Progressive Preoperative Pneumoperitoneum (PPP)

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45.1 Introduction

The functions of the abdominal wall are: visceral retention and protection, active participation in performing core movements, aids in defecation and urination, and regulation of the diaphragmatic movements for adequate pulmonary function.

During embryological development the abdominal viscera enter and expand the cavity, such that it adjusts to its newly acquired visceral content; because of its dynamic nature and constant response to change it exerts low pressure on the intra-abdominal viscera.

The capacity of the abdominal cavity varies according to the volume and content. In pregnancy or ascites, the abdominal wall gradually distends increasing the ability to contain the new content.

In giant abdominal hernias this process is reversed, as the viscera move into the peritoneal sac, the abdominal cavity shrinks, the visceral content protrudes into a "container", the peritoneal sac. In these hernias the volume of intra-abdominal viscera is reduced, and the intra-abdominal pressure adapts consequently, gradually reducing the contractility of the musculo-fascial structures, with a pronounced myofascial retraction that worsens with time [1–4].

Hernias are not only defects in the abdominal wall but are part of a whole pathological process which includes respiratory, vascular and visceral dysfunction. Moreover, they are frequently associated with obesity, chronic obstructive pulmonary disease, malnutrition, infection kidney and heart disease, which are predisposing factors for their development.

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When a patient has a giant hernia, changes in the mesentery, bowel, skin and subcutaneous tissue occur. Venous and lymphatic flow is reduced by compression from the annulus. This causes an edematous, thickened and difficult to reduce mesentery.

The loss of domain caused by the lateral fascial muscle retraction, the diaphragmatic relaxation and the frequent association between hernias, obesity and cardiorespiratory disease turns these patients into biologically and socially handicapped individuals [5–8].

45.2 Loss of Domain, Definition

Loss of Domain is defined as a large hernia, with a diameter of >10 cm or those whose contents of the hernia sack exceed the capacity of the abdominal cavity; technically it is one in which more than 50 % of the abdominal contents are located outside of the abdominal cavity. Generally they take years to form, the "giant" hernia sacs contain the viscera that can't be reduced because the abdominal cavity is no longer able to accommodate them.

Mason defined them as those in which it was not possible to reintroduce the contents of the sac into the abdomen. He estimated a volume contained in the hernia sac of over a litre or a diameter of the hernia ring exceeding 12 cm [9, 10].

Kingsnorth considers these hernias as those in which the peritoneal sac has a volume of more than 15–20 % of the natural volume of the abdominal cavity. He believes that if the ratio of the volume of the hernia sac over the volume of the abdominal cavity is less than 20 %, it is possible to perform a tension-free fascial closure.

According to Tanaka et al., the volume of the abdominal cavity is the main indicator of the loss of domain; it is easy to measure the volume of the abdominal cavity as is the volume of the herniated viscera or hernia sac. If the ratio of the volume of the sac over the volume of the abdominal cavity is greater than 25 %, it is considered a predictor for loss of domain [11–15].

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45.3 Loss of Domain, Pathophysiology

Giant hernias occur through fascial defects that gradually lose their domain in the abdominal cavity, with changes that are "tolerated" because they develop gradually but will ultimately reduce the intra-abdominal pressure and the capacity of the abdominal cavity.

The complexity of these patients lies in the loss of a functional abdominal cavity. There are pathophysiological changes caused by maladjustment of multiple organ systems: increased pressure causes decreased lymphatic and venous portocaval return to the chest, there is vasodilation and venous stasis in the abdomen, pelvis and lower limbs. Because of the decreased venous and lymphatic return chronic edema occurs in the omentum, mesentery and bowel. Friction exerted by the ring on the bowel conditions inflammation that causes adhesions between loops of bowel, the sac and the hernia defect. Intra-abdominal pressure decreases as more and more bowel protrudes into the hernia sac; this causes decreased diaphragmatic excursion which lowers the strength of the diaphragm and alters ventilatory physiology generating both an inspiratory and expiratory restriction [1–4, 11, 12].

45.4 Management of the Hernia with Loss of Domain

Loss of Domain implies that the abdominal contents are permanently found in the hernia sack (a second abdominal cavity).

