

OCCLUSIVE SYNDROME CAUSED BY MESENTERIC VENOUS INFARCTION, ASSOCIATED WITH RAYNAUD SYNDROME

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OCCLUSIVE SYNDROME CAUSED BY MESENTERIC VENOUS INFARCTION, ASSOCIATED WITH RAYNAUD SYNDROME (Abstract): Mesenteric infarction, a rare process in young, presents with unspecific clinical features of which abdominal pain and obstipation are dominant. Because many clinicians are unfamiliar with the condition, they fail to diagnose it early; the subsequent negative course then requires intestinal resection. We describe the case of a young private with acute abdominal pain and obstipation, diagnosed intraoperatively with extensive enteromesenteric infarction of venous type. Small bowel resection of almost half of the total length was sufficient and curative regardless the performance of the anastomosis with the last ten centimeters of the ileum.

KEY WORDS: MESENTERIC ISCHAEMIA, RAYNAUD SYNDROME

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INTRODUCTION

Enteromesenteric infarction is not a single entity, but rather a syndrome that includes: complete occlusion or stenosis of the mesenteric arteries by embolism, thrombosis or obliterative disease, thrombosis of the mesenteric veins, prolonged splanchnic vasospasm becoming intractable.

The relative incidence of mesenteric arterial as opposed to venous occlusions is not known, as well as the clinical distinction of each precise type is often difficult. It has been estimated that 15 - 20% of the mesenteric vascular obstructions are due to primary venous thrombosis and almost 50% to primary arterial occlusion; in the remaining 30 - 35% of cases intestinal infarction occurs in the absence of major arterial or venous occlusion [1].

Mesenteric venous thrombosis may be idiopathic, or may evolve secondarily as a complication of several clinical disorders, such as: intraabdominal infections, hematological disorders, portal hypertension or abdominal trauma [5].

The idiopathic cases, responsible for 25% of venous thrombosis, have sometimes a past history of peripheral thrombophlebitis, suggesting a common cause: an inherited hypercoagulable disorder, such as deficiency of protein C, protein S or antithrombin III [3].

Raynaud syndrome represents the particular situation of vascular disorders dominated by peripheral vasospasm and evolving with episodic cutaneous color changes from palor to cyanosis and pink discolorations of the digits, most commonly located at the upper extremities. The condition occurs in 70 - 90% of cases in women and it is also a characteristic of the young adults, almost all younger than age 40. Over 50% of patients with severe Raynaud symptoms have documented autoimmune disease [3].

The correlation between the two distinct entities may be illustrative according to the physiopathological bases.

CASE REPORT

A twenty years old man with a past history dominated by viral hepatitis type A, Raynaud syndrome, interstitial pneumonia and frequent respiratory infections, was seen at the

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emergency service of the hospital for acute periombilical pain, obstipation, nausea and vomissements. The acute onset was claimed to appear early in the morning at five o'clock, after a previous day with prolonged exposure to low temperatures (8 hours in outside winter conditions) and increased physical effort with mild Raynaud manifestations. The pain was intense, continuous, not alleviated by any position or by vomissements, partially decreased by antispastic drugs. The patient was admitted at the internal medicine service in the same morning, but the pain increased progressively and radiated in both lumbar regions, then it became somehow located in the right flank and iliac fossa. In the evening a surgical examination was requested and physical examination revealed a painful abdomen, slightly distended, with maximum intensity of the pain and tenderness in the right flank and right iliac fossa. Apart from these, a moderate splenomegaly was identified in the left hypochondrium and a peripheral micropolyadenopathy in the inguinal and axillary regions. Auscultation revealed decreased peristaltic sounds.

Although the routine blood studies were normal, except a mild metabolic acidosis, the patient was transferred in the surgical service, being suspected with acute appendicitis.

Abdominal X ray showed air-fluid levels and some distended loops of small bowel.

