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Embolization of the proximal splenic artery for injuries of the spleen

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The spleen, the organ most commonly injured by blunt trauma, affects all ages and both sexes but is most common in young adult males and children. More than half of patients sustain isolated splenic injury but many patients have associated abdominl injuries to the liver, kidney, gallbladder, pancreas, and hollow viscera. Injuries to the brain, the lungs and thoracic cage, and the long bones and pelvis are also frequently seen. The mortality associated with splenic injury is about 8% ~ 10%; major morbidity is seen in more than 20%.

The clinical signs of splenic injury are very nonspecific and correlate poorly with injury. In the most severe cases diffuse abdominal pain with distension, and hemodynamic instability with shock are present making the diagnosis of abdominal catastrophe obvious. On the other hand, symptoms may be absent for quite some time, resulting in missed or delayed diagnosis.

The spleen has until recently been a organ in search of a purpose. Little was known about the functions for the spleen and this lack of knowledge was used to justify splenectomy for most injuries. The development over the past four centuries of routine splenectomy as the surgical treatment of choice for injury was encouraged by this lack of understanding of the role of the spleen. The fact that most patients appeared to tolerate its removal without significant sequellae advanced the concept that the spleen might be vestigial, an unnecessary organ without function in humans.

In 1952 King and Schumaker first reported the occurence of fatal sepsis in some children who underwent splenectomy for a variety of indications including trauma and began the questioning of the policy of mandatory splenectomy for injury. This sepsis was characterized by overwhelming bacterial pneumonia and meningitis leading to septicemia, disseminated intravascular coagulopathy, multiple organ failure and death. The infections were often caused by the fulminant growth of encapsulated organism, most commonly Haemophilus influenzae, Streptococcus pneumoniae and Nesseria meningitidis.

This association between splenectomy and fatal sepsis has been confirmed by numerous studies. The risk appears to be highest in children with thallasemia and other serious systemic illness but it is also a real risk in otherwise healthy children who have had splenectomy for trauma. Fatal sepsis has been estimated to be as much as 50 times greater in splenectomized children than in the normal control population. This added risk is not confined to children. Overwhelming post splenectomy infection was first reported in adults by Whitaker in 1969. It is often delayed and frequently occurs more than five years after the splenectomy has been performed. Green, Shackford, Sise and Fridlund reported that while late major septic complications occurred less commonly after trauma than other reasons for splenectomy, it was still common enough(6%) to recommend attempting splenic salvage before removing the injured organ.

The fact that splenectomized patients are immunocompromised and at risk to fatal infection clearly demonstrates that the spleen serves important immunological functions. The spleen is responsible for the clearance of particulate antigens and old and damaged blood cells. It has been shown that patients who have overwhelming sepsis after splenectomy often have considerably higher numbers of bacteria in their

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blood than patients with intact spleens who die of sepsis. (Shaw) This suggests an inability to clear opsonized bacteria. The spleen also plays a major role in early immunological defense in nonimmune patients and both cellular and humeral immune responses are affected by splenectomy. Splenic IgM production is especially important in the early response to bacterial challenge. Loss of splenic function also affects polymorphonuclear (PMN) granulocyte activity. The spleen is responsible for the synthesis and/or incorporation of the peptide Tuftsin onto its carrier molecule leukokinin which enables PMNs to maintain their phagocytic and pinocytic functions at high levels during infection. Cellular immunity is also compromised by the asplenic state as well. Fragmentation of the organ or deep fracture with free arterial hemorrhage and avulsion of the splenic hilum can result in exsanguination or hemorrhagic shock. However many patients with splenic injury will be hemodynamically stable on admission or easily resuscitated. Hemorrhage may be minimal or stop spontaneously.

If the capsule remains intact, the bleeding will be self-contained within the organ and result in intraparenchymal hematoma, subcapsular hematoma or both. If the capsule is torn, the intraparenchymal or subcapsular blood can extend into the peritoneal cavity. However, it may also be contained by the fascia and the surrounding extraperitoneal hematoma. The resistance resulting from the surrounding organ, capsule, fascia or hematoma will either exceed arterial or venous pressure and tamponade the bleeding and allow organization of the hematoma and secondary intention of the fractures or the insufficient to prevent expansion or rupture of the hematoma.

Arterial disruptions which do not heal may result in pulsating hematoma of the spleen. These may result in intermittent hemorrhage or expansion of the hematoma. Splenomegaly may result. If the capsule is stretched or the fragments separate, this high tension hematoma may rupture.