These hernias are a challenge for the surgeon because of the difficulty to replace the contents of the visceral sac into the abdominal cavity. As the cavity, once emptied of its contents contracts, decreases in size, and is unable to accommodate the herniated viscera.

The forced reduction with primary closure can cause a devastating increase in intra-abdominal pressure which in turn leads to a reduction in cardiac output because of a decrease in venous return (preload) and an increase in peripheral vascular resistance (afterload). There is an indirect reduction in myocardial contractility caused by a decrease in left ventricular adaptability. There is also a decrease in mesenteric and splanchnic vascular flow; kidney function deteriorates as there is decreased perfusion which leads to oliguria and azotemia; hormones such as renin, which affects the systemic blood flow, are also released which further worsen vascular dynamics [6, 9, 11, 16, 17].

The reduced thoracic volume and pressure exerted on the diaphragm reduce the vital capacity that can lead to severe respiratory failure with hypoxemia and hypercapnia which further worsens diaphragmatic excursion leading to a reduction in venous return and hypertension.

An abdominal compartment syndrome ensues, causing intestinal ischemia, respiratory distress, renal failure, skin ischemia and/or necrosis. The surgeon might face hernia repair dehiscence or altogether find himself unable to complete the repair. We must bear in mind that these patients frequently have concomitant diseases such as obesity, heart, or respiratory diseases which aggravate this situation [4, 12, 13, 18, 19].

To avoid this, it is imperative that adequate preparation be performed, favouring the gradual rehabilitation of all systems, the reintroduction of visceral content into the abdominal cavity and the reconstruction of the abdominal wall.

45.5 Hernia Surgery with Loss of Domain

The use of prosthetic material in the repair of giant hernias is associated with complications in 32 % of patients: infection, enterocutaneous fistula, ileus, intestinal perforation, chronic pain, abdominal rigidity, intestinal obstruction, foreign body sensation and seroma. The quality of life of these patients is inversely proportional to the size of the implanted mesh.

The effects of the myofascial retraction in these cases influence the complexity of the wall repair. Current options for hernia repair with closure under these conditions are: a viable tissue bridge with permanent or biological prostheses, tissue flaps with autologous fascia lata, rectus femoris or latissimus dorsi and/or the use of tissue expanders and preoperative progressive pneumoperitoneum (PPP) [4, 20].

45.6 Preoperative Progressive Pneumoperitoneum

Before the advent of anti-TB drugs, pneumoperitoneum was used as a treatment for peritoneal tuberculosis.

In 1940, Goñi Moreno in Buenos Aires, Argentina, was the first to report the use of preoperative pneumoperitoneum in giant hernia repair. Goñi Moreno's work was presented at the American College of Surgeons in 1947. The reasoning behind his idea was to allow the reintroduction of the abdominal viscera into the cavity, and their readaptation to the abdominal cavity in a progressive fashion, reducing cardiovascular and respiratory complications immediately after surgery.

The technique of preoperative progressive pneumoperitoneum described by Goñi Moreno allows a more physiological adaptation of the patient and the abdominal cavity to the reintegration of the viscera into the abdomen, which favours adequate surgical repair [1, 2, 8, 9, 12, 16].

45.7 Objectives of the PPP

It takes time to restore the abdominal capacity during the PPP. One should perform abdominal CT scan to assess the volume of the hernia. Measurements proposed by Tanaka et al. confirm the loss of domain if the volume of the sac is equal to the volume of the abdominal cavity. The tomo-

graphic measurements should be done at the level of the third lumbar vertebra, corresponding to the midpoint of the abdominal cavity [11, 21, 22].

Transoperative pneumoperitoneum has been proposed to have the same benefits of the PPP, it can reduce the degree of visceral and mesenteric edema, it promotes lysis of adhesions between the hernia ring and sac and allows easier identification of hidden defects. These benefits without a prolonged hospital stay [10], however, reduction of the herniated viscera is only "temporarily" made possible by the muscle relaxing effects of the general anaesthesia, which will afterwards return to baseline with subsequent respiratory distress. These acute changes can lead to atelectasia formation, hypovolemia, shock, thrombophlebitis and thrombo-embólicos complications [3].

