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Splenic Infarction: An Unusual Condition with Numerous Difficult Options: A Case-Report, Literature Review and Surgical Observations

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Abstract

Splenic infarction is a rare cause of acute abdomen whose aetiology is stratified according to age group. In patients under the age of 40 most causes are attributable to hematological or genetic pathologies, while in patients over 40 years old, the thromboembolic manifestations play a major role. The diagnosis of splenic infarction can be almost an enigma for the doctor who is evaluating a patient with non-specific abdominal pain, possible hyperpyrexia or rise in phlogosis and LDH indexes. The scientific literature on splenic infarction is rather scarce and consists mostly of articles with few cases. In this article, with the help of a rather emblematic clinical case that has come to the attention of the Emergency Department of the S. Anna Hospital in Ferrara, and subsequently to the Surgery Unit, we want to reflect not so much on the diagnosis of this pathology but rather on the therapeutic strategies following the diagnosis. Is splenic infarction a pathology of surgical interest? On what terms and times should surgical treatment be proposed to the patient? Is there an alternative to surgery? How should follow-up be conducted in these patients? These are questions to which the literature currently available has not yet been able to answer and, in the future, we hope we will start researches on the treatment of a condition which does not seem to be so rare.

Keywords: Splenic infarction; Case report; Diagnosis; Surgery; Laparoscopy

Abbreviations: LDH: Lactic Dehydrogenase; LAC: Anti Lupic Coagulant; AF: Atrial Fibrillation; DVT: Deep Vein Thrombosis; PT INR: International Normalized Ratio Prothrombin Time; CRP: C-Reactive Protein; PLT: Platelets; ER: Emergency Room; CT: Computed Tomography; LMWH: Low Molecular Weight Heparin; PET: Positron Emission Tomography; FDG: Fluorodeoxyglucose; ENA: Extractable Nuclear Antigens; RF: Rheumatoid Factor; NOAcs: Non-Vitamin K Oral Anticoagulants; ASA: American Society of Anesthesiologists; OPSI: Overwhelming Post-Splenectomy Infection; OPSS: Overwhelming Post-Splenectomy Sepsis

Introduction: A Great Mime

Splenic infarction has always been counted among the rarest causes of acute abdomen, but, it does not turn out to be such a rare eventuality in the clinical practice [1]. The causes of splenic infarction are various; thromboembolic manifestations and haematological diseases play a prominent role. We can stratify the causes into two main subgroups: in patients below the age of 40-50 years; in this group it is mainly caused by haematological proliferative diseases while in over 50 years patients the various thromboembolic etiologies are the most important cause (atrial fibrillation, endocarditis, prosthetic heart valves, patent foramen ovale, pancreatitis and abdominal masses that create ab estrinseco compression). A separate chapter would be dedicated to the genetic causes of hypercoagulability that represent an intrinsic risk independent of the age factor (mutations of coagulation factors in the pro-thrombotic sense, protein C and protein S mutations, presence of LAC antibodies, hemoglobinopathies).

Further note deserves the iatrogenic forms post vascular procedures such as embolization procedures for splenic hemorrhage mainly due to traumas [2-3] (Table 1). Clearly, the presentation of the symptomatic cortege at least partially follows the etiology, it is depending on the basic condition that may be the cause of splenic infarction, we can have a series of manifestations that can create embarrassment in the interpretation of symptoms and in the attainment of the diagnosis.

The classic presentation of splenic infarction is a dull pain localized mainly to the left abdominal quadrants, which can be associated with hyperpyrexia, nausea or vomiting, neutrophilic leukocytosis with increase of the inflammation indexes and sometimes increasing of LDH [2-5]. Table 2 for clinical and laboratory presentations [6,7]. It is, unfortunately, a common clinical presentation of many abdominal diseases from a more common gastroenteritis up to the onset of an intestinal obstruction or an appendicopathy. At the ends of the possible presentations, on the other hand, we also have the possibility that the patient presents himself with peritonism or even the lack of abdominal pain symptoms; which clearly creates even more confusion on the possible causes.

