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REVIEW

Management of splenic and pancreatic trauma

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Splenopancreatectomy;
Pseudocyst;
Review

Summary The spleen and pancreas are at risk for injury during abdominal trauma. The spleen is more commonly injured because of its fragile structure and its position immediately beneath the ribs. Injury to the more deeply placed pancreas is classically characterized by discordance between the severity of pancreatic injury and its initial clinical expression. For the patient who presents with hemorrhagic shock and ultrasound evidence of major hemoperitoneum, urgent ''damage control'' laparotomy is essential; if splenic injury is the cause, prompt ''hemostatic'' splenectomy should be performed. Direct pancreatic injury is rarely the cause of major hemorrhage unless a major neighboring vessel is injured, but if there is destruction of the pancreatic head, a two-stage pancreatoduodenectomy (PD) may be indicated. At open laparotomy when the patient's hemodynamic status can be stabilized, it may be possible to control splenic bleeding without splenectomy; it is always essential to search for injury to the pancreatic duct and/or the adjacent duodenum. Pancreatic contusion without ductal rupture is usually treated by drain placement adjacent to the injury; ductal injuries of the pancreatic body or tail are treated by resection (distal pancreatectomy with or without splenectomy), with generally benign consequences. For injuries of the pancreatic head with pancreatic duct disruption, wide drainage is usually performed because emergency PD is a complex gesture prone to poor results. Postoperatively, the placement of a ductal stent by endoscopic retrograde catheterization may be decided, while management of an isolated pancreatic fistula is often straightforward. Non-operative management is the rule for the trauma victim who is hemodynamically stable. In addition to the clinical examination and conventional laboratory tests, investigations should include an abdominothoracic CT scan with contrast injection, allowing identification of all traumatized organs and assessment of the severity of injury. In this context, non-operative management (NOM) has gradually become the standard as long as the patient remains hemodynamically stable and there is no suspicion of injury to hollow viscera, with the patient being carefully monitored on a surgical service. The development of arteriography with splenic artery embolization has increased the rate of splenic salvage; this can be performed electively based on specific indications (blush on CT, pseudoaneurysm, arteriovenous fistula), and may also be considered for severe splenic injury, abundant hemoperitoneum, or severe polytrauma. For pancreatic injury, in addition to CT scan, magnetic resonance pancreatography (MRCP) or even endoscopic retrograde cholangiopancreatography (ERCP) may be necessary to identify a ductal

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rupture. If the pancreatic duct is intact, laboratory and CT imaging surveillance is performed just as for splenic injury. In case of pancreatic ductal injury, ERCP stenting can be considered. However, if this is unsuccessful, the therapeutic decision can be difficult: while NOM can still be successful, complications may arise that are difficult to treat while distal pancreatectomy, although initially more agressive may avoid these complications if performed early.

Introduction

Pancreatic or splenic injury is an indicator of the severity of abdominal trauma.

The spleen, an encapsulated and fragile organ, is the most commonly injured solid organ in blunt abdominal trauma [1], and this is a potentially serious event. In the 1950s, there was a marked surge of splenectomy for ruptured spleen because it was thought to be a lifesaving gesture that reduced mortality. With the growing awareness that splenectomy resulted in an increased risk of grave infections whether in the pediatric [2] or the adult patient [3], the number of splenectomies for trauma has steadily declined in favor of non-operative or splenic-preserving treatment [4,5], abetted by the use of more precise methods of exploration and surveillance (Fig. 1). Mastery of initial non-operative management (NOM) includes selective use of a timely deferred laparoscopy, awareness of surgical methods of total or partial splenic conservation, although the implementation of arterial embolization [6] has tended to supplant this option. Currently over 60% of splenectomies in France are performed outside the traumatic context (hematologic reasons, benign or malignant splenic tumors, contiguous invasion by adjacent cancers, and even ''accidentally'' during surgical procedures involving the esophagus, stomach or colon). Whatever the context, splenectomy significantly increases the risk of postoperative infectious complications [7].

Pancreatic trauma (PT) occurs in 3—5% of blunt abdominal trauma in the adult [8], and less than 1% of trauma admissions in children [9]. PT is rare but can be fatal. Associated duodenal injury must be considered in the same setting because its intimacy. Because of this organ's anatomical complexity, its intimate interrelations with neighboring organs, and the unique risk of injury evolving into acute pancreatitis (AP), there are multiple therapeutic options and making the appropriate choice is oftentimes difficult. NOM for PT became feasible when improvements in imaging by CT and MRI enabled detection of pancreatic duct rupture, and to a lesser extent by the development of interventional therapies such as ERCP and ductal stenting for treatment of these fractures [10,11]. Nevertheless, open laparotomy with possible pancreatic resection still retains an important place in the therapeutic arsenal of these injuries.

Splenic trauma

Classification

Although established some time ago, the American association for the surgery of trauma (AAST) classification of splenic

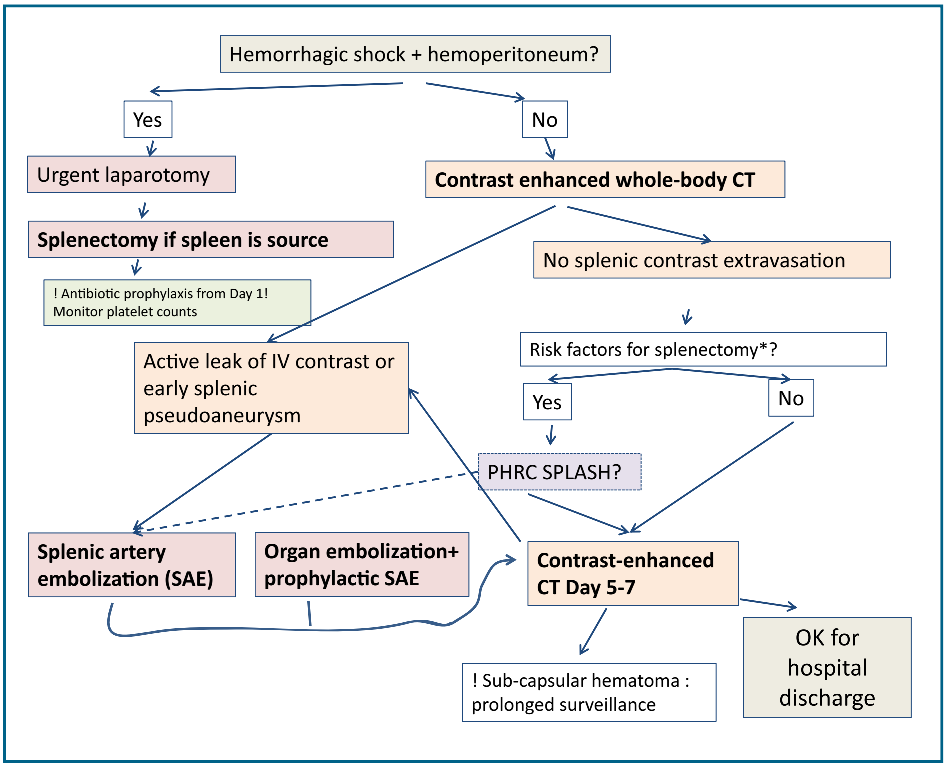

Figure 1. Decisional flowchart for initial management of splenic trauma. *: massive hemoperitoneum and/or multiple trauma.

Table 1 American association for the surgery of trauma [AAST]) classification of splenic trauma [14].

Grade	Subcapsular hematoma	Intraparenchymal hematoma	Capsular tear	Devascularization
I	< 10% splenic surface Non expanding	None	Depth < 1 cm Non hemorragic	0
II	Surface 10—50% Non expanding	Diameter < 5 cm Not progressing	Depth 1—3 cm Bleeding does not involve the trabecular vessels	0
III	Surface > 50% or rupture or expanding or bleeding	Enlarging or diameter > 5 cm	Depth > 3 cm Bleeding involves the trabecular vessels	< 25%
IV		Ruptured	Bleeding involving the segmental or hilar vessels	> 25%
V		Splenic avulsion	Hilar lesion(s) resulting in complete devascularization of the spleen	100%

Table 2 Baltimore classification of splenic trauma taking into account the severity of vascular lesions [15].

