



## RESEARCH ARTICLE

### ABDOMINAL COMPARTMENT SYNDROME AFTER A CHRONIC MESENTERIC ISCHEMIA: ABOUT ONE CASE

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#### ARTICLE INFO

##### Article History:

Received 16<sup>th</sup> October, 2020

Received in revised form

29<sup>th</sup> November, 2020

Accepted 07<sup>th</sup> December, 2020

Published online 30<sup>th</sup> January, 2021

##### Keywords:

Abdominal Compartment,  
Ischemia, Pressure, Decompression.

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#### ABSTRACT

Le sd du compartiment abdominale est une entité qui est rare mais grave secondaire à plusieurs étiologie dans les plus fréquent post traumatique, nous rapportant le cas d'un patient qui s'est présenté dans un tableau d'ischémie chronique bénéficié d'une revascularisation, en postopératoire le patient a présenté une aggravation de l'état hémodynamique avec une pression intra abdominale à 16 mmhg, raison pour laquelle il a été admis au bloc opératoire pour une laparotomie décompressive avec aspiration de 2 litre du liquide claire comme le décrivent les données de la littérature. Conclusion :Une fois le diagnostic est fait, une discussion collégiale entre chirurgie et réanimateur doit être faite afin de prendre une décision chirurgicale à temps.

#### INTRODUCTION

Mesenteric ischemia is an often unknown abdominal emergency and a rare cause of abdominal pain. It is the result of an interruption or decrease in splanchnic mesenteric blood flow. The severity and organ affected depends on the vessel involved and the development of collateral. Mesenteric ischemia can be arterial or venous, acute or chronic, occlusive or nonocclusive. The incidence is increasing and represents about one hospitalization per thousand 1 Mesenteric ischemia more frequently affects people over the age of 60 and preferably female. Abdominal compartment syndrome (ACS), described by analogy with traumatic and ischemic limb injury, occurs when intra-abdominal pressure (IBP) increases significantly.<sup>2</sup>

**Case report:** The patient is a 61 year old man, with a history of diabète type 2 and alcohol tobacco use. The patient suffered from post prandial and abdominal Pain chronic with a loss of 10kg in 3months. The cardiac evaluation and gastrointestinal exploration were négative, a computed tomography of the aorta and both lower limbs found an ischemia of the both mesenteric arteries responsible for a chronic ischemia mesenteric.

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The patient was admitted to the operating room for aorta iliac and aorta superior mesenteric artery bypass graft. Before the operation the patient was admitted to the resuscitation service where he had benefited of a right jugular catheter central and arterial catheter for cardiac output measurement by pulse wave contour analysis (vigileo). The induction was done by AIVOC: propofol 3ng/ml and sufentanil 0.15gamma/min, cisatracurium 4mg. Maintenance of anesthesia was done by AIVOC couple propofol-sufenta with metric depth of anesthesia by bispectral index. The exploration of the abdomen found no palpable pulses. The patient was under 3.5mg/h of noradrenaline with a heart rate of 82pulses/min, blood pressure 110/60mmhg, and a 300cc of urines in the end of the operation; the patient bled 100cc and received 2500cc of crystalline with no transfusion during all the 3 hours of the bypass graft operation. In post operative the patient was readmitted in the resuscitation service and was extubate one hour after his admission, the postoperative clinical examination where with no sign of gravity, until the 48h post operative, the patient present a State of shock: Anuria, PA 70/45mmhg under 7mg of noradrenaline and 5gamma/kg/min of dobutamine [cardiac index 2.1, vascular resistance systemic 284.5KPa-s-m<sup>2</sup> /l] the patient received 3litre of crystalloid and 500cc of colloid synthetic (Plasmion) with no amelioration of the hemodynamique state, The blood gas values were: aretrielle (pH: 7.27, pCO<sub>2</sub>: 34.5; PO<sub>2</sub>: 82 (under a masque with a 6litr/min of oxygen) HCO<sub>3</sub><sup>-</sup>: 16.2; lactates: 1.32) Venus (pH: 7.26, pCO<sub>2</sub>: 36.8; PO<sub>2</sub>: 48; HCO<sub>3</sub><sup>-</sup>: 16.8; lactates: 1.60) with a deltaPCO<sub>2</sub> of 2.3.

the abdominal pressure was monitored by urinary bladder pressures, she was at 28mmHg with a necessity of the readmission in operation room. The induction was done by etomidate 20mg + 100µg of fentanyl +50mg of rocuronium, the patient under 10mg of noradrenaline +10µg/kg/min of dobutamine, when her abdomen was opened, there was approximately 2litre of clear fluid within the peritoneal cavity. With the evacuation of the intra peritoneal fluid, the cardiac output where on amelioration after the evacuation passed from 1.2 to 2.5 also the hemodynamic state where progressively on amelioration. The bypass graft was intact. The surgeon put 3 drains on intraperitoneal and the peritoneal closure was done by separate points. The urinary bladder pressures at the end of the operation be claimed at 3, and the patient was returned to the intensive care unit, the postoperative suites were marked by an amelioration of the hemodynamic state (dégression of noradrenaline and dobutamine doses) and the resumption of duress.