The objective of the PPP is to "gradually" stretch the abdominal cavity with the concomitant increase in the length of the abdominal wall muscles. It increases intra-abdominal pressure gradually and improves diaphragmatic function, which in turn improves ventilatory dynamics.

As the intra-abdominal pressure gradually increases, there is a decrease in the thoracic compliance. The abdominal cavity progressively enlarges, and changes in the viscera allow for the uneventful reintroduction of the herniated contents during the procedure.

45.8 PPP Physiology

Patients with hernias with loss of domain have low intra-abdominal pressure. There is an imbalance between intra-abdominal and thoracic pressure as a result with a resultant weakened diaphragm, which leads to a lessened participation of it in respiratory mechanics.

PPP acts in a way similar to pregnancy or accumulation of ascitic fluid way: it expands the soft tissues of the abdominal wall without causing sudden increase in intra-abdominal pressure.

PPP causes distension of the musculo-fascial structures and increases the volume of the once retracted abdominal cavity. This happens with a subsequent elevation of the diaphragm which will resume its normal position once the pneumoperitoneum is released. Although it has been documented that the vital capacity decreases in approximately 25 % (maximum reduction) during PPP, stretching the diaphragm improves subsequent post-operative respiratory function. Pulmonary function tests performed immediately after surgery show a vital capacity of 60–75 % of pre-PPP values. This compares favourably with the 60 % reduction in vital capacity observed during a routine cholecystectomy during the first post-operative day [1, 7, 8, 19, 22].

With the elevation of the diaphragm and the lowering of the pelvic floor during the PPP there is an increase in the abdominal cavity volume. The turgidity of the herniated organs is restored reducing their volume. This relaxation of the abdominal wall promotes healing of any decubitus injury caused by the herniated viscus [5].

The gradual increase in the capacity of the abdominal cavity will allow for the intra-abdominal pressure to remain low despite the contents being reintroduced into the cavity. This results in improved diaphragmatic function and venous return, especially relevant for patients with cardiopulmonary co-morbidities who would otherwise have high risk of hemodynamic and respiratory complications.

Preparation of a patient with a giant hernia with PPP facilitates intraoperative dissection of the hernia sac and its contents due to the preoperative lysis of adhesions by the air.

The PPP acts as the conventional laparoscopic pneumoperitoneum, facilitating dissection of adhesions in an atraumatic way. Adhesions are stretched and enterolysis facilitated unless these adhesions are firm and therefore do not allow for the visceral reduction. This gradual pneumatic lysis of adhesions improves portal and mesenteric circulation and during the procedure itself will facilitate dissection and reduction of the herniated content [2, 3, 16].

It has been reported that the insufflation of air into the abdomen fills not only the cavity, but also the hernia sac. This prevents the sac from literally hanging and thereby decreases chronic edema of the mesentery and other intra-abdominal organs.

The effect of adherenciolisis explains the homogeneous distribution of air through the abdominal cavity; interestingly, air distends the abdominal cavity more than it does the hernia sac [1, 6, 9, 19].

The immediate result when performing the PPP is the distension of the hernia sac; however, over time the gradual increase in the size of the abdominal cavity will be apparent. As these changes transpire, the viscera return to the abdominal cavity, leaving the air filled sac over them and aided by gravity. This is possible to see with a plain lateral decubitus X-ray of the abdomen.

Another effect of pneumoperitoneum is increasing the length of the abdominal wall muscles. Studies have been performed utilising CT scans of the abdomen which document the effects of PPP in the size of the hernia and abdominal musculature. They confirm that the PPP causes passive stretching of the rectus abdominis muscles. Despite the longitudinal orientation of the rectus muscle the PPP increases the amplitude and length of the musculature, exerting a similar effect on the hernia ring [12, 22].

Intermittent insufflation causes stretch of the muscle fibres. Microscopic studies of muscle sections from experimental studies show muscle dilation of all layers without hypertrophy or hyperplasia. The effect is that of expansion and a reflex adaptation towards relaxation of the abdominal muscles. This expansion also causes areas of necrosis and lymphoid cell aggregates along with a reactive inflammation of the peritoneum [14].