Grade	Subcapsular hematoma	Intraparenchymal hematoma	Capsular tear	Other
1	Thickness < 1 cm	Diameter < 1 cm	Depth < 1 cm	
2	Thickness 1—3 cm	Diameter 1—3 cm	Depth 1—3 cm	
3	Thickness > 3 cm	Diameter > 3 cm	Depth > 3 cm	Rupture of splenic capsule
4a		Splenic avulsion		Active bleeding-subcapsular and intraparenchymal
4b				Active bleeding-intraperitoneal

trauma (originally described in 1989 by Moore et al. and amended in 1994) [12,13] is still widely used in practice. There is a great difference between splenic trauma classified AASTI-II and the more serious class III-V injuries (Table 1) [14]. A new classification of splenic trauma proposed by Marmery et al. [15] seems very interesting because of its simplicity, taking into account the presence of active bleeding on CT (''blush'' or extravasation of contrast material) (Table 2), which constitutes the major prognostic factor.

Techniques and therapeutic options

Severe injury evident at the outset: extremely urgent laparotomy

Diagnosis and initial management

The clinical presentation is clearly that of an important and extremely serious hemoperitoneum: the patient is in severe hypovolemic shock and fails to respond to volume resuscitation during transport, sometimes even requiring cardiopulmonary resuscitation at the accident scene, and arrives with no detectable blood pressure and a distended fluid-filled abdomen. Signs suggestive of splenic trauma may include left rib fractures or a seat-belt bruise on the left side. If the patient has isolated and obvious trauma to the left upper quadrant, hemoperitoneum and acute splenic injury should be confirmed by abdominal ultrasound performed while volume resuscitation is underway: the patient must be brought immediately to the operating room and be prepared — on the operating table — for immediate laparotomy while fluid resuscitation is completed. Operative delay is the best identified risk factor for ''preventable deaths'' [16] since delay in surgery allows the patient to descend into the vicious circle of coagulopathy (due to hemodilution, consumption of coagulation factors and fibrinolysis), acidosis and hypothermia [17,18]; this will very quickly lead to hemorrhagic syndrome and irreversible organ failure. As in any patient with serious trauma, prevention of coagulopathy is a constant concern of the care team, requiring a set of specific measures (external warming, volume repletion, vasopressors, and transfusion of packed RBC's, platelets and clotting factors). In the most serious cases, one should anticipate the risk of cardiac arrest (especially if cardiorespiratory resuscitation (CPR) has been initiated at the scene of the accident, during transport or in the emergency room), which may develop when the abdomen is opened (in this case, an intra-aortic balloon can be positioned under C-arm fluoroscopy. Once the abdomen is opened, the subxiphoid aorta should be manually compressed, followed by cross-clamping of the aorta above the celiac axis.)

''Damage control'' laparotomy

A wide but rapid skin preparation is followed by a midline incision, and the imperative indication for total splenectomy is confirmed if the spleen is the cause of the massive bleeding, as part of the surgical rescue strategy [19,20]. Splenectomy can be technically difficult, especially in obese patients with massive hemoperitoneum, when there are multiple hemorrhagic foci, or when the spleen is pathologically enlarged or fragmented. The two main surgical risks are accidental injury to the colon at the splenic flexure or to the tail the pancreas. Such accidental injury can be largely avoided by proper patient positioning and splenectomy technique, with immediate *en bloc* mobilization of the distal pancreas and spleen. The operator stands to the right, and only a single assistant is necessary if a left subcostal retractor can be affixed to the table. Use of a

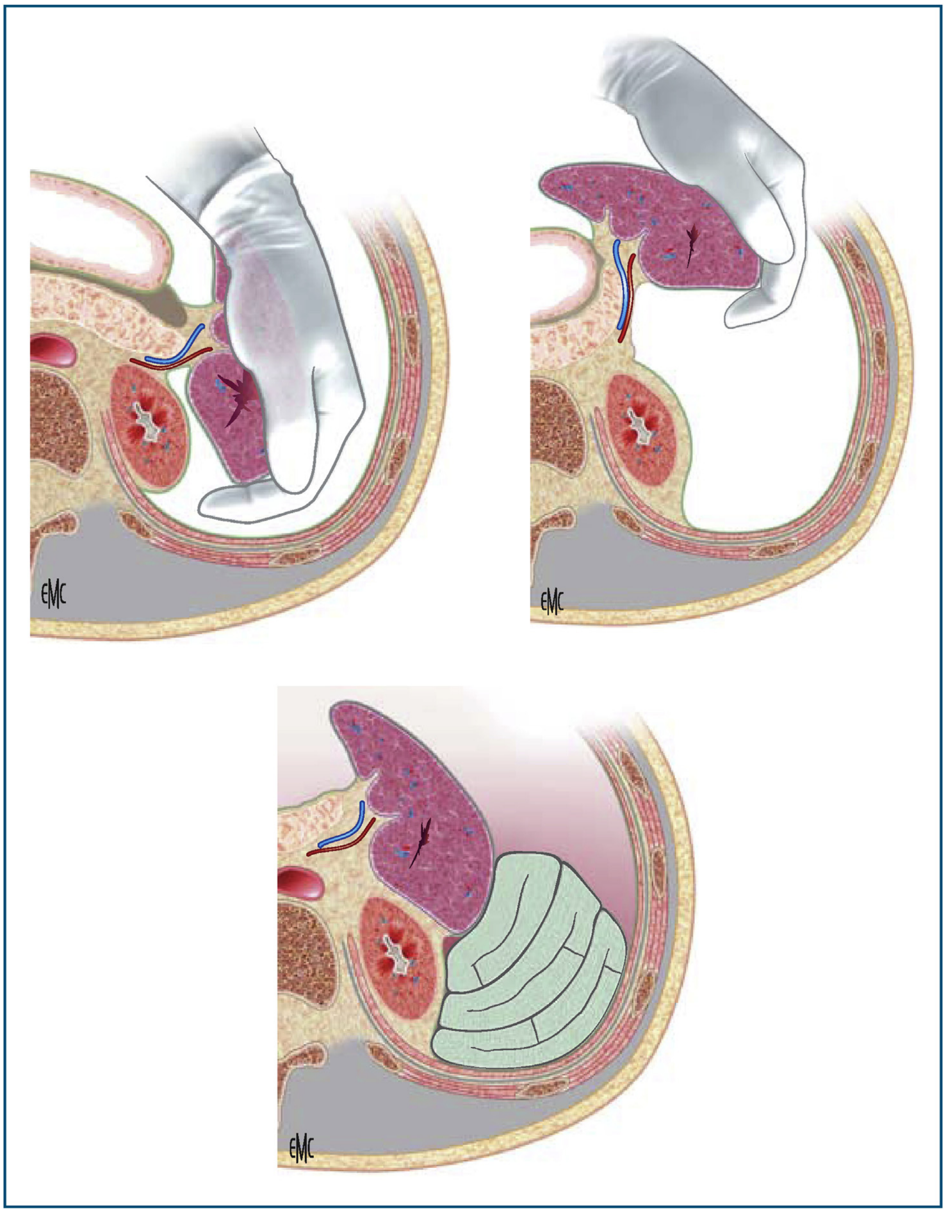

Figure 2. Operative exposure for urgent splenectomy.
Extract to: Arvieux et al. [86].

LigaSure®-type coagulating device will shorten the operative time and reduce the risk of gastrointestinal injury or secondary bleeding. The most effective gesture, even on a severely fractured spleen, is for the surgeon to encompass the spleen with the left hand and compress it against the spine while the right hand uses a sponge clamp to pack three or four laparotomy pads into the gutter, all the while maintaining gentle traction toward the midline and allowing incision of the posterior parietal peritoneum (Fig. 2). This will achieve temporary hemostasis in most cases while providing better exposure as the spleen is reflected medially and superficially. To mobilize the upper pole, the peritoneum is incised flush with the spleen, taking care not to damage the short gastric vessels along the greater curvature. At the lower edge of the gastrosplenic ligament, the gastro-epiploic vessels are divided opening the lesser sac and allowing direct access to the splenic pedicle. Following completion of splenectomy, a drain should be placed to externalize any eventual pancreatic fistula (PF).

