GASTRIC NECROSIS AND PERFORATION AS A COMPLICATION OF SPLENECTOMY.
Case report and related references+

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ABSTRACT *- Necrosis of the stomach after isolated splenectomy with the formation of gastrocutaneous fistula is a rare event that occurs in less than 1% of splenectomies. It is more frequent when the removal of the spleen is done because of hematological diseases. Its mortality index can reach 60% and its pathogenesis is controversial, as it may be attributed both to direct trauma of the gastric wall and to ischemic phenomena. Although the stomach may exhibit exuberant arterial blood irrigation, anatomical variations can cause a predisposition towards the appearance of potentially ischemic areas, especially after ligation of the short gastric vessels around the major curvature of the stomach. Once this is diagnosed in the immediate postoperative period, it becomes imperative to reoperate. The surgical procedure will depend on the conditions of the peritoneal cavity and patient's clinic status. The objective of this study was to report on the case of a patient submitted to splenectomy because of closed abdominal traumatism, who then presented peritonitis and percutaneous gastric fistula in the post-operative period. During the second operation, perforations were identified in anterior gastric wall where there had been signs of vascular stress. The lesion was sutured after revival of its borders, and the patient had good evolution. Prompt diagnosis and immediate treatment of this unusual complication are needed to reduce its high mortality rate.*

HEADINGS *- Stomach rupture. Splenectomy. Necrosis. Abdominal injuries.*

INTRODUCTION

Gastric necrosis after isolated splenectomy, with the formation of cutaneous fistula, is a rare but serious event. It attacks less than 1% of patients submitted to splenectomy[5, 7, 9, 24] and its mortality index is high, possibly reaching 60%[9]. It is more frequently found in cases where spleen removal is indicated because of hematological diseases, and less commonly after splenectomy due to trauma[23]. It is generally attributed to traumatism of the gastric wall originating during sectioning and ligation of the short vessels around the major curvature of the stomach.

The objective of this study was to report on a case of gastric necrosis after splenectomy due to trauma that evolved with necrosis and gastric perforations, which we successfully dealt with.

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CASE REPORT

A.S.J., a 15-year-old white female, was admitted to the Emergency Unit of the "Hospital Brasil", in Santo André, SP, Brazil, 2 hours after a motorcycle accident, complaining of generalized abdominal pain. The physical examination revealed discolored mucosa, tachypnea, heart rate of 140 beats/minute and arterial pressure of 100 x 50 mm Hg. Inspection of the thorax showed multiple fractures of the right rib arches, with a reduction in the vesicular murmur in that half of the thorax. Dislocation of the right shoulder, extensive cuts and bruises, and lesion of the patellar tendon were also observed. The abdomen was diffusely painful when palpated or abruptly decompressed.

She was submitted to abdominal ultrasonography, which disclosed rupture of the spleen and the presence of free liquid in the abdominal cavity. Paracentesis and peritoneal irrigation confirmed the presence of blood, thereby indicating the need for surgical exploration.

Laparotomy demonstrated the presence of 800 mL of sero-hemorrhagic liquid free in the peritoneal cavity, a spleen lesion involving the hilum and several spleen segments with great devascularization (grade IV, in accordance with the organ lesion scale of the American Association of Trauma Surgery). There was a laceration of 6 cm on the diaphragm surface of the left hepatic lobe and another of small extent on the anterior face of the pancreas tail.

In the course of the splenectomy, the short vessels were carefully individualized and sectioned. The lesions of the liver and pancreas were sutured. The body and fundic regions of the stomach were inspected without any inadvertent ligatures of the gastric wall being detected. The left subphrenic space was evacuated using a tubular-laminar drain exteriorized through an opening on the left flank. At the end of the intra-cavity surgical procedures, the coloration of the stomach was normal.

The bowel's sounds returned on the second postoperative day, and the gastric catheter was then withdrawn, with oral feeding being started. The evolution was normal until the 5th postoperative day, when the clinical conditions suddenly deteriorated, with signs of peritonitis being observed. The secretion expelled by the drain, which had until then been serohematic, became dark and the volume visibly increased. On this occasion, tomography revealed the presence of collected liquid in the left subphrenic space and the suspicion of gastric fistula was confirmed by the oral administration of methylene blue.