Over the past three decades several diagnostic modalities, including scintigraphy, arteriography, ultrasonography and computed tomography have been used as alternatives or adjuncts to DPL. While each has value, computed tomography has become accepted as a valid alternative to and, by many, a superior replacement of DPL in the management of hemodynamically stabilized patients with blunt abdominal trauma. CT is a highly accurate test for the detection and quantification of hemoperitoneum and retroperitoneal hematoma. In addition, CT has specificity and sensitivity greater than 90% in the detection and exclusion of splenic injuries. Furthermore, while there is overestimation and underestimation of some injuries, CT displays with fair accuracy the morphological character of the injured spleen and the extent of damage to the organ. Additionally it is a comprehensive study which allows the detection and exclusion of other associated intra abdominal injuries.

The direct CT signs of splenic injury are usually clearly visible on well performed scans. Intraparenchymal low attenuation and heterogeneous areas indicate splenic hematoma; areas of very high attenuration suggest active hemorrhage.

At Kings County Hospital Center abdominal angiography is usually indicated whenever a splenic injury has been identified on computed tomography in a hemodynamically stable patient. Unstable patients are treated by immediate laparotomy. The primary purpose of angiography is to further assess the splenic injury and to detect signs of active arterial hemorrhage. It is this author's assertion that arterial extravasation, either within or outside the spleen, indicates active bleeding or pulsating intrasplenic hematoma. These cases are the ones which are likely to fail a nonoperative course and, therefore require blood transfusion and some form of hemostasis.

Selective celiac arteriography is adequate as the initial study when CT prior to arteriography has shown that injury is confined to the spleen. The celiac artery is studied to assess the presence and adequacy of splenic collateral circulation, as well as to look at the liver and pancreas. There are both direct and indirect arteriographic signs of splenic injuries. Direct signs are manifested as injuries of the splenic vasculature. They include extravasation, occlusion, arteriovenous shunting and fistula, and focal vasospasm. Indirect signs of splenic injury include filling defects in the splenogram in the sinusoidal phase, stretching of the splenic vasculature, "according" of the splenic vessel and displacement of the spleen.

Filling defects in the sinusoidal phase or splenogram are the most common sign but most nonspecific sign of splenic injury. They may be isolated or associated with other signs of splenic injury. Avascular filling defects in the spleen may be caused by subcapsular or intraparenchymal hematoma, by splenic fracture, or by segmental vascular occlusion.

Parenchymal defects which are round and well-circumscribed represent intraparenchymal hematomas. Splenographic defects which are located in the periphery and are rounded or lenticular in shape are typical of subcapsular hematomas. Splenic fractures may also present as parenchymal defects; these are usually longitudinal and irregular and may branch. When filling defects are the predominant sign of splenic injury and are not accompanied by arterial contrast extravasation, a successful outcome to nonoperative therapy can be expected even when the filling defect is pronounced. Over the past fifteen years more than one hundred fifty patients have been observed without operation or embolization and none have failed conservative management. Transfusion requirements among these patients are negligible.

Extravasation of contrast medium is the most important angiographic observation. It is the most direct sign of intrasplenic arterial injury and signifies arterial bleeding or pulsating hematoma. It therefore identifies those patients at highest risk of failed nonoperative therapy. Extravasation may be unifocal but several sites of extravasation are often seen simultaneously. These may be adjacent to each other or may be diffusely distributed.

Significant contrast extravasations are large and coarse in nature. The are often irregular in size and shape and less evenly distributed. These result from shear injuries to intraparenchymal splenic branches. They are often seen to originate from a specific arterial branch. Such patients are managed by splenic artery coil occlusion with success.

Large rounded contrast accumulations which are well defined and dense represent false aneurysms resulting from transected splenic artery branches. They are most commonly seen in patients who present late after trauma or who have sustained clinical evidence of delayed rupture.

Most extravasations are confined within the spleen because there is often a hematoma containing it or because most of the capsule is intact to contain the hemorrhage. Furthermore active arterial extravasation which extends beyond the capsule is often associated with massive uncontained hemorrhage and such patients are often hemodynamically unstable from hemorrhagic shock. They often do not undergo CT or angiography because of their clinical stated and therefore the opportunity to see the findings are uncommon. When they are identified they are quite significant and the interventionalist must be extremely cautious and proceed with utmost haste. I have seen them most commonly in patients who were initially stable but began to deteriorate during the imaging studies. They should be embolized immediately. Visualization of the splenic vein before four seconds of the beginning of the arterial injection is a nonspecific finding and may be seen without evidence of arterial extravasation. Shunts or fistulas seen to originate from the hilum are considered evidence of hilar vascular injury. They are treated as if they represented arterial extravasation and all of these are managed by coil occlusion.