Complications of splenectomy

Early postoperative complications

We must distinguish early postoperative complications from the long-term risk of overwhelming post-splenectomy infection that starts on the day of splenectomy and persists throughout life [3]. Early technical complications occur after 2—7% of splenectomies for trauma [21]. The most common is PF, followed by bleeding at the surgical site, subphrenic abscess, caudal pancreatitis and gastrointestinal fistula. Technical complications occur more commonly when there is an intra-abdominal contamination due to intestinal spillage and in the elderly [5]. Thrombocytosis (defined as a platelet count greater than 400,000 platelets/μL) typically occurs in > 70% cases after splenectomy, regardless of the indication. Platelet counts generally increase from day 4 to day 6, peak between the second and third postoperative weeks, and return to normal within 2 months in most cases [22]. Portal vein and/or superior mesenteric vein thrombosis is a feared complication in the splenectomized patient. It is often asymptomatic and occurs early during the first few postoperative days. In a prospective study [23] of 147 splenectomized patients who underwent extensive search for thrombosis by Doppler imaging, a thrombus of the mesentericoportal axis was found in 4.8% of cases. Risk factors are a platelet count greater than 650,000/μL, the existence of a myeloproliferative disorder, splenomegaly, and a long splenic vein remnant [24]. In practice, current thrombosis prophylaxis in splenectomized patients is based on the administration of low molecular weight heparin (LMWH) and platelet aggregation inhibitors (low-dose aspirin/100—250 mg daily) [22]. As with any surgical patient, prophylaxis with LMWH should be prescribed for any ''high risk'' patients with a high probability of developing lower limb venous thrombosis due to their history (hematologic disease, past history of phlebitis, smoking, obesity, estrogen-progestin medications, etc.) Prophylaxis should continue as long as the platelet count exceeds 650,000/μL. For significant thrombocytosis (> 1,000,000/μL), an antiplatelet agent should be combined with LMWH. Finally, for any symptom suggestive of a thromboembolic complication, a Doppler examination and CT scan with IV contrast are imperative in all splenectomized patients.

Infectious complications

The risk of infectious complications alone justifies the need to avoid total splenectomy whenever possible. The spleen is the largest lymphoid organ of the human body and is actively involved in the organization of the immune response [25]. To perform its functions effectively, the spleen must maintain a physiological vasculature and a sufficient volume of parenchyma, the ''critical mass'', estimated to be between 30 and 50% [26]. After splenectomy, alterations of immune function, more severe in patients who are young or suffering from other diseases (cancer, immunosuppression, radiation, chemotherapy, diabetes, alcoholism, etc.) [27], may result in overwhelming post-splenectomy infection (OPSI). In children up to the age of 15, there is a marked risk of OPSI in the order of 4%, with the infection most often manifested by meningococcal or streptococcal meningitis [22]. In adults who have had a splenectomy for trauma, the risk is lower: 4/10,000 person-years with a lifetime risk of OPSI of 1—4 per 1000 [27]. Neither post-traumatic splenectomy splenosis, accessory spleens, nor autotransplantation are universally protective since authentic OPSI has occurred despite the presence of accessory spleens. OPSI is manifested clinically by a day or two of flu-like prodromal symptoms, with eventual abdominal pain and onset of fulminant septicemia with progressive purpura progressing to multi-organ failure resulting in death within 24—48 hours in more than half of patients. The interval from splenectomy to onset of fulminant infections is variable, but remains a lifelong possibility: 50% of deaths occur within 3 months after surgery and another 28% during the first 3 years, but one case of OPSI has been described that occurred more than 60 years after splenectomy [28]. Splenectomized patients also have a higher risk of serious complications from malaria. These facts, combined with the variable effectiveness of pneumococcal vaccine (because the wide variety of strains are not all included in the vaccine), is the source of updated recommendations promulgated by French Society of Anesthesia Resuscitation (SFAR) in 2013 [29] that reinforce the conclusions of a 2011 British consensus conference:

- antibiotic prophylaxis started on the first postoperative day followed by oral penicillin treatment for 2 years;
- vaccination against pneumococcus, haemophilus influenzae, and meningococcus with, probably in the near future, the development of routine assays of specific antibodies that will inform the need for reminders to update these three vaccinations [29];
- influenza vaccination recommended annually because of the risk of secondary bacterial infections;
- prescription of an empirically appropriate antibiotic for prophylactic therapy in patients with febrile syndrome.

In reality, the area where genuine progress for prevention of OPSI is most easily achievable is undoubtedly is the therapeutic education of splenectomized patient and provision of information to referring physicians. Two recent surveys in Switzerland and Ireland [30] showed that less than 75% of patients who underwent splenectomy for trauma were actually vaccinated, fewer than 60% had a prescription for antibiotics, and less than 50% of the patients knew their asplenia entailed a risk of life-threatening infection. In practice, only a minority of post-splenectomy patients had correct information concerning the role of vaccines and antibiotics. We conducted a study concerning follow-up and provision of information to 40 patients who underwent splenectomy on our service between 2004 and 2013; in 2003, we had established a protocol for post-splenectomy information with a booklet for the patient and systematic information of the primary care physician. We showed that this resulted in a doubling of the observance of recommended prophylaxis, which nevertheless remained below 60%; this shows that it is essential to implement therapeutic education programs adapted to a population that is often young and without regular medical care.

Severe traumatic injury without shock

Initial management

A rapid initial clinical examination must look for signs of respiratory circulatory or neurological distress triggering adapted emergency treatment. A chest X-ray and abdominal ultrasound are performed while monitoring and resuscitation are started; the effectiveness of volume resuscitation to reverse hypovolemia is a key element to decide if operation is needed. The clinical examination is the same as for any serious trauma: neurological examination, auscultation, assessment of peripheral pulses, a search for posterior wounds, abdominal palpation and examination for suggestive skin lesions (hematoma, seat belt bruising), assessment of pelvic stability, examination for extremity injury or deformity. Insertion of at least one large-bore peripheral intravenous catheter allows a blood draw for laboratory testing; subsequently, a central venous catheter and possibly a radial artery catheter to monitor blood pressure should be placed if major blood loss is apparent. The patient should undergo endotracheal intubation and assisted ventilation if this seems necessary to ensure adequate analgesia and sedation. The prevention and/or correction of hypothermia are fundamental and rely on accurate measurement of core temperature (which must be maintained at $\geq 35\,^{\circ}C$), with warming of all infusion fluids and of ventilation gases, and use of heating blankets. Bladder catheterization is performed after abdominal ultrasound and digital rectal exam have ruled out urethral trauma. The measurement of urine output is an essential part of the resuscitation, and measurement of intracystic pressure allows monitoring of the intra-abdominal pressure.