## DISCUSSION

Abdominal Compartment Syndrome (ACS) is defined as a persistent and sustained increase in intra-abdominal pressure (IBP) resulting in impairment of cardiovascular, respiratory, digestive, renal and cerebral functions (3). The first effect of the persistent increase in AIP is to decrease the cave flow by compression of the inferior vena cava and portal vein and by increasing intrathoracic pressure. Venous return is gradually altered from an IAP of 15 mmHg (4-5). This is the result of an increase in intra abdominal and intrathoracic venous resistance leading to the drop in cellular, gate and retroperitoneal outflow. The maximum resistance to the cave flow occurs at the lower vena cava in its supra-hepatic infra-diaphragmatic portion, where the zone of high intra abdominal pressure meets that of low intrathoracic pressure (6-7). The ventilatory consequences of the elevation of the AIP appear from 15 mmHg. Sustained and prolonged elevation of IAP results in respiratory failure resulting in hypoxemia and hypercapnia (8,9). Renal function impairment is observed from an IAP of 15-20 mmHg (10). Increased intrathoracic pressure may be responsible for reduced cerebral venous return resulting in increased intracerebral blood volume and increased intracranial pressure (PIC). This was observed in the context of an acute increase in AIP in both humans and animals (11-12)

The most common cause of ACS has increased intraperitoneal volume from any source, including hemorrhage, edema, bowel distention, ascites, tumor, or massive fluid resuscitation. It has been described as a result of bowel reperfusion injury with "third-space" fluid losses and increasing bowel edema. In patients surviving repair of a ruptured abdominal aortic aneurysm, ACS can also occur from the retroperitoneal hematoma pushing the viscera anteriorly and diminishing intra-abdominal space. Extrinsic compression can also cause increased IAP and can be attributed to burn eschars, tight abdominal closures, and hernia repairs (13). The urinary bladder pressure is the most accurate and easiest method for determining IAPs. With this technique, initially described by Kron et al (14), a Foley catheter is used within the urinary bladder. The bladder is filled with 100 to 200 ml of sterile saline, and the drainage tubing is clamped off distal to the aspiration port. A 16-gauge needle connected to a transducer is placed into the aspiration port of the Foley catheter, and the

bladder pressure is measured. The symphysis pubis is used as the zero reference point. Disturbances in the various organ systems have been found to occur with urinary bladder pressures as low as 15 mm Hg (15). One of the treatments proposed is massive vascular filling. Indeed, the deleterious consequences of HIA seem to be increased by hypovolemia. However, recent data suggest an aggravation of the SCR by too much filling. Thus, optimization of the volume appears to be delicate and the close monitoring of the value of PIA could allow to guide the filling. Surgical decompression of the abdomen through aponeurotic discharge incisions is the reference treatment. Although the effectiveness of surgery in the reduction of HIA is evident, no prospective studies have shown improved patient survival. Only a retrospective controlled study that is not randomized and only published in summary form provides an argument for improving the survival of surgically decompressed scars. The indications are discussed on a case-by-case basis according to the suspected cause of SCA: aponeurotomy without laparotomy for a retroperitoneal hematoma, laparotomy for an SCA after abdominal trauma with packing. When the aponeurotomy does not allow a wall closure without tension, other techniques can be used: a scalable wall bag («Bogota Bag»), partial closure. In total, the therapeutic attitude remains poorly codified because it is based on results from a small series of patients (16). In our clinical case the patient presented a compartment abdominal syndrome after a mesenteric revascularization, that caused state of anuria and renal failure. A urinary catheter was installed to confirm the intra abdominal pressure, which was at 28 mm Hg, the therapeutic attitude was the surgical resumption after a discussion between the surgeon and resuscitator which consists of a deep compressive laparotomy with setup of 3 drain so closed by points separating from the abdominal aponeurosis, which led to an amelioration in our case.

## Conclusion

Abdominal compartment syndrome is a little-known clinical entity. Once this diagnosis is made, consultation between surgeon and resuscitator must take place to decide on the indication and timing of a laparotomy decompression.

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