The patient was submitted to a new surgical intervention, during which the presence of around 200 mL of secretion in the left subphrenic space was found. This was coming from perforations located in an area of the anterior wall of the stomach, between the body and the fundus of the stomach, whose overall dimensions were 6 x 4 cm. The stomach wall around the perforations presented a darkened color. After revival of the borders of the lesion, the orifice was sutured on two planes, with separate stitches.

In the postoperative period, the patient presented left subphrenic collection with the characteristics of an abscess, diagnosed by computerized abdominal tomography, which was successfully treated using antibiotic therapy. The patient was discharged on the 27th postoperative day in good general conditions.

The histopathological examination diagnosed an acute perforated ulcerated lesion of the stomach, with signs of edema, vascular congestion and hemorrhage, suggestive of ischemic necrosis of the gastric wall. At the present time, two years after the surgery, the patient continues to show no symptoms.

DISCUSSION

The stomach has exuberant arterial blood irrigation, which makes the organ resistant towards postoperative ischemic phenomena[2]. Several studies have demonstrated the rich intramural and extramural anastomotic network by experiments[2, 22]. Nevertheless, there are some surgical procedures that interfere to a greater or lesser extent with the blood supply, such that reports of gastric necrosis are becoming more frequent[13, 20].

Gastric necrosis after isolated splenectomy is a serious complication with a high mortality rate reaching between 52% and 73%[8, 17, 23]. It is generally attributed to traumatism originating during the sectioning and ligation of the short vessels around the major curvature of the stomach[5, 9, 24]. This traumatism frequently occurs in the upper region of the stomach, where the splenic gastric ligament is found in close contact with the gastric wall. Thus, during ligation of the short vessels in this area, there may be involvement of segments of the organ wall[9, 11, 12]. Pertinent to our case, the possibility of gastric necrosis occurring in this situation has been experimentally demonstrated[11, 12].

After splenectomy, the insertion of invaginating stitches at the location of ligation of the short vessels around the upper part of the major curvature has been proposed, with the aim of protecting it from possible perforation[9]. The inadvertent involvement of the gastric wall during ligation of the short vessels is more frequent among obese individuals because of the greater technical difficulty, increasing the possibility of complications occurring in this group of patients[17, 24].

Nevertheless, accidental ligation of the gastric wall does not explain all of the cases described, as gastric necrosis may extend to areas distant from the locations where ligatures of the short vessels were performed[4, 6,10,19,20,25]. One possible explanation for such an occurrence is the existence of anatomical variations in the distribution of arterial supply to the stomach, creating a predisposition towards the appearance of potentially ischemic areas[1, 14]. Other factors contributing towards the onset of gastric necrosis after splenectomy include arteriosclerosis, diabetes, renal insufficiency, uremia, cardiac insufficiency and the use of corticosteroids and other immunosuppressors[7, 18].

Surgical procedures on the stomach itself or lower esophagus may also provoke gastric necrosis. The pathogenesis of ischemic necrosis of the small curvature after proximal gastric vagotomy is explained by the relative blood deficiency characteristic of this region, associated with the reduction in mucous blood flow consequent to the vagotomy[15, 16, 21]. Concomitant splenectomy may further aggravate this risk due to the additional blood deprivation it causes[1, 10, 20] due to ligation of the short vessels and occasionally the left gastric omental artery or the posterior gastric artery when present.

The performance of fundoplication, whether associated with other surgical procedures or not (gastrectomy or proximal gastric vagotomy) may also provoke gastric necrosis[1, 10]. The release of the fundus of the stomach by the ligation of its short vessels, often necessary for non-tensional anti-reflux valves to be made, may promote or aggravate the local circulation deficiency. When splenectomy is associated with these procedures, the risk is increased even more[10].

