CAUSES OF FAILURE IN ABDOMINAL WALL DEFECTS SURGERY

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Abstract: Abdominal wall defects occupy one of the leading places in the general surgery wards and tend to grow in recent years. Parietal defects are not always a pathology easy to approach because the abdominal wall reconstruction may result in total or partial failure intraoperatively or postoperatively. Parietal defects reconstruction failure may be influenced by factors related to the patient’s biological and general status (congenital anomalies, ascitic decompensation, gender etc.), factors related to the surgical act itself (septic contamination, tissue changes, failure to follow the correct sequence of the surgical steps, alloplastic material) or a combination of the two categories which we called “borderline” failure factors (parietal hematoma, parietal suppuration, alloplastic material rejection, etc.). The probability of surgical failure in the abdominal wall defects pathology increases with the parietal defect size, the urgency degree and the more unprepared patient, therefore the surgical indication is so prevalent in the chronic regimen.

Starting from Dr. M. Rosen statement – “If 50 years ago, Prof. Rives called hernia a breach in the abdominal wall, today we consider hernia as the start of an abdominal drama”, abdominal wall defects occupy one of the leading places in frequency in the general surgery departments, which demonstrates the importance of this pathology for the surgeon and especially its socio-economic implications.(1)

The failure of the surgical treatment followed by the possible postoperative complications and/or surgical reinterventions leads to increased hospitalization, late social reintegration, prolonged postoperative recovery and, subsequent, higher costs.

Abdominal wall defects are usually associated with other comorbidities and unfavourable local factors may subsequently lead to a failure of the parietal surgery.(2)

An important aspect of this pathology is the increased risk of postoperative complications, initially localiz – from seromas in the postoperative wound, thread granulomas, wound dehiscence, etc. up to incarceration, strangulation, enteral fistulas, adhesion syndromes and later, bowel obstructions. Although the pathology is initially located at the abdominal wall, it may finally lead to systemic complications, sometimes irreversible.(3)

The probability of surgical failure in the abdominal wall defects pathology increases with the size of parietal defect, the urgency degree and the more unprepared patient, both in general terms (decompensated cardiac pathology, severe metabolic disorders etc.) and also, in a digestive point of view (filled bowel loops, in bowel obstruction). These are some of the reasons why surgical indication in chronic regimen is wide, with appropriate preoperative preparation of the patient. According to Prof. Dr. Rădulescu, “no parietal defect is too small to be operated”.(4) We classified the failure factors of parietal defects surgery in three main categories: patient’s biological and general status related failure factors, surgical act related failure factors and “borderline” failure factors.

1. Patient’s biological and general status related failure factors

With an increased incidence, congenital anomalies (omphalocele, gastroschisis, congenital umbilical and inguinal hernia etc.) are primarily parietal defects or may be subsequently determining factors that may favour an abdominal wall defect due to an insufficient resistance of the abdominal wall at the exercised intraabdominal pressure.

Prenatal ultrasound has a high sensitivity in the diagnosis of these anomalies from the first trimester of pregnancy.(5)

Collagen diseases and changing the type I collagen/type III (immature) collagen ratio causes the formation of lower quality connective tissue. This favours the appearance of hernia associated with another condition where the collagen pathology is suspected (osteogenesis imperfecta, joint hyperlaxity, etc.).(4)

Consumptive diseases are also included in the risk factors category, being able to determine the apparition of parietal defects. For example, we encounter acute or chronic inflammatory disorders, neoplastic diseases, cardiovascular disorders, all of them affecting the nutrition of the abdominal wall.(6,7,8)

Ascitic decompensation of cardiac or hepatic origin causes major electrolyte imbalances, which affects the quality of the entire abdominal wall from the peritoneum to the skin. This thickened, edematous abdominal wall has a poor quality (figure no. 1).

Increased duration of the surgical interventions in patients with multiple comorbidities seems to play an important negative role over the reconstruction of the abdominal wall.(9)

Gender, by specific anatomical particularities in

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women (larger pelvic transverse diameter), explains the frequency of the femoral hernias in this genre.

Figure no. 1. CT examination of a cirrhotic patient with an abdominal wall defect

2. Surgical act related failure factors
Septic contamination is a major failure factor in the abdominal wall surgery. The rehabilitation measures of the abdominal wall septic foci represent a priority. However, there is a possibility of accidental intraoperative discovery of microabscesses, thread granulomas, especially in case of reinterventions.