To this symptomatic cortege then, the possible symptoms related to the cause of the splenic infarct are to be added; a patient who has pancreatitis or an abdominal mass can have a symptom of the most varied linked to the entity of pancreatitis or to the endoabdominal organs affected by the mass; a patient presenting with the symptom cortex that we have previously defined and to whom a new onset AF or an endocarditis is found, could be attributed its symptomatology to the cardiological episode and the same for all the presentations related to the causes listed in Table 1.

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Under 40-50 y	Over 40-50 y	Genetic Causes	Iatrogenic Causes	Trauma
Hematological malignancies (leukemia, lymphomas, myelofibrosis)	Cardio-embolic disorders (endocarditis, atrial fibrillation, Vsn wall-related thrombosis following acute myocardial infarction, prosthetic heart valves, patent foramen ovale, cardiac myxoma)	Genetic hypercoagulable states (protein C or S deficiency, antiphospholipid antibody syndrome)	Acquired hypercoagulable conditions (use of oral contraceptives, erythropoietin therapy)	Closed abdominal contusive trauma
Benign hematological diseases (polycythemia vera)	Embolic disorders (thrombosis of the splenic vein)	Hereditary hemoglobinopathies (sickle cell anemia)	Post-embolization procedures (splenic artery embolization)	Traumas with torsion of the splenic vascular pedicle
Miscellaneous (pancreatitis, sarcoidosis, toxic shock syndrome, amyloidosis, pancreatic neoplasms, acute distress respiratory sindrome- ARDS, cholesterol emboli syndrome - MCES4)			Post-surgery (esophagectomy, gastrectomy, liver transplantation, sclerotypy of bleeding gastric varices, vasopressin infusion)	

Table 1: Causes of splenic infarction.

Symptom or manifestation	Frequency of presentation (% of patients)
Pain in the left abdomen side	40-50%
Unspecified abdominal pain	32-36%
No abdominal symptoms	25-30%
Hyperpyrexia (TC>38°)	30-36%
Neutrophilic leukocytosis	56-67%
PCR extension	90%-97.5%
Increasing LDH values	71-72%
Nausea or vomiting	30-32%

Table 2: Frequency of presentation of the symptoms associated with splenic infarction.

Clinical Case: Splenic Infarction or Imitation??

A 70-year-old male patient, which we will identify with the abbreviation AT, appears in the Emergency Department complaining pain in his left side radiating to the lower abdominal quadrants associated with urinary disorders for several days. He does not complain of hyperpyrexia nor nausea or vomiting. The patient has been referring constipation for many years, so he regularly takes intestinal cathartics. The medical history that Mr. AT presents to the PS doctor includes previous DVT (deep vein thrombosis) in the lower right limb, pulmonary asbestosis and OAT (oral anticoagulant therapy) in progress for a cardiac pathology not better specified by the patient.

From this first glimpse of our patient we see how the clinical presentation is suggestive of many pathologies from the most trivial to the most investigative. The patient is 70 years old, although he has not presented a history of an BPH (benign prostatic hypertroph) with a simple bladder globe, or a renal colic or a simple gastroenteritis or an initial episode of diverticulitis, even though colic diverticulosis is not among the anamnestic data.

At the first aid physician's visit, the patient has a globose abdomen, widely treatable, painful to deep palpation along the whole colic and hypogastrium even if there is no sign of peritonism. negative Blumberg sign, negative Murphy sign, negative bilateral Giordano sign. There are no signs of bladder globe; placed a foley catheter with a small quantity of hyperchromic urine. There are traces of normochromic feces in ampoules to rectal exploration. Negative cardio-pulmonary objectivity, and Electrocardiogram in the limits.

Complete blood chemistry tests are performed with no leukocytosis but increased CRP (13.43 mg/dL), LDH elevation (307 U/L), and mild thrombocytopenia (PLT 91 $\times$ 10^{-3}/uL). In addition, chest X-ray and direct abdomen X-ray are performed from which it is exclusively coprostasis to the right, widespread pneumatization of intestinal loops in the absence of significant levels of hydro-aviation. The patient is therefore redirected to the treating physician with the diagnosis of diffuse colonic coprostasis and concomitant urinary tract infection; he has been prescribed an intestinal cathartic and an antibiotic therapy.