After the second week of PPP, peritoneal irritation caused by the air insufflated into the cavity stimulates the immune system and improves leukocyte macrophage response, promoting the subsequent healing of the wounds [8, 18].

45.9 Progressive Pneumoperitoneum Preoperative Technique

PPP requires frequent insufflation of air into the abdominal cavity.

In his first case Goñi Moreno used oxygen for insufflation; he later changed to ambient air. You can use oxygen, CO_2, nitrous oxide and ambient air. However, one must remember that oxygen and CO_2 have faster absorption (four times faster) into the intraperitoneal space than ambient air [12, 17, 21].

Some authors prefer nitrous oxide because it is more readily absorbed than air; in these cases its use should be avoided by the anaesthesiologist. This is because the gas used in the respiratory cycle dissolves into the blood and quickly diffuses into the peritoneum. This can lead to a sudden increase in intra-abdominal pressure which can lead to hypovolemia, respiratory failure and death; this complication is quickly dealt with by opening the abdominal cavity [1, 3, 5, 8].

Since its original publication, multiple modifications to the technique have made it easy to perform and safe. In 1990, Caldironi proposed daily abdominal punctures with a Veress needle and use of CO_2. Initially the technique required for repeat abdominal punctures with a lumbar puncture needle (blunt tipped) gauge 16–18 performed every 24–48 h. Vascular access catheter such as the dual lumen 16 g angiocath placed percutaneously have also been used. Double lumen catheters inserted through a Veress needle has also been used, pigtail 5Fr catheters inserted under ultrasonographic or CT guided control have been used. Other techniques involve a modified Seldinger technique with the insertion at different points such as the left midclavicular line in the subcostal space, in the semilunar line, at Palmers point (intersection of the linea alba and the semilunar line), in the left iliac fossa or on a remote site from the hernia or previous scar.

The procedure is generally performed in the operating room under local anaesthesia and sedation, but can also be performed on the patient's bed under strict aseptic conditions and antisepsia [1, 2, 5, 6, 9, 11].

Once the needle is in the abdominal cavity, ambient air is passed through the needle using a 50 cm^3 syringe. The initial amount of 100 cm^3 is used to allow a small amount of distance to be introduced between the bowel and the Veress needle, thereafter catheter insertion is performed.

500–4000 cm^3 is used to inflate the cavity.

Once finished, an abdominal X-ray is performed to visualise the correct position of the catheter in the abdominal cavity and to visualise the pneumoperitoneum in the abdominal cavity.

The subsequent insufflation of the abdominal cavity can be performed as an inpatient procedure or in the physician office. Air is insufflated daily in an amount of 500–1500 cm^3 of ambient air via a syringe connected to a three-way stopcock which is connected to a mercury sphygmomanometer. Pressure should not exceed 15 mmHg during any given session [3, 8, 19, 23].

Gradual expansion of the cavity is performed, with the duration depending on hernia type and size, approximately 7–10 days in inguinal hernias, 9–28 days in ventral hernias. The total volume will range from 3800 to 5000 cm^3.

Insufflation is performed according to the patient's tolerance (besides not exceeding 15 mmHg). It is suspended when the patient manifest a feeling of fullness, pain, nausea, shortness of breath, tachycardia, hypertension, hypotension or decreased blood O_2 saturation [7, 12, 16, 19].

45.10 Preparing for PPP

1. The preparation of the patient with a hernia with loss of Domain begins with smoking cessation, respiratory therapy and placement of a belt or abdominal binder to prevent migration of air into the peritoneal sac [19].
2. DVT prophylaxis is administered daily as a single dose of low molecular weight heparin [7].
3. At the time of initial insufflation: Foley catheter placement and stomach decompression with nasogastric tube is performed.
4. Initial insufflation until the abdomen is taut is performed; PPP can reveal unknown defects [24].
5. Oral antibiotic therapy with cephalosporins or third generation fluoroquinolones is began before the start of the insufflation. A prokinetic agent like metoclopramide 10 mg every 8 h and analgesics are administered [1].

