

# Pathophysiology of giant incisional hernias with loss of abdominal wall substance

*Luigi De Santis, Flavio Frigo, Andrea Bruttocao<sup>1</sup>, Oreste Terranova<sup>1</sup>*

Unità Operativa di Chirurgia Generale - OC Valdagno (VI); <sup>1</sup>Clinica Chirurgica Geriatrica, Azienda Ospedaliera di Padova

**Abstract.** Incisional hernia represents the most common wound complication after abdominal surgery. The repair of large incisional hernias requires an accurate knowledge of the interactions between the tissues of the abdominal wall, the prosthetic materials and the bowel. At the same time a careful attention must be placed on the physiopathology of abdominal hypertension. Repair of giant incisional hernias with heavy loss of substance may take to a sudden increase of intra-abdominal pressure and, sometimes, to Abdominal Compartment Syndrome (ACS). The aim of preventing recurrences very often requires the use of a prosthesis, which must be placed on a low-tension environment to avoid early failures and excessive increase of intra-abdominal pressure. It is also necessary to employ as much parietal tissues as possible to prevent visceral adhesions and lesions and to pay attention to an appropriate employment of prosthesis. Utilization of composite materials, absorbable prosthesis or of combinations of mesh and flaps looks promising in preventing endo-abdominal hypertension without increasing the rate of recurrences, infections and adhesive complications.

**Key words:** Physiopathology, incisional, hernia

## Introduction

Surgery of giant abdominal incisional hernias is still challenging. Postoperative reduction of abdominal girdle and repositioning of viscera suddenly increase the pressure inside the abdomen. The aim of preventing recurrences very often requires the use of a prosthesis, which must be placed on a low-tension environment to avoid early failures and an excessive increase of intra-abdominal pressure. Nevertheless it is sometimes difficult to cover adequately the inner side of a mesh to prevent a direct contact with the intestine. These multiple requirements can yield different results in different patients. A good knowledge of the physiopathologic mechanisms underlying the repair is a must for adequate treatment since failures

are often due to lack of consideration of physiological aspects.

## Historical perspective

The first description of elevated intra abdominal pressure – intraabdominal hypertension (IAH) was recorded in 1863 when Marey and Burt demonstrated the relationship between intra-abdominal pressure and the respiratory function (1). In 1951, Baggot reported the high mortality associated with forced closure of the abdomen with distended bowel, addressing the fatal effect of severe IAH (2).

The use of prosthetic mesh as a fascial substitute or reinforcement has been widely reported in the last

decades. In wounds with unstable soft tissue coverage, however, the use of prosthetic mesh poses an increased risk for extrusion or infection. If the peritoneal coverage is incomplete or missing mesh prostheses pose a risk of tight adhesion and fistulization to the intestine. E-PTFE prostheses are safe even if in direct contact with intestinal loops but are less resistant to infection and poorly integrated in normal tissues.

## Physiopathology

### *Intra-abdominal-pressure (IAP) and Intra-Abdominal Hypertension (IAH)*

The most accurate and simple way to determine the IAP is indirectly by measurement of the bladder pressure using a Foley catheter. The bladder pressure can be considered equivalent to the IAP. A measurement can also be obtained via NG tube in stomach, by measuring the inferior vena cava pressure or, directly, via an intra-peritoneal catheter. IAH has been defined as a pressure reading greater than 20 mmHg (3).

### *Haemodynamic effects of increased intra-abdominal pressure*

Increasing intra-abdominal pressure leads to a fall in cardiac output, due mainly to compression of the inferior vena cava (IVC) and reduction in venous return to the heart. Cardiac output is reduced despite apparent rises in central venous pressure, pulmonary artery occlusion pressure and systemic vascular resistance (4, 5). This distortion of standard monitoring modalities makes diagnosis and treatment difficult. Pressure on the IVC predisposes to venous stasis and increased risk of pulmonary thromboembolism.

### *Pulmonary effects of increased intra-abdominal pressure*

Raised intra-abdominal pressure elevates the diaphragm and leads to decrease of thoracic compliance, a rise in peak airway pressure and intra-thoracic pressure, and subsequently, a reduced venous return to the heart. Basal atelectasia may develop with V/Q mismatch. Pulmonary vascular resistance increases. Increased work of breathing may take to respiratory failure with the need of mechanical ventilation. The

increase in airway pressures and the need of PEEP may also exacerbate barotrauma and contribute to the development of acute respiratory distress syndrome with further reduction of cardiac output.