Many patients access Emergency Rooms every day, complaining of varied and non-specific symptoms. In most cases with non-significant hematochemical examinations and in the absence of a certain diagnosis, they are referred to their doctor for subsequent evaluations and often the symptoms regress but as we will see this is not our case. After 3 days, Mr. AT goes to the same ER sent by the attending physician for suspected diverticulitis, presenting pain in the left iliac fossa. Blood tests show leukocytes at the upper limits, more neutrophils, substantially unchanged PCR as well as the thrombocytopenia observed at the previous evaluation.

No increase in amylasemia or lipasemia, relatively increased PT INR also in consideration of oral anticoagulant therapy (PT INR 3.72) and slight drop in hemoglobin from 14.1 mg/dL a few days earlier to 12.6 mg/dL. There are no significant alterations to the remaining tests, even the direct abdomen X-ray is superimposable to the previous one. Thus, it was decided to ask for the opinion of the General Surgeon who did not see objectivity different from the evaluation of the colleague of the ER but in the suspicion of acute diverticulitis he asked for a diagnostic deepening with contrasting abdomen CT.

Mr. AT refuses to undergo the examination, although it is not allergic, so the diagnosis is performed without intravenous contrast injection and reports:

- Slight inhomogeneity of the peripancreatic fat with apparently regular pancreas.
- Left kidney with cocoon margins with thinned cortex at the upper third-middle pole. Aneurysmal dilatation of the abdominal aorta (3.5 cm above the carrefour). Litiasic gallbladder.
- Diverticulosis of the sigma currently without signs of inhomogeneity of the surrounding fat or effusion.
- A different surgeon called in consultation to evaluate the requested CT recommends hospitalization in the doubt of a pancreatitis or biliary colic with repetitive indication of abdomen CT with contrast in case of worsening of the clinical picture or blood tests.

The patient is then admitted to the Internal Medicine department. Colleagues in Internal Medicine start with an accurate medical history and reassessment of the medical documentation held by the patient and some interesting data emerge. In addition to pulmonary asbestosis, a previous DVT to the lower right limb already indicated by the patient, it emerges that oral anticoagulant therapy has been undertaken due to

the presence of a patent foramen ovale as well as the DVT and ischemic brain lesions, moreover the patient is affected from Castelman's disease, Sjogren's syndrome, psoriatic arthropathy, glaucoma, arterial hypertension and dyslipidemia. From the patient's documentation it also emerges that he performs periodic checks at the Rheumatology clinics for positive anti-nucleus, anti-DNA, anti-phospholipid, rheumatoid factor, LAC and thrombocytopenia antibodies. A picture of widespread autoimmunity and hematologic diseases is thus highlighted, without considering the previous history of thrombosis and the persistence of the patent foramen ovale. On the other hand, however, it is necessary to consider the patient has been on anticoagulant therapy for some time, in addition the last data of PT INR is 3.72.

On admission to the Internal Medicine Unit the patient is paucisyntomatic, an antibiotic therapy is set up with meropenem and ceftriaxone, IV rehydration and analgesics as needed. Upon admission, acenocumarol therapy is discontinued and is first replaced with LMWH at scoagulant dosage then with Fondaparinux due to worsening of thrombocytopenia (PLT 65 × 10^{-3}/uL on day XII of admission). From the haematochemical tests performed at the time of admission there were no noteworthy alterations: leukocytosis and RCP progressively fall within the normal ranges, LDH is lowered to the normal limits and the chemical-physical examination of the urine does not show pathological findings. On the III day of admission the venous echocolordoppler is performed on the lower limbs showing on the right, peroneal DVT, medial twinning, popliteal vein and small saphenous vein; the thrombotic process extends to the femoral venous axis with maintained patency of the common femoral artery.