45.11 Preoperative Progressive Pneumoperitoneum Complications

PPP has a low complication rate (7 %) and is well tolerated by most patients. The discomfort associated with the procedure is epigastric pain, feeling of gastric fullness and early satiety, which decreases with administration of analgesics and prokinetic in nearly all of the patients.

Shoulder pain is the most frequent complaint in 41 % of patients with PPP, occurs early and is usually transient and moderate. It is caused by the stretching of the suspensory ligament of the liver that is not usually under tension and becomes tense when the PPP is established.

Diaphragmatic irritation or moderate wall stress may generate pain in the neck region. It is a sign to suspend insufflation.

Major complications which can occur as a product of the technique are: (a) accidental insufflation of air into the colon,

visceral perforation, peritonitis, solid organ injury, air embolism, (b) severe respiratory distress, pulmonary embolism, pneumonia, pulmonary embolism due to deep venous thrombosis, and myocardial infarction (rare complications) and (c) subcutaneous emphysema of the neck and chest and mediastinal emphysema [2, 6, 8].

Other complications include acute exacerbation of underlying lung disease, and wound hematoma.

PPP does not increase wound infection rates or other wound complications and does not affect the incidence of transoperative or post-operative complications [2, 12, 25].

An important contraindication for performing the procedure is the presence of a hernia with a small ring as this may lead to strangulation [4, 11].

45.12 Conclusions

The management of large hernias requires a multidisciplinary approach. PPP is recommended in the management of patients with giant hernias and a large volume of their viscera in the hernial sac, where it would otherwise not be possible to reintroduce the contents and make the repair, or where their reduction of the contents would lead to an abdominal compartment syndrome [6].

The PPP is used to restore the right of residence of the abdominal viscera before surgery that would otherwise be inoperable; the gradual air insufflation leads to an adjustment of intra-abdominal pressure with the progressive stretching of the abdominal fascia similar to the effects of pregnancy or the accumulation of fluid ascites [1, 26]. PPP will stretch the abdominal wall and increase the volume of the abdomen which facilitates reduction of the herniated content without ventilatory and circulatory compromise [8, 17, 19].

PPP is a simple procedure that can be performed under local anaesthesia and sedation on an outpatient basis; it is well tolerated by patients and is a safe and useful tool in the repair of giant hernias.

45.13 Clinical Case

Fifty-one-year-old female with psychomotor delay secondary to neonatal hypoxia. Forearm fracture at 46 years of age which was managed with surgery. Morbid obesity since she was 35 years of age. No previous abdominal surgery.

She began 10 years before her initial visit with progressive increase in her abdominal girth, which was accompanied by intermittent colicky abdominal pain of variable intensity which became worse after ingestion of meals and improved in the lateral decubitus position.

Weight 95 kg, 1.60 m tall, BMI 37.1.

Alert and oriented, holosystolic cardiac murmur, no aggregates heard on lung auscultation. Abdomen is large bulgy, yet soft. A very large ventral hernia with abdominal viscera is found, adequate peristalsis is heard (Figs. 45.1 and 45.2).

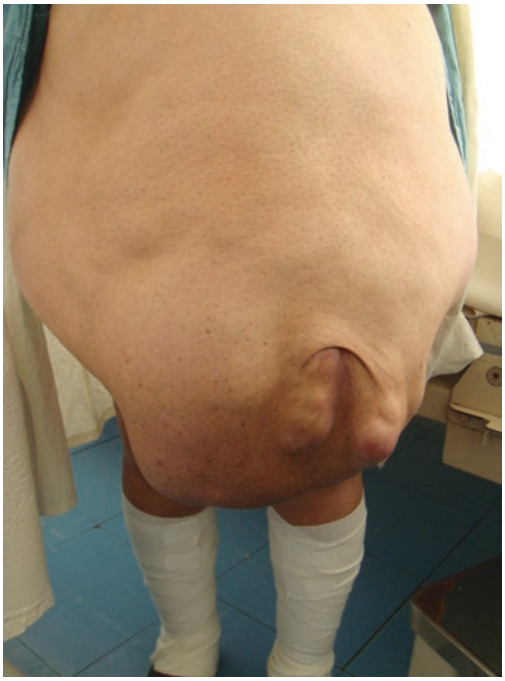

Fig. 45.1 Front view of the patient with her large abdominal wall defect (pre op)