Therapeutic embolization of the left gastric artery in patients previously submitted to splenectomy may also provoke gastric necrosis by taking away an even greater fraction of the blood irrigation[3]. In this situation, gastric necrosis will occur without there being surgical traumatism of the stomach wall.

It is possible to speculate that the necrosis that occurred in the patient we treated may have been due to the ligation of the short vessels associated with deficient anastomoses between the various arterial territories of the stomach, as the ulceration occurred in the anterior gastric wall, albeit far from the ligatures of the short vessels, in a location where signs of deficient irrigation were found to an appreciable extent. This hypothesis is reinforced by the fact that during the performance of the splenectomy we carefully inspected the major curvature without observing local traumatism or alteration in stomach coloration at the end of the operation. In this way, an invaginating suture around the major curvature done after ligation of the short vessels would not have prevented the perforation in free peritoneum in our case.

Normally, the symptoms of gastric necrosis after splenectomy appear between the 2nd and the 10th postoperative days in the form of upper digestive hemorrhages, subphrenic abscess or peritonitis with sudden onset, as in the case of our patient. The presence of an effective abdominal drain facilitates the diagnosis of the complication by revealing changes not only in the volume but also in the characteristics of the drained effluent, which become suggestive of gastric secretion[9, 17]. The confirmation of the presence of fistula is performed using abdominal radiography after the ingestion of water-soluble contrast medium[24] or the oral intake of methylene blue as used in our case.

The treatment of this complication is preferably surgical, although occasionally there is success using a conservative treatment[9]. Once it has been diagnosed in the immediate postoperative period, it becomes imperative to operate again. The type of surgical procedure to be used is dependent on the conditions of the peritoneal cavity and of the patient. Suture of the gastric wall may be done on smaller lesions where local debridement is possible. In cases of larger lesions affecting significant areas of the stomach, there is a preference for gastric resection.

Anatomical knowledge of the arterial irrigation of the stomach with its variations, and careful execution of the surgical technique avoiding ligation of segments of the gastric wall, are fundamental in reducing the occurrence of gastric necrosis after splenectomy. Gastric necrosis should be suspected when a patient who has had splenectomy presents unfavorable postoperative evolution. The high rate of mortality of this complication can only be reduced by prompt diagnosis and immediate treatment.

Martinez CAR, Waisberg J, Palma RT, Bromberg SH, Castro MAP, Santos PA dos. Necrose e perfuração gástrica como complicação de esplenectomia. Relato de caso e referências relacionadas. Arq Gastroenterol 2000;37(4):227-230.

***RESUMO** - A necrose do estômago após a esplenectomia isolada com formação de fístula gastrocutânea é evento raro que ocorre em menos de 1% das esplenectomias, sendo mais comum quando a retirada do baço é feita por doenças hematológicas. Seus índices de mortalidade atingem 60% e sua patogênese gera controvérsias, podendo ser atribuída tanto ao trauma direto sobre a parede gástrica, quanto a fenômenos isquêmicos. Apesar do estômago exibir exuberante irrigação sangüínea arterial, variações anatômicas podem predispor ao surgimento de áreas potencialmente isquêmicas, especialmente após a ligadura dos vasos curtos ao longo da grande curvatura gástrica. Uma vez diagnosticada no pós-operatório imediato, a reoperação se impõe, estando o procedimento cirúrgico na dependência das condições da cavidade peritonial e do enfermo. O objetivo deste estudo é relatar o caso de uma doente que, após ter sido submetida a esplenectomia por traumatismo abdominal fechado, apresentou no pós-operatório, peritonite e fístula gástrica percutânea. Durante a reoperação, identificou-se perfuração em peritônio livre da parede gástrica anterior onde havia sinais de sofrimento vascular. A lesão foi suturada após reavivamento de suas bordas e a enferma teve boa evolução. Somente o diagnóstico precoce e o tratamento imediato desta inusitada complicação poderão diminuir sua alta taxa de mortalidade.*

***DESCRITORES** - Ruptura gástrica. Esplenectomia. Necrose. Traumatismos abdominais.*

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