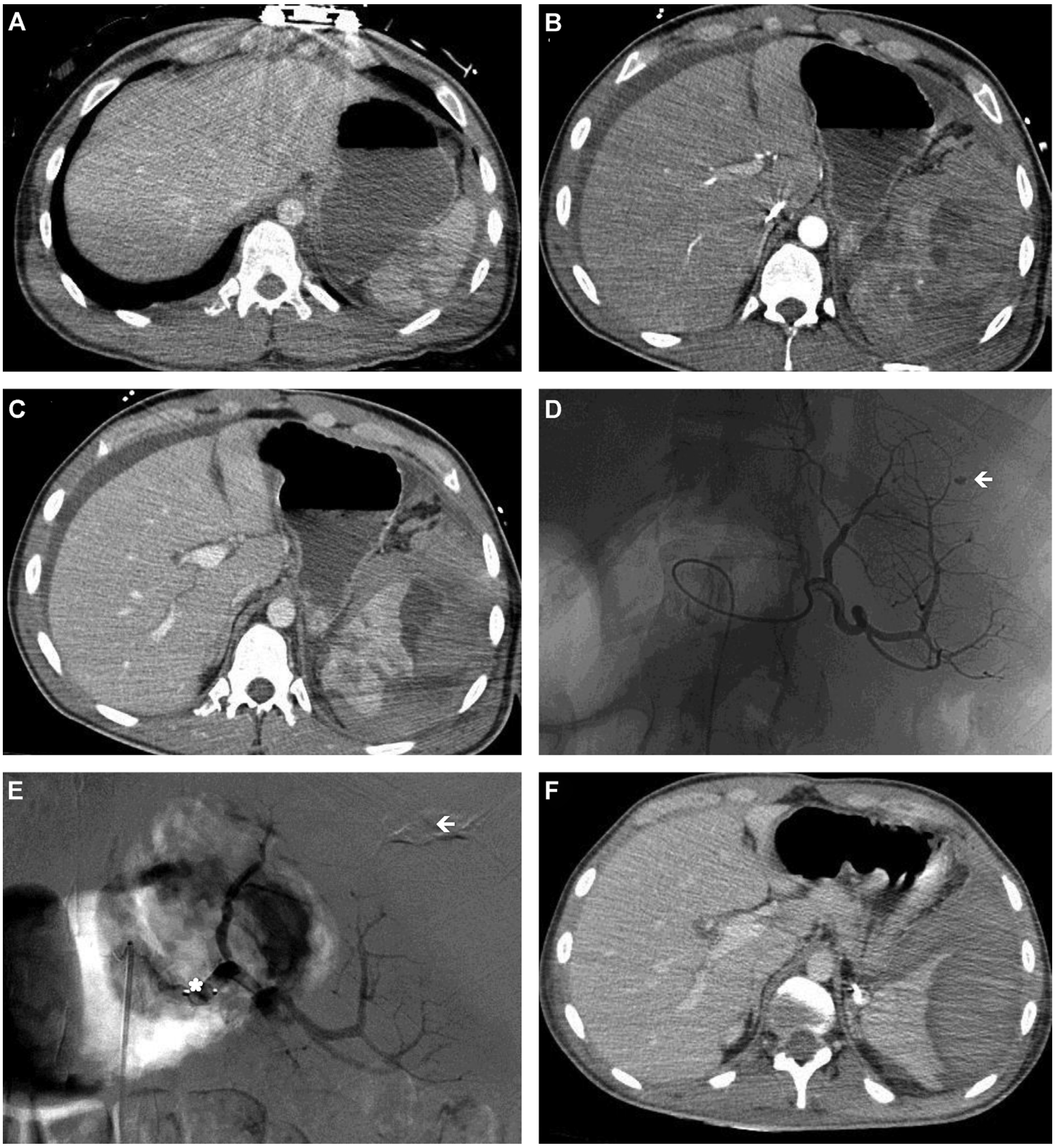

Figure 3. A 24-year-old male patient with no prior history fell from 20-m height while riding an all-terrain vehicle; vital signs were stable when patient presented to the emergency room. Multiple trauma noted on contrast-enhanced CT scan; multiple rib fractures, pneumothorax, grade 3 splenic trauma with several parenchymal lacerations, moderate-sized hemoperitoneum, but without ongoing bleeding or contrast extravasation [3A], fracture of the right acetabulum and femoral diaphysis. Patient monitored in the ICU and then managed by the orthopedic surgery service. The patient's vital signs, clinical exam and hemoglobin were initially stable. A CT scan on day 5 showed no progression of the splenic lesions. On the 9th day, the patient had acute onset of severe abdominal pain with an increase in abdominal girth and a fall in hemoglobin/hematocrit. Urgent CT scan showed: several leaks of contrast in the arterial phase from the region of the splenic hematoma [3B] with a marked increase in the hemoperitoneum in the portal phase [3C]. The patient was transferred to the ICU for management of hemorrhagic shock and urgent angiography with splenic artery embolization. Arteriography revealed several very localized contrast extravasations, one of which was sizeable and associated with a more rounded image (arrow) [3D]. Hyperselective embolization of this arterial branch with a fragment of Curaspon® gelatin sponge (arrow), followed by proximal arterial embolization with a plug were carried out successfully (asterisk) [3E]. The patient's clinical and laboratory course were good. Follow-up CT scan on the 24th day post-embolization [3F] showed a well-vascularized parenchyma with regression of the splenic hematoma.

Complementary tests

Whole body CT scan with IV contrast injection is the absolute rule in all severely traumatized patients whose vital signs can be stabilized (Figs. 1 and 3). Abdominothoracic CT should be performed after CT of the head and neck. CT is a very powerful tool because it provides information on the volume of peritoneal effusion, the other abdominal organs and diaphragm, and the spleen itself, allowing assessment of the gravity of organ injury. Vascular phase images help to identify persistent bleeding evidenced by extravasation image or blush, which may be an indication for angiography and embolization.

Decision to perform urgent laparotomy

A decision to proceed to laparotomy is based on several criteria that may be associated: frank evidence of peritonitis or the discovery of a gastrointestinal perforation, persistent or secondary hemodynamic instability requiring continued volume resuscitation, particularly if the patient is in poor general condition (advanced age, cirrhosis, anticoagulation). The feasibility of arterial embolization also weighs on the decision for surgery in the polytrauma patient with an actively bleeding splenic injury who requires another urgent surgical procedure (typically a spinal fracture with spinal cord compression that requires prolonged prone operative positioning).

With the rise of embolization, splenic repair techniques have tended to decrease since they were typically performed in patients who are relatively stable hemodynamically and are currently the best candidates for a radio-interventional gesture. However, conservation techniques deserve to be described because they still afford a chance of splenic salvage and are particularly useful for accidental splenic injury during another surgery. Biological materials and adhesives have some interest especially for AAST grades I and II splenic lesions [13]. They can also help to improve surface hemostasis of the resection margin after partial splenectomy. Collagen derivatives are contraindicated if cell-saving autologous transfusion is being used (risk of microthrombi). Splenorraphy has been practically abandoned in trauma surgery because of the risk of aggravating the original injury. Partial splenectomy requires total mobilization of the spleen to completely evaluate the topography of the lesions, it is particularly suitable if the anatomy is favorable and the trauma involves only one of the splenic poles. Transection is then performed along the line of demarcation between vascularized and ischemic parenchyma, using electrocautery or cold knife, or even better if possible, application of a TA-55 or GIA stapler if the spleen is sufficiently thin. A bio-absorbable mesh prosthetic screen with a series of pursestring sutures can be wrapped around the spleen and tightened down to apply hemostatic compression to the parenchyma while avoiding compression of the pedicle; its use is probably associated with an increased postoperative infection risk since experimental data have suggested that there is a higher risk of abdominal infections in animals with perisplenic mesh [31]. Autotransplantation has fallen out of use because it has been shown that keep a physiological vasculature, with central arteries and portal venous drainage is necessary to ensure effective splenic function [32]). We have identified only two cases of splenic repair in our series on 336 cases of splenic trauma.

Hemodynamically stable patient

Non-operative management (NOM)

NOM has seen a steady increase in use. It allows the best chance for splenic salvage and avoids many of the postoperative complications of splenectomy. It avoids an invasive surgical procedure and the need for lifelong preventive measures against OPSI. In children, more than 85% of splenic injuries undergo NOM, and surgical treatment of children is most often performed because of associated injury of hollow viscera [4]. In adults, the Eastern association of surgery for trauma (EAST) multicenter retrospective study published in 2000 reported the experience of 27 US trauma centers involving 1448 cases of blunt splenic injury in patients older than 15 years [33]. In this large series, 62% of patients had initial NOM; the success of the NOM approach decreased progressively depending on the severity of the grade of splenic injury: respectively 75%, 70%, 49%, 17% and 1% for AAST grades I, II, III, IV and V.

Splenic artery embolization (SAE)

Because of the high rate of late secondary splenic rupture [33] (Fig. 3), embolization has experienced a major boom in trauma practice. [34]. In the series by Gaarder et al. [6], the overall rate of splenic salvage increased from 57% to 75% thanks to SAE. Published series of splenic embolization (therefore performed only on patients who were not in hemorrhagic shock) revealed a splenic salvage rate approaching 90% [6,35,36]. According to the review by Olthof et al. established using the Delphi methodology, the current indications for SAE include [37]):

- CT evidence of extravasation of contrast material;
- pseudoaneurysm and arteriovenous fistula;
- higher AAST grades of injury (III?)-IV-V;
- massive hemoperitoneum;
- severe multiple trauma.

In France, a consensus of opinion favors SAE if contrast leakage is detected within the splenic parenchyma in a stable or stabilized patient, or if pseudoaneurysm develops. The other indications are more debatable since the procedure performed is usually a prophylactic embolization of the proximal splenic artery [36]. There is an ongoing hospital clinical research program (PHRC) nationwide study to evaluate whether the benefits outweigh the risks of SAE, since its rapid adoption has exposed a variety of associated problems. In actuality, not all trauma centers in France have 24-hour availability of an experienced radiologist and an equipped interventional radiology room adjacent to their trauma-receiving center. SAE can be time consuming and poses a risk of falling into irreversible hemorrhagic shock during transportation or radiological intervention. Careful patient selection for this procedure is therefore imperative. Moreover, even though the majority of studies show a priori retention of immunological function of the embolized spleen [35—38], the ability of SAE ability to fully preserve the immunological function of the spleen is not certain. Finally, SAE has a complication rate of approximately 20% in some historical series [21,39,40]. The most frequent complications were persistent hemorrhage, splenic abscess or infarction, the migration of embolized coils, thrombosis of the vascular access site, wound infection at the access site, contrast agent-related renal failure, and late pancreatic pseudocyst. However, in two recent series from expert centers reporting the follow-up of than 250 cases of splenic trauma [36,41], the rate of severe SAE-specific complications was less than 4% and the technical success rate was greater than 96%.