Respectively in the IV day and in the V day of admission, the patient is subjected to Gastroscopy, which shows a chronic erythematous gastropathy and erosive duodenitis, and Colonoscopy that shows a picture of diffuse diverticulosis of the sigmoid and polyp of the sigmo-rectus joint that is removed (Histological Examination: Tubular adenoma with low-grade dysplasia). Some autoantibodies are also repeated with a finding of positivity, however weak, for the lupus anticoagulant (or LAC) and positive anti-nucleus antibodies (1:320). In the VI day from admission, however, he underwent a CT with contrast:

- The scans performed at abdominal level showed complete thrombosis of both the splenic artery at the origin and the vein splenic up to the portal olive with a large hypodensity of three quarters of the splenic parenchyma referable to ischemia.
- Presence small peripancreatic arterial and venous collateral circles that revascularize the spleen wall (Figures 1-3).

Given the TC response, Mr. AT undergoes a Hematologic revaluation that recommends performing a global body FDG PET to settle the eventual reactivation of Castelman's disease (performed in X day by the admission that shows no signs of disease recovery) and Rheumatologic which recommends a series of in-depth studies on autoimmunity: C3-C4 dosage (results in the normal ranges), ENA, anti-DNA and RF (negative results) and capillaroscopy (framework compatible with non-specific alterations of the microcirculation). Cardiological evaluation performed during the hospitalization period does not indicate the correction of the oval foramen defect and recommends the introduction of NOAcs therapy. The patient is re-evaluated immediately before discharge on the 16th day from admission, by the same General Surgeon who advised him to be admitted to an internal environment which directs the patient to ambulatory control 30 days after discharge. The patient is therefore discharged with the following hypothesis about the nature of his clinical condition:

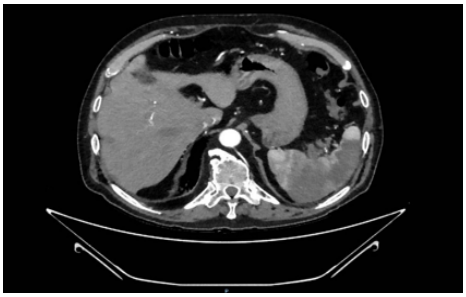

Figure 1: CT scan showing splenic infarction (transverse plane).

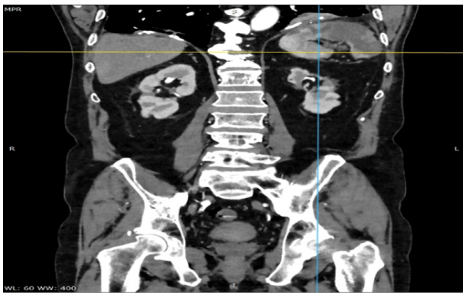

Figure 2: CT arterial phase showing splenic infarction (coronal plane).

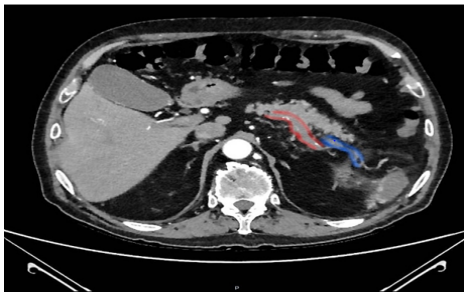

Figure 3: CT arterial phase showing artery and splenic vein thrombosis (transverse plane).

The genesis of venous thromboembolism occurring in apparently well-behaved anticoagulant therapy, now does not appear to be unambiguous; the investigations conducted so far reasonably exclude the presence of an underlying neoplastic pathology. The suspicion of rheumatologic pathology for which the patient will be followed in an outpatient procedure persists. As far as the splenic infarction is concerned, it is reasonable to hypothesize that it is secondary to embolization from the venous thrombotic process in the lower limbs in the context of a phenomenon of paradoxical embolism, given the presence of patent foramen ovale.

