LETTER TO EDITOR

SPLENIC INFARCTION WITHOUT THROMBOSIS SECONDARY TO SEVERE ACUTE PANCREATITIS

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Abstract

Introduction: Acute pancreatitis is a common disease that is usually mild, although in some cases it may present complications, among which are vascular ones such as splenic infarction.

Clinical case: A 71 years old woman was admitted for acute pancreatitis, who presented a torpid evolution throughout the first admission with associated acute cholecystitis and an encapsulated necrotic collection that required endoscopic drainage using a luminal apposition prosthesis and endoscopic necrosectomy. A month and a half later, the patient was readmitted for abdominal pain, with abdominal CT scan showing a very thinned splenic vein and patent artery without identifying thrombosis (with 700,000 platelets/mm3), associated splenic infarction as well as worsening of the inflammation of pancreatitis. Given these findings, we were decided to start anticoagulation with enoxaparin at prophylactic doses for splenic vascular stenosis, achieving resolution of the thrombocytosis (410,000 platelets/mm3), disappearance of the splenic necrotic area and improvement of the pancreatic inflammatory component confirmed by CT scan.

Conclusion: When splenic infarction is associated with severe pancreatitis with splenic thrombosis, anticoagulation is indicated. However, when there is no splenic thrombosis and the splenic infarction occurs in the context of critical vascular stenosis with high thrombocytosis, the indication for anticoagulation is controversial, with no consensus in the literature. Furthermore, at present it is not establish the active ingredient to be used or its dose, and highlights the need to carry out clinical trials in order to establish clinical or consensus guidelines.

Keywords: splenic infarction; severe acute pancreatitis.

Introduction

Acute pancreatitis is a disease with a high and increasing incidence in our environment and is a frequent cause of hospitalisations[1]. Vascular complications are an infrequent complication of severe pancreatitis[1], including splenic infarction, which is increasingly being described in association with inflammatory processes of the pancreas, justified by the close relationship between the pancreas and the splenic hilum[2].

Clinical case

A 71-year-old woman was admitted for acute pancreatitis with a torpid evolution throughout the first admission. Two

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weeks later, she presented clinical and analytical worsening due to acute cholecystitis, which was detected in imaging tests and treated with empirical antibiotic therapy. One month after admission, he presented a new clinical and analytical worsening, and an echoendoscopy was performed, visualising a small collection of 4cm in the pancreatic body with a permeable splenic axis and another collection suggestive of an infected encapsulated necrotic collection measuring 6x6x8cm adjacent to the compressing gastric body, and transmural drainage was performed using a Hot-Axios luminal apposition metal prosthesis, draining abundant pus. On successive days, endoscopic necrosectomy was performed through the prosthesis, which was finally removed and a 7Frx10cm pig-tail plastic prosthesis was placed, with radiological improvement prior to discharge. In addition, enzyme replacement treatment with Kreon 50000 was started after each main meal.

One and a half months later, the patient came to the emergency department complaining of severe abdominal pain in the left hypochondrium-flank without fever. Laboratory tests showed elevated acute phase reactants, thrombocytosis, mild coagulopathy and a normal pancreatic profile. An abdominal CT scan was requested, showing radiological worsening with a greater inflammatory component and peripancreatic fluid, very thinned splenic vein with permeable splenic artery and signs compatible with splenic infarction without identifying thrombosis (Figure 1) with associated thrombocytosis (700,000 platelets/mm3). It was decided to anticoagulate the patient with enoxaparin at prophylactic doses due to splenic vascular stenosis. After discharge and review in consultation, resolution of the thrombocytosis (410,000 platelets/mm3) and stenosis was observed due to a decrease in the pancreatic inflammatory component, homogenisation of the spleen and disappearance of the splenic necrotic zone (Figure 2) with no new collections and a decrease in inflammatory changes in the control CT scan; anticoagulation was therefore withdrawn.