Currently nonoperative management of hemodynamically stabilized patients with splenic injury is considered the standard of care throughout the United States by general surgeons. Laparotomy is now limited to those who are hemodynamic unstable or who have associated intraabdominal injuries which require repair.

While it may diagnose splenic injury, CT does not accurately grade splenic injury and has not predicted with sufficient reliability which patients can be successfully managed nonoperatively. CT captures and isolated event at a moment's time. It does not describe the progression of the injury over time. It does not detail the degree of trauma to the splenic intraparenchymal vessels nor whether hemorrhage has ceased or persists.

Numerous authors have documented that CT both overgrades and undergrades splenic injury when compared to operative grading. Furthermore they have shown that there is a poor correlation between CT grades and the ability to predict the success of treatment.

We have found that arteriography is superior to CT for triaging splenic injuries to nonoperative therapy or surgery. It can reliably predict which patients can be treated by bedrest without complication. When splenic arteriography does not demonstrate extravasation of contrast medium either within or outside the spleen, bedrest without any form of hemostasis has been universally successful. Low pressure venous and parenchymal bleeding will be tamponaded by the surrounding hematoma.

Therefore celiac and splenic arteriography are performed as an emergency procedure immediately after CT demonstrates a splenic injury regardless of the grade of splenic injury. When arteriography shows no

arterial extravasation, patients are admitted to an ICU setting for 24 hours and, if stable, are then placed in a lower level of care. Gradual ambulation is allowed and patients are discharged in five to seven days depending upon the extent of the splenic fracrtures and the associated injuries. Patients are advised to restrict physical activity.

When splenic arteriography demonstrates that there is arterial contrast extravasation either within or external to the spleen, some form of hemostasis is warranted in the adult population. Transcatheter coil occlusion of the proximal splenic artery is a highly effective method of hemostasis and we do it in all adults who have angiographic extravasation.

Splenic artery embolization is an indirect method of hemostasis. It does not directly obliterate the source of arterial hemorrhage. Rather it acutely diminishes arterial inflow into the spleen and decreases temporarily splenic pulp pressure. This strategy allows hemostasis by facilitating spontaneous clotting of the pulsating hematoma without persistent bleeding.

The distal intraparenchymal arterial branches are end-vessels with minimal collaterals. Therefore, distal embolization with particulate matter such as gelfoam and ivalon results in infarction of splenic tissue and is undesirable since the one of the primary goals of the treatment of splenic injury is salvage of splenic function.

Proximal splenic artery coil occlusion avoids infarction because splenic perfusion is maintained, although at a lower pressure and at a lower flow rate by using the extensive collateral circulation. The short gastric branches connect with branches of the left gastric artery. The pancreatic magna branches connect to the dorsal pancreatic and the pancreaticoduodenal branches through the transverse pancreatic artery. The left gastroepiploic branch connects to the right gastroepiploic branch of the gastroduodenal artery. The omental branches of the superior mesenteric artery can provide splenic blood flow by connecting to the omental branches of the splenic artery.

Embolization is contraindicated in a variey of situations where other injuries take precedence over transport to the interventional radiology suite. These include hemorrhagic shock caused by intraperitoneal injury, associated abdominal injuries requiring laparotomy, intracranial mass lesions requiring urgent craniotomy, situations requiring urgent thoracotomy, coagulopathy and spleen with preexisting pathology. Laparotomy is clearly indicated in most of these situations because angiography is more time consuming and less definitive than splenectomy. Moreover, operative exploration can definitively control other simultaneous sites of hemorrhage, such as mesenteric vessels and hepatic veins which may not be amenable to transcatheter control. Associated injuries to the diaphragm, bowel and pancreas are often present in hemodynamically unstable patients. Subdural and epidural hematomas that require urgent craniotomy can be treated in the operating room at the same time as laparotomy.