The patient was thus re-evaluated in the clinic at 30 days, showing complete blood chemistry tests, which were normal, and negative abdominal objectivity. There is still no surgical indication for the moment and a 30-day abdominal ultrasound is scheduled. After a further 30 days the abdominal ultrasound shows a spleen of 43 × 74 mm hypo-anechogenic; an indication is thus given to laparoscopic or laparotomic splenectomy surgery and the patient is placed on a waiting list. After about 40 days the patient is evaluated by the Anesthesiologist (ASA classification: 4th class, he might need postoperative intensive care) therefore the Surgeon, in consideration of the asymptomatic nature of the clinical picture and the high risks associated with multiple

comorbidities, opts for a Wait & See treatment with instrumental and ambulatory follow-up. The patient is still apyretic, abdominal objectivity is negative, and the laboratory and instrumental follow-up are unchanged.

Discussion

The literature and the "Wait and See" strategy: Will it be the right choice?

The literature concerning the treatment of splenic infarction is quite rare and varied; most are case reports or case series.

The recently published articles are mostly related to the discussion of other pathologies potentially related to splenic infarction [8-9], while articles dealing exclusively with the presentation of splenic infarction and its diagnosis are few articles and not too recent in the international literary panorama [2-7].

Reviewing most of the published papers, the treatment of splenic infarction is medical; surgical treatment can be proposed in case of complications and in any case, unless the presentation is already a complication, it is never an urgent surgical treatment [10,11]. From the data reported in the literature we can deduce that the complication rate in case of splenic infarction is between 20.5 and 34% [2,11], and the main ones are abscess and rupture of the spleen [2,12].

Conservative treatment is to be pursued, at least as a first approach, in all patients who do not present complications at onset; if some of the complications should occur, surgical splenectomy could be performed. In case of complications it is important to perform splenectomy as quickly as possible to further reduce mortality [12]. The conservative treatment in a first phase consists of analgesic, antibiotic and supportive therapy. Furthermore, in cases of splenic infarction, it could be useful to subject the patient to post-splenectomy vaccination as soon as the acute phase of diagnosis and initial medical treatment is resolved.

After a first diagnostic-therapeutic phase, clinical, instrumental and laboratory follow-up is recommended, giving relevance to LDH values [13]. From the instrumental point of view, standard radiological examinations are not very useful in diagnosis, much less in surveillance, the only established method capable of giving consistent answers is abdomen CT with contrast [14]. Clearly it is not possible to periodically subject patients to a higher level radiological examination such as CT, both for costs and time but even more for radiological exposure; therefore, on the basis of our experience, it is useful to intersperse the CT with the Abdomen Ultrasound with the possible use of contrast (type SonoVue) to check the status of the hypoechoic area of the splenic parenchyma.

One aspect that we should consider is the absence in the published literature of a therapeutic and surveillance algorithm that could constitute a sort of "guideline" on the follow-up, on its duration and on the times for the surgery. Once the patient, who has been diagnosed a splenic infarction, even as an occasional finding, is discharged from the hospital, so the initial acute phase has been successfully resolved; the doctor, generally a surgeon, who is in charge of the patient what kind of follow-up is most suitable to propose to him? Clinical and instrumental surveillance? How long after the event is surveillance indicated? What are the risks of the "wait and see" strategy? It would be reductive to answer: "Until a complication arises".

We have already said that the complication rate, in terms of splenic abscess and its rupture, is around 20% to 35% and no one can predict when a hypothetical complication will occur. However, if the patient encounters one of these, the price to pay in terms of mortality and morbidity would be very high with a risk of sepsis of around 12% and mortality of over 50% [15,16]. It is also necessary to say that it would be conceivable that the majority of patients presenting with a splenic infarction, such as the protagonist of our case, are burdened by a series of such comorbidities that even the surgical option could present significant complications.

Now-a-days the first-line surgical approach for splenectomy is undoubtedly the laparoscopic technique that is proposed even in rather complicated cases such as splenic metastasis, splenomegaly or need to change the classical technique with anterior access [17,18]. Laparoscopic or laparotomic splenectomy is in itself burdened by a series of complications such as peri-operative haemorrhage, whose presentation rate is between 3 and 15% but may also be greater depending on physical state and comorbidities of the patient; thromboembolic manifestations with a percentage around 10%, whose correlated mortality rate depends on the body area concerned (several retrospective studies report a percentage of portal thrombosis of 0.7-8%) [19]; and infections such as subphrenic abscesses, empyema or postsplenectomy-related infections such as OPSI (Overwhelming post-splenectomy infection) or OPSS (Overwhelming post-splenectomy sepsis) [20].