Surveillance after splenic trauma

The risk of delayed secondary splenic rupture requires close monitoring of patients in a surgical environment, whose duration depends on the severity of the lesions observed and the patient's home environment. Several authors have emphasized that a large part of the published cases of so-called secondary rupture were actually hemorrhagic lesions that had gone unnoticed initially due to clinical context: addicted patients presenting several days post-trauma, patients whose multiple trauma made surgical exploration difficult, etc. [42]. In fact, true secondary ruptures are almost exclusively due to rupture of a subcapsular hematoma [43], or early rupture of pseudoaneurysms. The maximum risk is in the first days after the injury and then slowly declines: 92% of secondary fractures take place in the first 6 days after the accident [33]. This explains why in the USA, where hospitalization is generally shorter than in France, the average interval to secondary rupture is 6 days. One study has described 12 well-documented cases of secondary splenic rupture with normal initial CT scan [43], and we found three cases in our series with similar characteristics. For these 15 patients, the time delay from injury to rupture was between 2 and 45 days after the accident (2—7 days in 5 patients, 10—14 days in 5 patients, and 21—45 days in 5 patients). The initial splenic lesion can go undetected by CT because of radiologic artifacts (e.g., rib fractures) when the CT is performed shortly after the accident or when the timing of splenic imaging was not coordinated with contrast injection. Because of this, for any post-traumatic hemoperitoneum, even of low volume, one should not hesitate to order a repeat CT scan in search of a splenic injury that had been undetected on the initial abdominal ultrasound or CT scan, since there is a finite risk of delayed splenic rupture. In France, the period of surveillance after splenic trauma varies among different trauma teams and according to the severity of the injury but averages around 15 days. The previously cited studies suggest that earlier hospital discharge, as early as the 7th day, may be feasible, as long as an interval CT scan with IV contrast is performed prior to discharge to rule out delayed subcapsular hematoma or early pseudoaneurysm and the patient is properly informed and has sufficient support at home to allow prompt return to the hospital in case of emergency.

It should be noted that current splenic trauma-related deaths are largely due to delayed laparotomy in patients with a deceptively reassuring clinical picture, a lack of surveillance or in whom the abdominal trauma had gone unnoticed [42]. When NOM of splenic trauma is decided, it is imperative to consider the possibility of other associated intra-abdominal injuries; although this is a rare event (2%), the risk of associated intestinal injury is doubled when there is combined injury of both the spleen and the liver [44,45]. The most frequently associated intra-abdominal injuries involve the diaphragm and pancreas. In a patient who presents with a picture of delayed abdominal pain, the possibility abdominal compartment syndrome (ACS) must also be considered, even if though this occurs much less commonly with splenic trauma than with liver trauma [46], since at least 10% of patients with high-volume hemopertitoneum have severely elevated intra-abdominal pressure. Although the risk of decompensation towards a genuine ACS is low, it justifies the measurement of intravesical pressure (IVP) when in doubt. In cases of poorly tolerated ACS manifested by significant pain, oliguria, azotemia and ventilatory difficulties, surgical exploration to evacuate hemoperitoneum should be performed. The patient's clinical and CT status and should be closely monitored in a surgical environment or ICU, with surgical exploration for the slightest doubt by either laparotomy or laparoscopy [47].

Late complications

The most common late complications after NOM for splenic trauma are:

- splenic pseudocyst (SPC);
- pseudoaneurysm;
- arteriovenous fistula (AVF) [1]:
  - post-traumatic SPC is a classical complication in children, with an incidence of 5—10% [4] and with spontaneous reabsorption observed in half of the cases; pseudocyst resection with epiploplasty via laparotomy or laparoscopy is required for the other half of the cases. SPC is relatively rare in adults but its frequency has increased with the increasing use of NOM while spontaneous pseudocyst reabsorption occurs in only one of six cases. Radiologically-guided percutaneous drainage is both ineffective and often a source of complications, especially when subsequent surgery will be needed anyway [48]. Symptomatic and/or non-regressing SPC should be treated surgically, ideally by laparoscopy, performing a partial splenectomy or resection of the prominent dome of the pseudocyst [49]. A total splenectomy may be required if dissection is difficult particularly after failed radiological drainage and/or partial splenic infarction causing a significant local inflammatory reaction,
  - splenic pseudoaneurysm has already been described in the early complications. It has an estimated incidence of 5—13% in adult patients who undergo NOM. It may result in delayed secondary splenic rupture from day 1 out to 4 months. Although the pseudoaneurysm may thrombose spontaneously, the risk of secondary rupture with severe consequences justifies systematic performance of a CT with IV contrast ± embolization prior to patient discharge,
  - splenic arteriovenous fistula (AVF) is a rare condition representing < 5% of indications for splenic embolization [40]. The origin of AVF can be congenital or acquired. Acquired AVF occur due to post-traumatic or spontaneous rupture of a splenic pseudoaneurysm [50]. Symptoms suggestive of splenic AVF include abdominal pain and diarrhea [51]. The full-blown clinical picture of untreated AVF may include a pulsatile abdominal mass with portal hypertension and/or heart failure. Abdominal CT and angiography allow early diagnosis and treatment before clinical symptoms develop, but technical difficulties can make embolization difficult with a higher failure rate than for aneurysm embolization, and may require splenectomy [40].

Pancreatic trauma

Classification

Lesion classification allows evaluation and comparison of the various methods of treatment, and also codification of the therapeutic approach. The clinical severity of pancreatic trauma (PT) clearly correlates with injury to the pancreatic duct. The most commonly-used classification is that proposed by the American association for surgery of trauma (AAST) [52] (Table 3). Lucas has described another classification [53] that has the advantage of taking into account associated duodenal injury, whose treatment is closely linked to the pancreatic injury (Table 4).

Techniques and therapeutic decisions

The diagnosis of PT may be made either intra-operatively, when urgent open laparotomy is required in the setting of hemodynamic instability or peritonitis, or non-operatively, when the patient's condition does not require immediate laparotomy, allowing time for diagnostic studies.

Operative diagnosis and treatment

Two different situations may be encountered intra-operatively: in one case, the PT is only one constituent in a tableau of multiple trauma requiring abbreviated damage-control laparotomy, and in the other case, rapid stabilization allows full exploration of the pancreatic injury, and allows the choice of the most appropriate treatment (Fig. 4[b]).

Table 3 American association for the surgery of trauma [AAST]) classification of pancreatic trauma [52].

Grade[a]	Hematoma	Laceration	AIS score
I	Minor contusion without ductal injury	Superficial laceration without ductal injury	1
II	Major contusion without ductal injury and without tissue loss	Major laceration without ductal injury and without tissue loss	2
III		Transection of the distal pancreatic duct or parenchymal injury with ductal injury	3
IV		Transection of the proximal pancreatic duct or parenchymal injury with proximal duct injury[b]	4
V		Massive injury of the pancreatic head	5

AIS: Abbreviated injury scale.
[a] Add one grade if there are multiple injuries of the same organ.
[b] To the right of the patient's superior mesenteric vein.

Table 4 Lucas classification of pancreatic trauma [53].

Class	Pancreatic lesion	Wirsung duct	Duodenum
I	Contusion or pancreatic laceration with limited parenchymal damage	Intact	Intact
II	Laceration, perforation or complete transection of the pancreatic body or tail	Disrupted	Intact
III	Crush, perforation or complete transection of the pancreatic head	Disrupted	Intact
IVa	Limited injury of the pancreatic head	Intact	Injured
IVb	Severe pancreatic injury	Disrupted	Injured

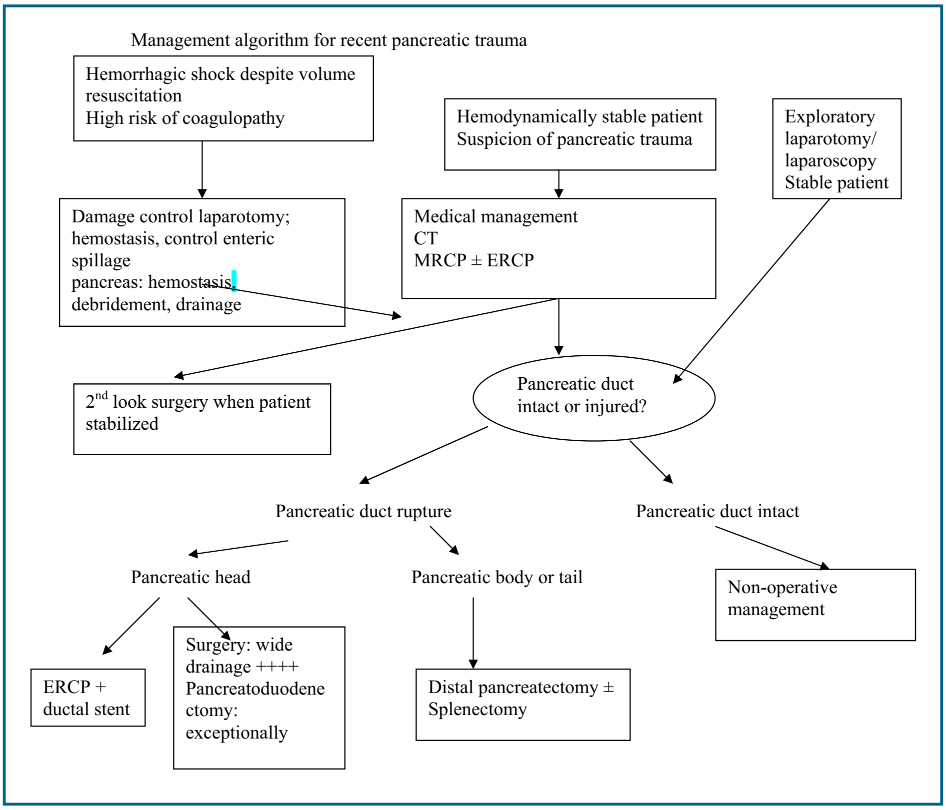

Figure 4. Decisional algorithm: management steps when pancreatic injury is found at open laparotomy. PD: pancreatoduodenectomy; CT: computed tomography scan: MRI: magnetic resonance imaging; ERCP: endoscopic retrograde cholangiopancreatography.