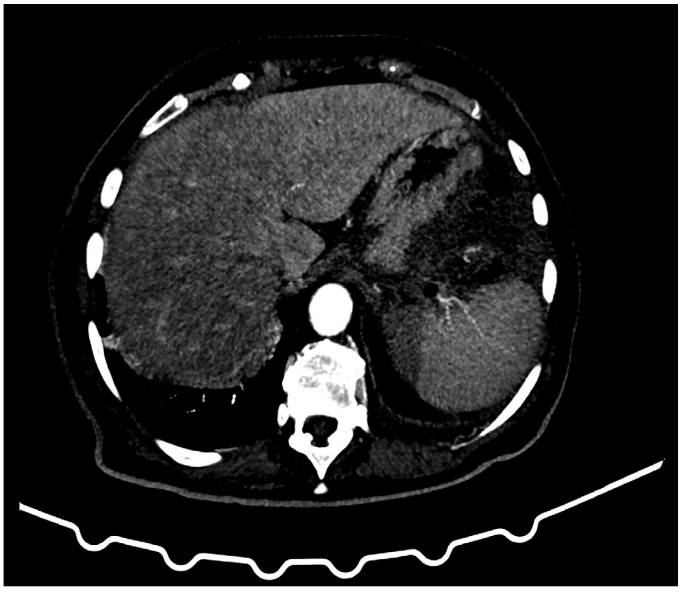

Figure 1. ***The arterial phase shows a thinned splenic artery at the level of the splenic hilum with a hypodense splenic area compatible with splenic infarction.***

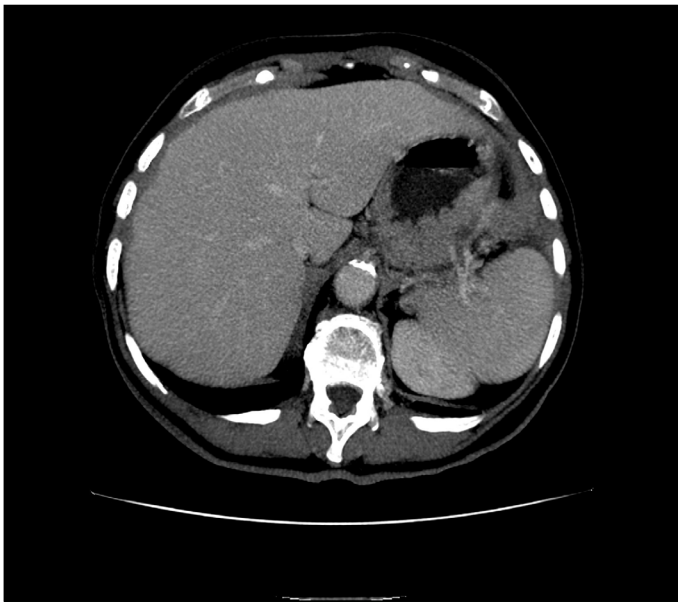

Figure 2. ***In the portal phase, an increase in the calibre of the splenic artery and repermeabilisation of the splenic vein at the level of the hilum with homogenisation of the spleen and disappearance of the splenic infarction are identified.***

Discussion

Splenic infarction is a complication increasingly described in association with inflammatory processes of the pancreas, the incidence of which is increasing. The most frequent symptom is left hypochondrium pain, which may be accompanied by fever, chills, nausea and vomiting, pleuritic pain and left shoulder pain (Kher's sign)[2] as described in our patient.

In splenic infarction associated with arterial and/or venous thrombosis, the treatment of choice is anticoagulation without specifying the drug used[3]. A recent retrospective cohort study has compared the benefits of initiating anticoagulation with both heparin followed by vitamin K antagonists and the use of direct-acting oral anticoagulants, establishing that there is an improvement in survival without increasing the risk of bleeding[4]. Thrombolysis and/or thrombectomy would be indicated if symptoms persist despite anticoagulation, reserving surgery if complications such as mesenteric ischaemia develop[5].

In splenic infarction associated with severe pancreatitis, the indication for anticoagulation is controversial and there

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is no consensus in the literature. No references have been found on how to proceed in cases of splenic infarction without thrombosis and with critical vascular stenosis in the context of a complication of severe pancreatitis.

In our case, we considered whether or not to anticoagulate the patient because the splenic infarction was associated with marked thrombocytosis without thrombosis. Given her age, we opted to use enoxaparin at prophylactic doses in order to avoid splenic thrombosis until the inflammation in the area improved or resolved, which would secondarily improve the splenic vascular stenosis. Subsequently, months later, after analytical and radiological control, the resolution of thrombocytosis and vascular stenosis was verified, and enoxaparin was discontinued.

In summary, the treatment of splenic infarction without thrombosis secondary to severe acute pancreatitis is a therapeutic challenge. The decision of whether or not to anticoagulate, the active ingredient to use and its dose, is currently undefined and highlights the need for clinical trials in order to establish clinical or consensus guidelines.

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