OPSI / OPSS are acronyms that indicate a series of fulminating infections that occur in splenectomized patients and that are believed, although it is still a debated topic, to be due to some bacterial serotypes not covered by vaccinations and that represent the greatest long-term risk for these patients. After splenectomy, the life-time risk of OPSI is 0.1-0.5% with a mortality of 50-60% and can occur at any time with a peak incidence within the first three years after the operation, but there they are there are confirmed cases even 20 years after the surgery [20].

The risks are therefore consistent for both options; there are risks related to the waiting conduct as there are risks related to the surgery. Probably there is no better therapeutic strategy regardless; the proposed treatment should be shared with the patient, the possibilities and risks related to each of the two options should be explored. The therapeutic choice must always be tailored on the individual patient, on his comorbidities and carefully balanced both risks. If we decide to opt for the surgical treatment in election, we believe it is surely preferable to have a laparoscopic approach, in fact laparoscopy is not contraindicated in patients with previous splenic infarction [21]. In recent decades, the increasing use of laparoscopic techniques has profoundly influenced the approach to thromboembolic complications. In fact, minimally invasive surgery would seem to be associated with a modest thrombogenic activation of the coagulation system, although surgical times, longer than traditional surgery, would favor the venous stasis of the legs; on the other hand, laparoscopic procedures are characterized by shorter hospital stays and earlier mobilization; considering therefore a balance between the two approaches does not result in a greater incidence of thromboembolic phenomena exclusively due to the laparoscopic technique [21,22]. Despite the implementation of a correct prophylaxis, the residual rate of thromboembolic phenomena in surgical patients is, still today, around an average of 10%, higher for patients undergoing surgery for oncological pathology, without a different incidence rate of thromboembolic events among patients undergoing laparoscopic or laparotomic surgery [22-24].

Conclusion: A Tailored Strategy

Currently the literature is rather scarce regarding the treatment and follow-up of splenic infarction especially in the long term. Most scientific papers consist of case reports and case series. Diagnosis in

patients with suspected splenic infarction is difficult, complicated by the fact that such patients often present numerous comorbidities also associated with the age factor. An accurate history, with attention to symptoms attributable to hematological or rheumatologic / vasculitic pathologies, and the decision to subject the patient to abdomen CT represent the two key points for a correct diagnosis of splenic infarction. Thanks to a review of the literature it was possible to infer that for uncomplicated splenic infarcts (the main complications are represented by splenic abscesses and rupture of the spleen) the treatment of choice is the medical conservative one, the surgical approach is reserved exclusively in case of complications. However, many doubts remain. First, it is not clear how to proceed with the follow-up, the timing and the modalities; or if, on the contrary, propose to the patient the surgical option, and even if in this case too, the doubt about the timing remains.

Surgical treatment and the waiting conduct (wait and see) are two antipodal options; the decision to take one of the two roads exposes the patient, on the one hand, to the risks associated with the surgical act, which could also be extremely high in view of the physical state of the patient and his comorbidities, on the other hand, to the risks involved to the possible onset of complications such as abscess and rupture of the spleen. In the clinical case we presented, the patient underwent initial abdominal CT and subsequently abdominal ultrasound, blood chemistry and ambulatory serial evaluations after a few months. The patient was, in any case, subjected to a preoperative anesthesia evaluation, but in consideration of the high surgical and anesthetic risks and the stability of the clinical situation, in agreement with the patient, it was decided to continue with the conservative follow-up.

Both strategies have risks related to unpredictable morbidity and mortality rates. In our opinion the treatment cannot, in the current state of scientific evidence, be standardized; but it must be weighed on the individual patient according to his comorbidities, informing him of any connected risks. However, it is desirable to have, in the future, more studies with a greater number of patients and the possibility of comparing the two therapeutic strategies so as to be able to draw up a standardized treatment for the approach to patients with finding uncomplicated splenic infarction.

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