When damage-control laparotomy is necessary

Patients with the most serious injuries arrive in a state of refractory shock with obvious hemoperitoneum; they should be brought immediately to the operating room while continuing volume resuscitation. If the surgeon must perform a damage-control laparotomy, shortened because of coagulopathy, complex pancreatic surgical gestures are prohibited, and simple pancreatic drainage with packing of the pancreatic bed is the most appropriate choice. In most cases, injury to the peripancreatic vascular structures (Henle's gastrocolic trunk, etc.) is the source of bleeding and these injuries should be treated according to their location and severity by ligation, suture repair or bypass. However, in extreme cases, the severity of the injury is such that vascular hemostasis and/or control of intestinal injury require a pancreatoduodenectomy (PD) (particularly for gunshot wounds). In this setting, gastrointestinal re-anastomoses should be performed at a second stage. There have been isolated reports of successful two-stage PD [54].

If hemodynamic instability can be controlled at open laparotomy

During a laparotomy for abdominal trauma, exploration of the pancreas must be systematic and comprehensive. This exploration should allow the surgeon to establish whether there is concurrent duodenal injury or injury to the pancreatic duct. Preoperative examinations such as cholangiography or ERCP should be prohibited; they are time-consuming and potentially dangerous, potentially risking the transformation of an isolated PT into a duodenopancreatic injury.

Lesions classified as AAST grades I or II (simple bruise or superficial glandular tear without ductal injury) represent 70% of reported cases [55]. Their treatment should be simple contact drainage after exploration and hemostasis. While dependent drainage may be adequate, closed suction drainage reduces the postoperative morbidity by nearly half [55,56].

If there is injury to the pancreatic duct (30% of PT), treatment depends on the location of the ductal rupture:

- if the ductal injury lies to the left of the superior mesenteric vessels (AAST grade III), distal pancreatectomy is associated with fewer postoperative complications than drainage alone [57,58], even in the child [59]. Splenopancreatectomy extended to the right as far as necessary is the most appropriate emergency gesture, and can be performed quickly and easily. Sacrifice of the spleen, as is commonly performed in some surgical services, may result in subsequent serious immunologic compromise with decreased resistance to infection (see chapter 2.1.3.2); therefore, distal pancreatectomy associated with splenic preservation is recommended whenever feasible, particularly in children [59]. Roux-en-Y pancreatico-jejunostomy to internally drain the pancreatic stump has been proposed but its efficacy has not been demonstrated [60,61]. Simple pancreatic drainage, which may sometimes be appealing in the urgent setting, is not recommended when ductal injury is present because it may result in severe complications that require further surgical intervention, sometimes including difficult delayed pancreatic resection (often for lesions that were underestimated at the initial intervention), with a 100% morbidity rate and a 50% mortality rate in Lin's series [58], and 100% morbidity and 20% mortality in our series;
- for lesions involving the pancreatic head (AAST grade IV or V), the surgical options may appear much more difficult, between cephalic PD and conservative methods. In such cases, simple wide contact drainage has the advantage of being fast, and leaves several options thereafter:
  - early secondary re-operation with resection by an experienced team,
  - endoscopic stenting,
  - an attempt to obtain directed pancreaticocutaneous drainage, knowing that most pure pancreatic fistulas heal spontaneously or progress to PPC;
- endoscopic retrograde cholangiopancreatography (ERCP) with stent placement is effective when the stent placement is successful [11] but carries the risk of late ductal stricture [62]. PD may seem a risky emergency gesture. Nevertheless, it may be the only possible solution in some cases of serious pancreatic injury: devitalization of the pancreatic head with damage of the choledochus and/or pancreatic duct, duodenal devitalization, or duodenal bulb perforation [63,64]. But PD can be implemented in several stages [54], allowing initial hemostasis and closure of intestinal injuries, with a several day delay re-establishing intestinal continuity. In 2003, Asensio published a series of PD for serious open wounds of the pancreatic head; the 33% global mortality rate after Whipple PD [64] must be compared with the known mortality for grade V PT that varies from 50% 100% depending on the series [58,65]. In our series, one patient with multiple trauma including a grade V duodenopancreatic lesion underwent PD in three stages according to the principles of damage-control laparotomy because of the severity of associated injuries.

For duodenal injury associated with PT, it is essential at exploration to locate the papilla and check the status of the pancreatic duct. Duodenal injury is considered serious if intestinal damage exceeds 75% of the circumference [66]. For simple duodenal lesions without pancreatic injury, the best option seems to be simple duodenal suture repair protected by a decompressive tube gastrostomy, a feeding jejunostomy, and possibly a cholecystectomy (which allows localization via a transcystic duct catheter, as well as performance of postoperative cholangiography); these procedures are complemented by wide peripancreatic drainage [67] (Fig. 5). For complex duodenal injury, a Roux-en-Y duodenojejunostomy will avoid stricture and is less risky than simple suture repair [66]. For combined pancreatic and duodenal injury involving the pancreatic duct, the therapeutic attitude must balance between PD (either immediate or in multiple stages) or, if PD seems too daunting, duodenal exclusion corresponding to the modified Berne technique [68] — wide drainage, a simple duodenal suture repair plus stapled pyloroduodenal occlusion and loop gastrojejunostomy.

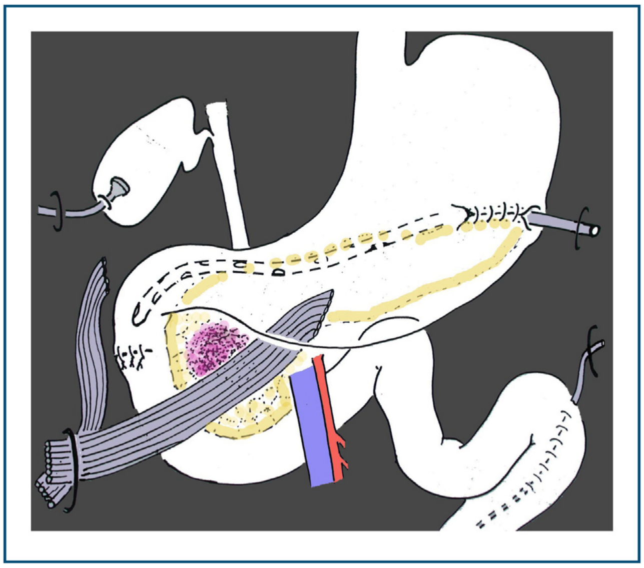

Figure 5. Duodenal suture repair protected by proximal tube gastrostomy with the tip of the tube advanced into the proximal duodenum. Drainage of the biliary tree by a cholecystostomy. Wide drainage of the region of the pancreatic head. A feeding jejunostomy completes the procedure.

Management of postoperative pancreatic fistula

The most common post-surgical complication is the formation of a pancreaticocutaneous fistula (8.1% for Vasquez et al. [65], 9.1% for Pata et al. [8], 12% in our series, 33% for Asencio et al. [64]). With octreotide and nutritional support, most fistulas heal within 1—4 weeks, provided they are well drained from the outset [8,69]. Fistulas that persist despite medical treatment are often fed by a pancreatic duct lesion; this can be treated by endoscopic placement of a ductal stent [70,71]. If the fistula persists after drain removal, it may evolve to the formation of a PPC, at best, or to an abdominal inflammatory tableau requiring distal pancreatectomy for lesions of the body or the tail or a Roux-en-Y pancreaticojejunostomy for cephalic injury [69]. Just as in elective pancreatic surgery, the presence of a fistula increases the risk of bleeding or septic complications.

