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Midline Fasciotomy for Severe Acute Pancreatitis with Abdominal Compartment Syndrome

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DESCRIPTION

Abdominal Compartment Syndrome (ACS) is a progressive increase in intra-abdominal pressure above 20 mm Hg with new onset of thoracoabdominal organ dysfunction. Primary abdominal compartment syndrome refers to increased pressure due to injury or disease in the abdominal and pelvic regions. Secondary abdominal compartment syndrome refers to disorders that begin outside the abdomen, such as: Severe burns or sepsis. Increased intraabdominal pressure leads to organ failure, with the kidneys and lungs most commonly affected. Administration of ACS is multidisciplinary. Conservative management with appropriate volume, flexibility and positive hemodynamic support is the first step. Laparotomy decompression is indicated when ACS does not respond to conservative management and is associated with multiple organ failure. ACS can have a high mortality rate and successful treatment requires collaboration between physicians, critical care physicians, and surgeons. ACS has received increasing attention in critical care, and prevention of IAH and ACS is of critical importance in the care of critically ill, surgical, and trauma patients. The etiology of ACS is diverse and can be complex. Diagnosis is made by clinical symptoms and measurement of Intra-Abdominal Pressure (IAP). Continuous or continuous IAP measurement is essential for timely diagnosis, appropriate treatment and successful recovery of these patients. Measurement of bladder pressure is an excellent method of estimating IAP because it can be easily performed in patients at risk of significantly elevated IAP. A pressure greater than 12 mmHg is considered IAH and an IAP greater than 20 mmHg with new organ failure is ACS. Medical management is usually started first, and decompressive laparotomy is indicated if medical management fails. The development of ACS can have a significant impact on patient recovery and outcome. The rate of renal replacement therapy was much higher in his ACS than in patients with normal intra-abdominal pressure. Both intensive care and her 90 day mortality were also significantly higher in his ACS patients than in her normal IAP patients. Abdominal compartment syndrome is defined as persistent his IAP of at least 20 mmHg associated with *de novo* organ dysfunction/incompetence. It should be noted that the IAP ranges associated with these grades have been revised downward in recent years due to the recognized negative impact of elevated IAP on end-organ function. Physiologically, IAP increases with inspiration (diaphragm contraction) and decreases with expiration (diaphragm relaxation). The pathophysiology of ACS is multifactorial. Increased pressure in the abdomen may reduce blood flow to the organs because it first compresses the arterial inflow of the viscera and then the venous outflow of the viscera. Compression of blood vessels also damages heart function. Also, upward displacement of the diaphragm can lead to hypoventilation, respiratory rate changes, and ultimately hypoxia. This complex physiological change in the organ systems mentioned above applies to all body systems in terms of the effects caused by ACS. Elevated IAP can create a vicious cycle and lead to multiple organ failure. Increased intra-abdominal pressure compresses the IVC and reduces venous return, thus reducing venous return and cardiac output. Therefore, many organs suffered from low blood flow and exhibited organ dysfunction as a clinical manifestation. Aggressive fluid therapy may be prescribed, and tissue edema develops as intra-abdominal pressure increases. Abdominal Perfusion Pressure (APP), calculated as MAP minus IAP, and has been proposed as a predictor of visceral perfusion and a potential endpoint of resuscitation.

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CONFLICT OF INTEREST

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