The operative wound becomes contaminated, scarring will be difficult and the use of alloplastic material is not recommended in the reconstruction of the abdominal wall. Local infections of the abdominal wall after laparotomy have a percentage of 5-10% and if not evacuated in short time and are not responding to appropriate antibiotic treatment, they can create real problems in the integrity of the abdominal wall.(10)

Tissue changes occurred after repeated surgical interventions in the abdominal area, especially in case of large eventrations which require alloplastic material (11), give rise to a modified, fibrous, retractable tissue, with multiple adhesions. (figure no. 2). Extending the resection of these tissues is difficult to assess and the scarring process is sometimes questionable. Failure to follow the correct sequence of the surgical steps can cause visceral lesions, especially in case of eventrations and eviscerations, where the adherence process is more intense and the possibility of iatrogenic damage is higher. The surgical technique itself may be a cause of failure, sometimes too large anatomical incisions can cause an iatrogenic parietal defect. Median and transverse incisions lead to similar occurrence rates of parietal defects.(12)

Figure no. 2. Fibrosis after insertion of alloplastic material

Inadequately used alloplastic material can be a major cause of surgical failure. Parietal alloplasty as a principle of surgical treatment of the inguinal hernia was first introduced by Prof. Dr. Rives in 1966 and represents the “gold standard” procedure.(13)

A microporous prosthesis (shrinkage) of small dimensions can suffer a disinsertion from the muscle-aponevrotic layer due to increased parietal pressure and can migrate from the original site, causing a decreased abdominal wall resistance and a place for possible recurrence of the parietal defect.

Closing the abdominal wall in multiple layers favours the occurrence of eventrations more than the closure in one single layer, especially if inadequate suture materials in structure and size are used. Sutures are responsible for the first months of postoperative wound integrity. Using a continuous thread is useful for uniform distribution of tension in the wound, but its failure can lead to the whole wound dehiscence. Successively failed surgical techniques require finding new surgical solutions tailored to the patient. The abdominal wall should not be closed in excessive tension (figure no. 3).(14)

Figure no. 3. Tension in the graft material

3. “Borderline” failure factors
We take into consideration such factors when the etiology of the surgical failure in case of a parietal defect is a bad combination between the surgical act and the diseases of the patient. One such example is the parietal hematoma occurred due to inefficient intraoperative hemostasis and/or preexisting vascular pathology – antiplatelet or anticoagulant treatment. Postoperative hematomas can be treated conservatively in small dimensions but with prolonged hospitalization days (15), or may require surgical reintervention for the completion of hemostasis.

Superficial or deep parietal suppuration is a feared complication and can completely compromise the outcome of the surgical intervention, especially in immunocompromised patients. The suppuration is more serious when it appears in a presence of a wall prosthesis and can ultimately determine the surgical removal of the alloplastic material. One advantageous solution could be the component separation technique, with the advantage of the recovering of the white line for superior functional outcome (figure no. 4).(16)

Figure no. 4. Abdominal wall abscess after rejection of the alloplastic material
Rejection of the alloplastic material is a rare cause of failure, but it can occur after an improper insertion of the prosthesis or because of an allergy to one of the components of the prosthetic material. Graft rejection requires removal of the graft, remediation of the septic local focus and rethinking the parietal reconstruction.\(^{(17)}\)

Postoperative intestinal fistula occurred, either due to improper placement of the alloplastic material directly on the visceral mass, or due to neoplastic background of the patient with severe electrolyte imbalances, who underwent radiotherapy, is an extremely serious complication which predisposes to the destruction of the abdominal wall. Surgical intervention focuses primarily on the digestive fistula, only afterwards we consider the reconstruction of the abdominal wall.

**Conclusions:**

1. Parietal defects surgery tends to become an important branch of general surgery, due to this increasingly complex pathology, often associated with intraoperative or postoperative complications.

2. Failure of the parietal defects reconstruction surgery depends on the patient’s associated pathology and also on the surgical act itself. One cannot always determine a limit between these two causes of failure, sometimes there is a combination of risk factors that subsequently leads to an imperfect reconstruction of the abdominal wall parietal defect.

3. The development of alloplastic materials with high biocompatibility created the notion of “gold standard” and reduced the risk of parietal defects recurrence.

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