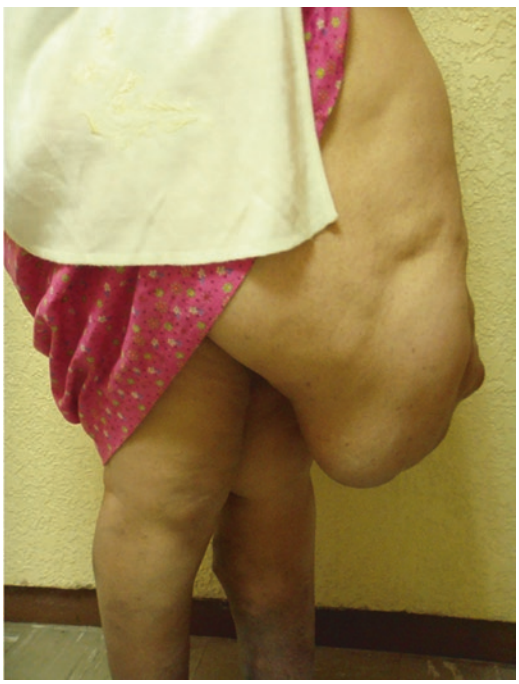

Fig. 45.2 Side view of the patient with her large abdominal wall defect (pre op)

Under sterile technique and local anaesthesia a puncture was performed using a Veress needle in the left hypochondrium, midclavicular line. A double lumen catheter was placed. Initial insufflation was performed with 2300 cm^3 of air; thereafter daily fillings with 2000 cm^3 were performed during 10 consecutive days. The second lumen of the catheter was connected to a sphygmomanometer to control insufflation pressure as described above. On her third day, she referred postprandial fullness which was not a contraindication for further insufflation (Figs. 45.3 and 45.4).

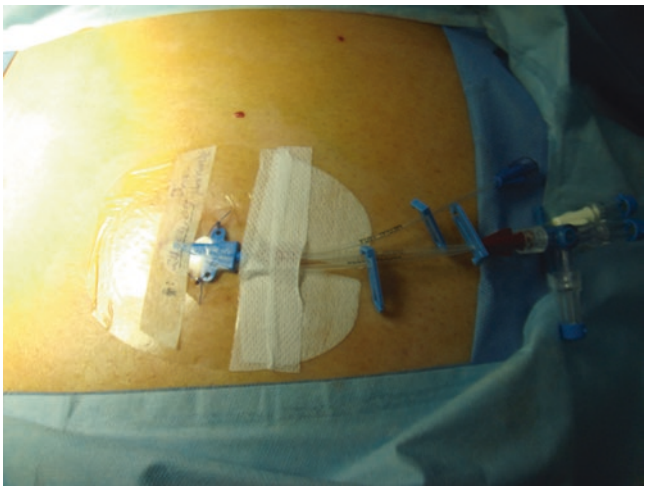

Fig. 45.3 Double lumen catheter placed in abdomen through which ambient air was insufflated

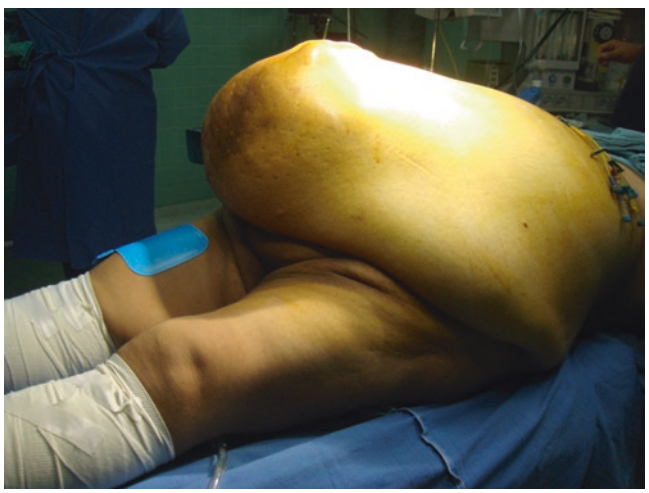

Fig. 45.4 Preoperative picture after 10 days of PPP (decubitus position)

After successful PPP, the abdominal wall reconstruction is performed using mesh and refunctionalization of the abdominal wall (Figs. 45.5 and 45.6).