Non-operative diagnosis and treatment

Non-operative diagnosis

In an era when the treatment of abdominal trauma has become increasingly non-operative, evaluation without the benefit of laparotomy must pay particular attention to the existence of signs suggestive of PT; studies should permit detection of PT and also allow evaluation of injury to the pancreatic duct and duodenum

Clinical presentation

Initial clinical signs are extremely variable, from almost asymptomatic patients (20% of cases in the series of Bradley et al. [72]) to patients with frank signs of peritonitis. Pain (inconstantly present and often delayed for several hours after the traumatic incident) is associated with nausea or vomiting, radiation of pain to the back or shoulders, and epigastric skin bruising. No symptoms can confirm a ductal injury. When associated duodenal injury is present, the initial picture may be muted but quickly evolves into one of peritonitis [69].

Laboratory testing may be suggestive but is not diagnostic

Serum levels of lipase and/or amylase have a low sensitivity, being elevated from the onset in only half the cases [72,73]. Evolution of lipase/amylase levels is more important than the initial values [74]. The specificity of enzyme levels is low since they are often elevated for any intestinal injury or cranial trauma.

Imaging is essential

Contrast-enhanced multi-detector CT is essential for prompt detection of PT, with a sensitivity of 80% [69,75]. A recent study by Lawson shows that in more than 26,000 abdominal trauma cases, only two cases of PT were missed on the first CT examination, i.e. a delayed diagnosis in only 0.34% of cases [76]. Nevertheless, out of all the abdominal organs, the diagnostic accuracy of CT is lowest for the detection of traumatic injury of the pancreas. When any clinical doubt persists after a negative initial CT, because of either clinical signs or laboratory findings, it is important to repeat the CT exam a few hours later. For detection of complications, CT is the gold standard. However, at the present time, CT cannot detect ductal injury with certainty except in the presence of complete pancreatic fracture [9] (Fig. 6). But great progress has been made in CT techniques in recent years allowing diagnosis of ductal injuries with ever increasing sensitivity [75].

Figure 6. Abdominal trauma from an all-terrain vehicle accident. Grade III pancreatic trauma with disruption of the pancreatic duct identified by CT scan. Non-specific signs include a heterogeneous appearance of the pancreatic head, free intraperitoneal fluid, and lesser sac fluid collection. Specific signs include the appearance of the pancreatic fracture as a hypodense line (arrow), enlargement of the pancreatic gland, and infiltration of the peripancreatic fat, with fluid present between the splenic vein and the posterior surface of the pancreas (asterisk).

MRCP is the gold standard for detecting ductal injury, allowing visualization of the entire pancreatic duct [58,77]. The presence of blood and fluid around the pancreas limits the effectiveness of MRCP in the immediate post-traumatic period, when the injured pancreatic duct is not dilated [58]. Sensitivity is optimal after a few days, which allows time, in practice, to arrange for this study, which is not always readily available in an emergency. Disruption of the pancreatic duct is typically visualized as a focal interruption with upstream dilatation. The fracture line, which still contains some static fluid, is hypo-intense in T1 and hyper-intense in T2 [78]. MRCP can also provide information that ERCP cannot provide: the presence of peripancreatic or peritoneal fluid collections, with or without communication with the pancreatic duct, as well as damage to other organs [78].

ERCP is the most sensitive test to detect ductal injury, however the upstream pancreatic duct cannot be visualized [70]. Ductal disruption is evidenced by contrast extravasation during ERCP: the contrast may remain within the gland or leak into the peritoneal cavity [79]. ERCP is also very useful in the subsequent search for late complications of trauma. The advantage of ERCP over MRCP is the addition of a therapeutic option, since it is possible to insert a multiperforated stent to bridge or drain the injured pancreatic duct.

Therapeutic decisions in non-operative management

If there is no ductal injury

Non-operative management (NOM) is best indicated for pancreatic contusion without pancreatic duct injury. The results of NOM under these conditions is excellent, with < 20% morbidity, and < 5% mortality [8]. This underscores the value of NOM and early ERCP [10]. The patient must be closely monitored in a surgical environment. Surveillance is based on the above-mentioned clinical, laboratory and radiological criteria: abdominal CT [80] remains the study of choice to detect possible complications. The medical management of grade I or II contusion is similar to that of acute pancreatitis. It includes dietary restriction and the insertion of a nasogastric tube in case of vomiting, adapted electrolyte intake, and analgesics; the use of prophylactic antibiotics is questionable. Intravenous octreotide®, which aims to reduce the fistula rate by inhibiting pancreatic exocrine secretion, has been the subject of several studies with small numbers; the results are conflicting [81]. One can nevertheless propose administration of octreotide to patients who are suspected of ductal injury and appear to be at high risk of pancreatic fistula.

Probable or definite pancreatic duct injury

In the stabilized non-operated patient with pancreatic ductal disruption identified by abdominal CT with IV contrast, MRCP and/or ERCP, and with no suspicion of duodenal injury, treatment depends on the location of the ductal injury, and clinical context.

If a disruption of the pancreatic duct is diagnosed early, whether involving the pancreatic head or the body or tail, endoscopic placement of an intraductal stent by a well-trained team yields excellent results in the child, and even in adults according to some authors [79]. The strongest predictor of success is the proper positioning of the stent, which must be placed on either side of the fracture, if possible, to bridge the defect [79]. If this proves too difficult, transpapillary positioning of the stent, combined with sphincterotomy, should reduce the volume of fistula drainage by decreasing the intraductal pressure [61]. When this is not possible (unavailability or failure of the procedure), the remaining two treatment options are surgery and medical treatment with close monitoring.

The surgical alternative, preferable whenever there is suspicion of an associated lesion, is by exploratory laparotomy treating the lesions by resection or drainage according to the patient's condition and findings at surgery. For pancreatic head injury, surgical intervention is often justified by associated duodenal or other intraperitoneal injury, since isolated grade IV-V pancreatic lesions are very rare. Treatment can consist of wide peripancreatic drainage, however in some cases, PD may be justified due to necrosis. If the pancreatic duct injury lies to the left of the mesenteric vessels and local conditions permit (early diagnosis), splenopancreatectomy or distal pancreatectomy is preferable to simple drainage since this entails a decreased hospital stay, and a lower rate of complications.

NOM for PT with ductal injury has been increasingly reported, always relying on certainty of perfect clinical stability monitored several times a day, and supported by transcutaneous drainage in case of PPC that may develop within 2—3 weeks, or by cyst-enterostomy if percutaneous drainage fails. However, for pancreatic duct disruption, NOM, despite the fact that it is ''fashionable'' at the moment, has been responsible for many irremediable situations. There is a risk of pancreatic necrosis, autodigestion of peripancreatic vascular or visceral structures, and infections; their consequences can be severe. The traumatized pancreas cannot be considered like other solid intraperitoneal organs for which NOM is feasible with the possibility of remedial surgery in case of deterioration [46]. A treatment delay of even 24 hours is associated with higher rates of morbidity and mortality: 80% morbidity and 11.1% mortality for Lin et al. [58]. In our experience, treatment delay is associated with 66% morbidity with all complications requiring surgical management. Delayed surgery is always more difficult because of tissue inflammation, oftentimes in a septic patient. Even in children, NOM is not less morbid. In Wood's study of 43 pediatric PT with ductal injury, the complication rate was significantly lower in the ''distal pancreatectomy'' group that in the ''non-operative treatment'' group — 21% and 73%, respectively [59]. Several other retrospective series of pediatric PT have confirmed this trend [9,82], with fewer complications and decreased length of hospital stay in the children treated surgically. When NOM is chosen, treatment is similar to that for acute biliary pancreatitis, as described in the preceding paragraph. Octreotide® administration is justified in this case by the high risk of pancreatic fistula. Spontaneous healing of the fistula is likely in less than 20% [9]. This treatment may require an intervention for drainage if PPC occurs, which will happen in 80% of cases [9,72].

Delayed diagnosis and treatment at the stage of complications

When the NOM option is not possible or a pancreatic injury is not initially recognized, pancreatic contusion can progress to infected post-traumatic acute pancreatitis [9,72] with a deep abscess or frank peritonitis. Disruption of the pancreatic duct promotes the development of PPC. Other rare but specific complications of PT include false aneurysm, portal vein thrombosis and duodenal stricture [69].