### *Renal effects of IAH*

An acute increase in intra-abdominal pressure leads to oliguria and anuria probably due to compression of the renal vein and renal parenchyma. Oliguria is often the earliest sign of ACS. Renal blood flow, glomerular filtration are decreased with a corresponding increase in renal vascular resistance. No dilatation of collecting system is usually seen on abdominal U/S and there is no improvement with the positioning of urethral stents. The low cardiac output contributes to the poor renal perfusion (6).

### *IAH and Splanchnic Flow*

Increases in IAP have adverse effect on splanchnic flow; when it reaches 15 mm/Hg, there is a marked reduction in hepatic artery and portal venous blood flow, superior mesenteric artery blood flow decreases, leading to mucosal acidosis and oedema (7, 8). Hepatic ischaemia, if left untreated, may take to coagulopathy, hypothermia and acidosis, which further increase the IAP and take to ACS. Free oxygen radicals produced locally enter systemic circulation, leading to distant organ damage.

### *Intracranial complications of increased IAH*

The rise in intra-abdominal pressure and intrathoracic pressure leads to a rise in central venous pressure which prevents adequate venous drainage from the brain, leading to a rise in intracranial pressure, decrease of cerebral perfusion pressure, cerebral ischaemia and intracerebral oedema.

### *Abdominal wall problems*

The direct compressive effect of IAH causes reduction in abdominal wall blood flow inducing ischaemia and edema. This may then contribute to abdominal wound complications such as dehiscence or necrotizing fasciitis. IAH also reduces abdominal wall com-

pliance which may be further affected by tissue edema as a result of fluid resuscitation.

#### *Abdominal compartment syndrome (ACS)*

In 1984 Kron defined ACS as the end result of a progressive, unchecked increase in intra-abdominal pressure from a myriad of disorders that eventually leads to multiple organ dysfunction (9).

The diagnosis of ACS requires the measurement of an intra-abdominal pressure of 25 mm/Hg (30 cmH<sub>2</sub>O/urine) or more, and one or more of the following signs of clinical deterioration: oliguria, raised pulmonary pressure, hypoxia, decreased cardiac output, hypotension, acidosis. The confirmation of the diagnosis comes from a clinical improvement after abdominal decompression (10).

#### *Prosthetic materials and flaps*

The ideal prosthesis should be resistant, elastic, biologically neutral, without foreign body effect, should be resistant to infection, allow the ingrowth of connecting tissues and blood vessels without causing adhesions to bowel loops.

Unfortunately no material possesses all the above properties at the same time.

Polypropylene meshes are resistant, elastic, well integrated in the tissues but sometimes dangerous to the intestine. E-PTFE prostheses are well tolerated but poorly integrated in the tissues. If infected, they must be removed.

Composite prostheses seem to reduce the risks of complications by coupling the advantages of different materials, e.g. the adhesion to tissues of polypropylene and the neutrality to bowel loops of e-PTFE.

In difficult cases vascularized autogenous tissue may be required to achieve herniorrhaphy with stable coverage and repair may be done by conjugation of flaps and mesh (11).

#### **Discussion**

The treatment of abdominal wall defects remains a difficult problem. The achievement of a successful

repair, even in cases with heavy loss of abdominal wall substance, requires the prevention of a sudden increase of intra-abdominal pressure by use of tension free techniques. These techniques must combine prevention of local complications due to interaction between prosthesis and viscera with durable strength.

Intra-abdominal hypertension signs and symptoms need to be sought so that early diagnosis and treatment can be anticipated.

The use of different combinations of composite (polypropylene and e-PTFE) or resorbable prosthetic materials, of mesh with hydrophilic coatings and of mesh coupled with flaps can provide a solution, even in cases of abundant loss of abdominal wall substance, when adequate covering of the inner surface cannot be achieved with peritoneum or omentum (12, 13).

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Correspondence: Dr. Luigi De Santis  
Clinica Chirurgica Geriatrica  
Azienda Ospedaliera di Padova  
Via Giustiniani, 2  
35128 Padova  
Tel: 049-8213185  
Fax: 049-8213184  
E-mail: gerichir@unipd.it