Few hours later the patient was operated under general anesthesia. Laparotomy has established the final diagnosis according with the aspect of the distal half of the small bowel, sparing only the last 10 cm of the ileum and almost the entire jejunum. The involved loops were distended, edematous, cyanotic, aperistaltic, with congestion of the correspondent mesentery. A second degree splenomegaly was confirmed intraoperatively. An extended enteral resection was performed with a two layers side to side jejuno-ileal anastomosis, with the last 10 cm of the ileum.

The postoperative course was favorable, with moderate difficulty in regaining the intestinal peristalsis. Postoperative ileus was overcome in the sixth day only with therapeutic enemas and parasympathomimetic drugs. Heparin therapy was started immediately after the operation at high doses for seven days, and then switched on oral anticoagulants. High dose antibiotherapy with piperacillin and metronidazol was administered for five days during the postoperative period.

Further investigations were made attempting to define the etiology of the infarction. Blood analysis revealed moderate leucocytosis $15,000/\text{mm}^3$, hepatocytolysis (ALT = 230 UI/L, AST = 190 UI/L), slightly elevated erythrocyte sedimentation rate 20 mm/h and normal electrophoresis. The urine sediment presented rare leucocytes, erythrocytes and albumin. The patient was referred from the hospital in the eighth postoperative day.

DISCUSSION

Mesenteric infarction is an unusual condition in young adults, but the diagnosis must be considered as a real alternative in every case of atypical abdominal pain and obstipation, when there are enough doubts about more common possible diagnosis [2, 4].

Leucocytosis which characterizes somehow the pathologic condition of a mesenteric infarction, could have a late appearance, with a considerable delay from the irreversible necrosis of the bowel. Therefore, in such doubtful conditions, the leading element guiding towards the operation should be the progressive pain, without any significant relief on usual analgesics. It is preferable to perform a laparotomy, or even better a laparoscopy, anytime when a suspicion about the existence of a vascular enteral pathology is raised, obviously, if the emergency service has not the possibility of CT (computed tomography). Ultrasound exam lacks enough specificity and accuracy in identifying the edema of the involved loops, with the characteristic aspect described in literature as thumb printing [2].

An early diagnosis is extremely important as a mean of decreasing the necessity for bowel resection imposed by necrotic irreversible lesions. Early high doses heparin therapy could have spectacular effects in situations of reversible ischemia [3].

Another goal of the postoperative study was to confirm the diagnosis of venous mesenteric infarction by hystopathological examinations, excluding the possibility of some type of necrotizing or toxic enteritis. Anatomopathological examination showed hemorrhage and hemorrhagic necrosis involving all the layers of the intestinal wall, associated with edema and inflammatory infiltrate; small vessels at the edge of the mesentery were obstructed with thrombi.

The next step was to establish the correlation between the mesenteric infarction, splenomegaly and Raynaud syndrome on physiopathological basis. Elevated values of transaminases associated with splenomegaly guided the clinical thought towards cirrhosis with portal hypertension, as a favoring factor of the venous infarction. Instead, during the postoperative course, splenomegaly vanished, proving its congestive nature. Moreover, the echografic appearance of the liver, spleen and portal vein were normal, sustaining the normal macroscopic aspect of the liver found during the operation. Thus, the diagnosis of liver cirrhosis became improbable.

Raynaud syndrome could be a convincible favoring factor of the mesenteric venous infarction, as well as a prolonged arterial mesenteric vasoconstriction developed on low temperatures, could induce a venous stasis in the splanchnic territory, due to the absence of the propulsive force before the vein (“vis a tergo”). The hypothesis is sustained with the clinical manifestations of the disease during the previous day before the onset. Venous stasis was probably followed by venous thrombosis in the portal venous system, with different degrees of obstruction. Postoperative heparin therapy was responsible for the spectacular remission of the splenomegaly. [3]

Regarding the anastomosis with the last ileal loop, it seems the classical critical area of Treves was overemphasized; usually, the last 1 - 2 cm of the ileum are insufficient vascularized, but more proximal, a side to side anastomosis could be performed safely.

CONCLUSSION

Raynaud syndrome and probably other more vasculospastic disorders could precipitate the development of severe ischemic lesions in splanchnic territory.

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