Acute post-traumatic pancreatitis

In patients with multiple trauma, the diagnosis of PT may be suggested by clinical deterioration after a delay of 4—5 days due to acute pancreatitis. CT will help to establish the diagnosis and assess its theoretical severity. This type of pancreatitis is very serious and potentially life-threatening with a mortality of up to 40% [83]. Laparotomy allows assessment of the injuries and performance of necrosectomy and drainage if necessary; but above all, it serves to eliminate doubt regarding other possible associated lesions of the duodenum or the rest of the digestive tract. In cases where there is no clear indication for laparotomy, endoscopic posterior retroperitoneal drainage of infected pancreatic necrosis may be feasible [84].

Secondary post-traumatic pseudocyst

PPC may be discovered very late, and may even pose the diagnostic possibility of a cystic tumor of the pancreas. But PPC usually becomes evident within weeks following the traumatic incident with classic signs (gastric emptying disorder, pain, elevated lipase). Treatment decisions depend on whether or not the PPC is symptomatic, its size, and its position relative to other organs. A small PPC (< 5 cm in diameter) without clinical signs of infection requires only simple monitoring especially when the patient is young [85]. Post-traumatic PPC tends to heal spontaneously in 25—50% of cases [77]. Otherwise, conventional methods of treatment should be considered: radiologically-guided external drainage (effective in half of the cases), and the pseudocyst-enterostomy with ductal drainage by ERCP.

Duodenal stricture

Duodenal stricture occurs more commonly in children, often due to duodenal intramural hematoma whose reabsorption may lead to stricture with an upper GI obstructive syndrome that may develop after a delay of more than 1 month [69]. Treatment is surgical by gastrojejunal or duodenojejunal bypass [66].

Conclusion

In patients presenting with hemodynamic instability, emergency laparotomy is required and, where the spleen is involved, splenectomy remains the most simple lifesaving gesture. If there is pancreatic injury, only the simplest and fastest hemostatic gestures should be performed. Patients who require splenectomy have an increased risk of postoperative venous thrombosis and thrombocytosis requiring close monitoring of platelet counts and evaluation for suspected thromboembolism. Due to the life-long risk of fulminant infections, post-splenectomy patients must undergo antibiotic prophylaxis and be vaccinated regularly; They and their primary care physicians must be educated to react quickly and correctly in case of febrile syndrome and to guard against certain infections, particularly malaria.

For the patient whose hemodynamic status can be stabilized at surgery, the surgical techniques that may permit splenic preservation, while rarely applicable, are useful to know in case of splenic damage in pediatric patients or iatrogenic splenic injury during another surgery. For pancreatic trauma, the treatment of superficial injury is simple drainage, while drainage with decompressive gastrostomy is indicated for deep pancreatic head injuries and distal pancreatectomy for deep injuries of the pancreatic body and tail.

NOM should be considered in a patient who is hemodynamically stable without peritoneal signs. Monitoring must be done in a surgical environment, with availability of good-quality imaging and quick access to the operating room for deferred surgery in good conditions if this becomes necessary. Patients with actively-bleeding splenic lesion evidenced by contrast extravasation on CT, a pseudoaneurysm, or arteriovenous fistula may benefit from splenic artery embolization. In non-operated patients at high risk for splenectomy (abundant hemoperitoneum associated with severe grade 3 splenic injury, multiple trauma), angiography with prophylactic splenic artery embolization appears to increase the rate of splenic salvage rate and should be further evaluated. For non-operated patients, the monitoring period in a surgical environment may be reduced to 7 days provided a CT scan with IV contrast is performed prior to discharge.

For patients with PT, while mortality is most often related to associated injuries, mortality directly due to PT is usually related to delay in diagnosis of severe injuries involving the pancreatic duct. The search for ductal injury is the key to decision-making: if ductal disruption is certain, interventional options should be discussed, aimed at either endoscopic stenting of the ductal injury, pancreatic resection, or the establishment of a broad local drainage with the possibility of subsequent operative intervention. Only in the calmest of clinical situations affirmed by close monitoring can NOM for pancreatic ductal disruption be proposed. This attitude relies on close surveillance, making the most of the resources of CT and/or MRCP, and employing interventional endoscopy when necessary.

KEYPOINTS

After total splenectomy:

- anticoagulation is indicated because of the risk of venous thrombosis in all at-risk patients because of lower-extremity trauma, using a standard dose of low-molecular-weight heparin (LMWH). Patients who undergo splenectomy should have bi-weekly platelet counts. If the platelet count exceeds 650,000/mm^3, it is appropriate to administer a LMWH such as 40 mg of Lovenox® and to additionally prescribe low-dose aspirin if the platelet count exceeds 1,000,000/mm^3;
- antibiotic therapy: Starting on POD-1, amoxicillin 500 mg BID for 2 days, then 1 million units of oral Penicillin-Vee-K twice a day; in case of allergy, erythromycin can be substituted;
- vaccination against pneumococcus; schedule 1: combined use Prevenar 13® on day 7 or at hospital discharge followed by Pneumovax 23® 2 months later; schedule 2: Pneumovax 23® at least 2 weeks after splenectomy);
- annual influenza vaccination;
- vaccination against meningococcus and hemophilus influenzae (for patients older than age 30, the utility of this is debatable);
- education regarding long-term treatment: vaccination reminders (antipneumococcal every 5 years), annual influenza vaccination, urgent consultation or prophylactic antibiotic therapy for any fever $\geq 38^\circ$, discussion of increased risks associated with malaria, domestic animal bites or scratches, and tick bites.

Wallet card for post-splenectomy patients:

- patient instruction sheet;
- date of splenectomy;
- dates of vaccinations;
- prescriptions for oral antibiotics at hospital discharge;
- patient education: vaccination reminders (antipneumococcal every 5 years), annual influenza vaccination, urgent consultation or prophylactic antibiotic therapy for any fever $\geq 38^\circ$, discussion of increased risks associated with malaria, domestic animal bites or scratches, and tick bites.

Conditions for non-operative management after splenic trauma:

- the patient's initial hemodynamic stability guides the choice between immediate surgery and CT scan;
- generally-agreed indications for splenic artery embolization are: (1) contrast extravasation during the arterial phase of a contrast-enhanced CT scan in a hemodynamically stable patient; (2) the development of a pseudo-aneurysm or arteriovenous fistula;
- authentic delayed splenic rupture (DSR) may occur between the first and 45th days following trauma and is due to either of two etiologies: subcapsular hematoma or pseudo-aneurysm. Given the prolonged time interval in which DSR may occur, the following principles of management should apply:
  - the imposition of strict bed rest has fallen into disuse since prolonged recumbency increases the risk of deep venous thrombosis (DVT) with no evidence that it decreases the risk of splenic rupture and it has no influence on the conditions that may lead to DSR,

- patients at high risk for DVT should undergo prophylactic anticoagulation with LMWH starting 48—72 hours after the trauma,
- the patient should be closely monitored in an intensive care unit (ICU) or surgical service for at least seven days (80% of DSR) and possibly for 10 days (95% of DSR),
- systematic performance of a contrast-enhanced CT scan prior to discharge is indicated, particularly since patient discharge occurs earlier than in the past; it should be the rule for any patient with a AAST splenic injury of grade III or higher,
- when a subcapsular hematoma has been detected, close observation in an ICU setting should continue until there is stabilization of the lesion by imaging studies,
- from the 10th to the 45th day following trauma, the risk of DSR is low but real: when discharged from the surgical service, the patient should be thoroughly informed of the risk of DSR and arrangements made to anticipate the need for rapid transport back to the hospital;

Diagnosis and treatment of pancreatic trauma:

- When any patient presents with abdominal trauma, hemodynamic stability is of primordial importance. If the patient is in shock, immediate laparotomy should be performed;
- When pancreatoduodenectomy is the only surgical solution for injury to the pancreatic head, the surgeon must consider the possibility of staging the resection with re-establishment of intestinal continuity 1—2 days after the initial damage-control surgery;
- The key to the treatment of pancreatic trauma is to accurately recognize the exact nature of the injury and, particularly, to recognize an injury to the pancreatic duct;
- The surgeon should systematically search for associated duodenal injury when pancreatic trauma is found at exploration;
- Contrast-enhanced CT scan is the best emergency study to detect pancreatic injury. In the non-urgent setting, MRCP, which can visualize the entire length of the pancreatic duct, is the best study to detect ductal injury;
- If PT with ductal disruption is discovered early, endoscopic placement of a ductal stent has yielded good results in both children and adults, when performed by a well-trained team;
- For pancreatic trauma with ductal disruption involving the body or tail of the pancreas, distal pancreatic resection with or without splenectomy has less morbidity and a shorter duration of hospitalization in both children and adults.

Disclosure of interest

The authors declare that they have no competing interest.

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