Patient had an adequate post-operative course. She was discharged on her fourth post-operative day with outpatient follow-up performed thereafter (Fig. 45.7).

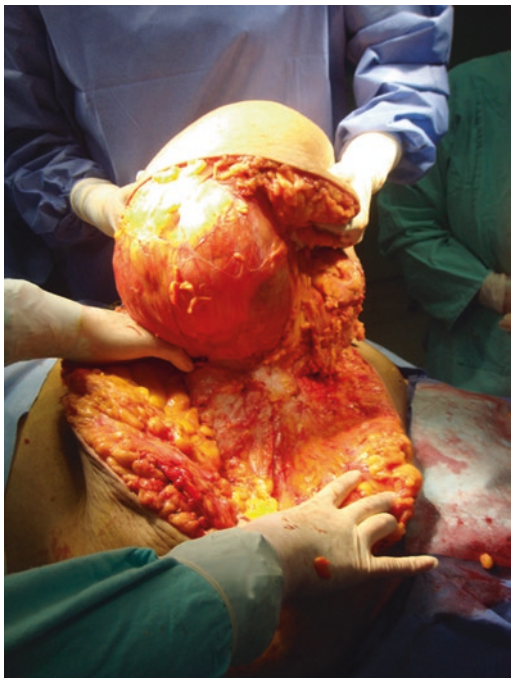

Fig. 45.5 Transoperative picture showing hernia sac and abdominal wall defect

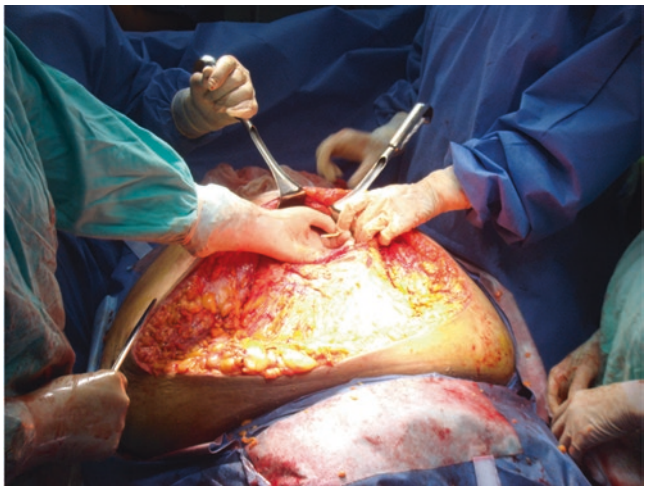

Fig. 45.6 Operative picture showing reduction of hernia

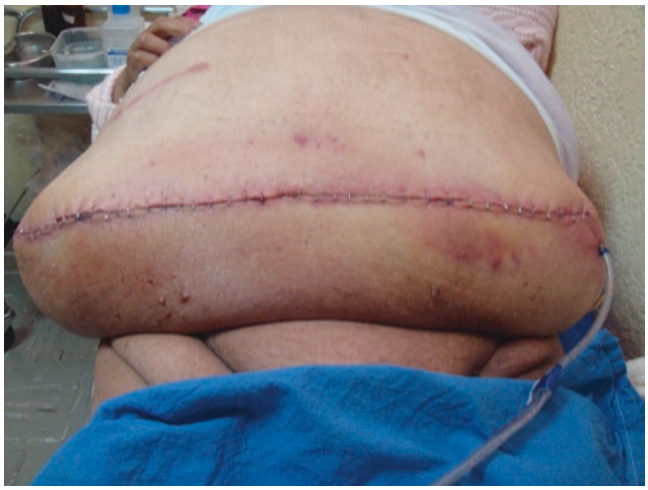

Fig. 45.7 Outpatient follow-up

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