Radiologic treatment of splenic injury requires an intact coagulation mechanism since splenic artery embolization does not directly control splenic bleeding but rather diminishes splenic inflow and pulp pressure thereby allowing spontaneous hemostasis. Spleens with preexisting disease such as lymphoma and leukemia, hemophilia, should not undergo splenic artery embolization unless the patient is considered inoperable or has high inherent risks of surgery.

This treatment of splenic injury is a generally safe and expeditious technique. The catheter should be seated securely within the splenic artery. Its tip should be placed distal to the dorsal pancreatic artery which is a major source of collateral flow. Coils are the device of choice for the embolization. Proper sizing of the coils critical. The coil must be larger than the diameter of the splenic artery so that it does not flow distal to the position of the catheter tip, lodge in the splenic hilum and block the major collateral routes. Neither must it be too large either since coils which are too large will not reform into a helical shape but rather will remain elongated as they are deployed and result in deployement of the coils within the celiac axis. Other complications beside errant emboli can occur. Many of these complications are unrelated to the procedure. These include dissection of the splenic artery (< 1%), infarction requiring splenectomy (1% ~ 2%), splenic or perisplenic abscess (1%), missed associated injury, atelectasis and pneumonia and deep vein thrombosis.

Patients who undergo splenic artery embolization are monitored observed in an intensive care environment for 24 hours after treatment. We consider it prudent to maintain a higher level of observation in the first day. If the patient maintains hemodynamic stability, patients are then placed in a standard hospital environment. Oral intake is begun when the pa-

tient has return of bowel activity. Activities are gradually increased over the next 4~ 6 days graduating from bed to chair to walking to discharge from the hospital.

Healing of splenic fractures should be substantial within three weeks. Limitation of physical activity after discharge from the hospital is suggested for a variable length of time. However, there are no good data regarding the optimal duration of restricition of physical activity. I suggest limitation of activity for six weeks for all patients. This should provide sufficient time for most minor and many major parenchymal injuries to heal and most hematomas to resorb and organize. A CT scan is repeated at that time to assess the degree of healing before making any further recommendations to the patient. If CT shows that the injury is healed, I permit the patient to resume full activity including contact sports. If, however, there is residual fracture or hematoma, I advise against full activity and continus a conservative approach. Outpatient CT scans are repeated at six to eight week intervals until complete healing has occurred at which time resumtion of complete activity allowed.

In conclusion, nonoperative treatment of splenic injury has been shown to be a safe, effective alternative to operative therapy in hemodynamically stabilized patients. CT improves the diagnosis of splenic injury while also proving the absence of other injuries. It is ideally suited as the first step in this conservative management. However, CT is not very good at predicting when nonoperative management will be successful. The author recommends splenic arteriography to make this determination. If there is no extravasation from the spleen, the nonoperative management will succeed almost always. If extravasation of arterial contrast is seen on angiography, then some treatment is necessary. Embolization of the splenic artery has been shown to be an effective method of controlling splenic bleeding.

·病例报告·

肺癌大咯血介入治疗一例

刘惕生

患者男性, 53 岁。经平片、CT、及支气管纤维镜检查确诊为右下肺中央型、低分化鳞癌。病灶与下后纵隔、横膈及右后下内胸壁粘连。病后共行三次经支气管动脉化疗灌注治疗, 间隔期为 4 周。经 2 次治疗后, 病灶影像表现无明显变化; 但第 3 次治疗后约 10 小时(夜间), 患者突发大咯血, 咯出鲜血约 1000ml。之后 3 小时内反复发作 6 次, 共计出血约 3000ml。间歇期平片示原右下肺大块密影已完全消失, 提示病灶液化排出。

患者行介入诊治: 首次介入诊疗采用选择性支气管动脉、肋间动脉造影, 寻找出血动脉, 发现右侧第九肋间动脉与肺动脉相通, 右支气管动脉已变细且未见出血迹象, 于是当时对右第九肋间动脉实施了明胶海绵栓塞。术后病情稳定了 20 小时。之后再次复发出血。第二次介入采用主动脉弓降部造影寻找靶血管, 此次发现右侧 7、8、9 肋间动脉及右膈下动脉相互间存在侧支交通, 并与右肺动脉交通, 形成多处体-肺动脉瘘。为避免造成严重栓塞并发症, 故对有关动脉进行了有限栓塞, 复查主动脉造影显示体-肺动脉交通征象已消失。基本达到目的。术后稳定了 4 天无咯血, 拔除了气管瘘管。但第 5 天又因一次咯血造成窒息, 抢救未能奏效。

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