandatory. By contrast, asymptomatic patients without history of bleeding, in whom SVT was identified through imaging, were found to have an incidence of bleeding of only 3.8 % and a conservative management could be adopted.

3.4.2 Splenoportal Thrombosis

The real prevalence of splenoportal thrombosis (SPT) is not well-known. Sometimes it is an incidental finding on radiological imaging performed to assess the severity of an attack of AP. Some studies [62, 63] reported and incidence of 25 % in patients with AP, so this entity has to be ruled out in these cases. The problem is that its clinical manifestations may include signs and symptoms that overlap with those of the pancreatitis. Although the natural history of splenoportal vein thrombosis in pancreatitis is unclear; severe

haemorrhage, bowel ischemia, portal hypertension and liver failure have been reported.

Diagnosis of SPT is essential even in asymptomatic patients because this could lead and modify the surgical or endoscopic technique. Arteriography is mandatory, but a CT-angiography could also be performed, reporting changes in pancreatitis.

3.4.3 Mesenteric Thrombosis

Incidence of mesenteric thrombosis (MT) is difficult to assess, and normally it is an incidental radiological diagnosis without intestinal ischemia or in the necropsies series. Some authors describe an incidence higher than 10 % [62].

Subacute MT is characterized by large evolution abdominal pain without intestinal ischemia, meanwhile patients with chronic MT remains asymptomatic and develops signs and symptoms of portal hypertension. Treatment of choice in cases of ischemia is surgery but in absence of this complication, anticoagulation with heparin is useful.

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