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รายงานผู้ป่วย

Fulminant hepatic failure following abdominal compartment syndrome

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Increased intra-abdominal pressure has been shown to cause adverse consequences on various organ system. Decreased hepatic blood flow has also been observed. Here we report a case of fulminant hepatic failure following abdominal compartment syndrome in a patient with non-hepatic penetrating abdominal injury. The cause of abdominal compartment syndrome was intra-abdominal bleeding after the first operation for repairing of injuries to the stomach, duodenum, and inferior vena cava. Multiple organ failure, including acute fulminant hepatic failure, developed in spite of a successful decompressive celiotomy. Although the patient's clinical condition was improving after a period of intensive therapy, he eventually died from massive hemoptysis of non-specific etiology.

Key words: *Abdominal compartment syndrome, Hepatic failure.*

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สุวิทย์ ศรีอัษฎาพร. ภาวะตับวายอันเนื่องมาจากการมีความดันภายในช่องท้องเพิ่มขึ้นอย่างมาก. จุฬาลงกรณ์เวชสาร 2540 ก.พ;41(2): 149-54

การมีความดันภายในช่องท้องเพิ่มขึ้นอย่างมาก มีผลทำให้การทำงานของอวัยวะต่างๆ ทั้งในและนอกช่องท้องเลวลง ในภาวะดังกล่าวปริมาณเลือดที่ไปเลี้ยงตับจะลดลงด้วย ได้รายงานผู้ป่วยที่เกิดภาวะตับวายภายหลังการมีความดันภายในช่องท้องเพิ่มขึ้นอย่างมาก ผู้ป่วยถูกแทงที่ช่องท้องมีการบาดเจ็บต่อกระเพาะอาหาร, ดูโอเดนั่ม และ inferior vena cava ซึ่งได้รับการผ่าตัดซ่อมแซมแบบ primary repair หลังผ่าตัดเกิดภาวะความดันภายในช่องท้องเพิ่มขึ้นอย่างมาก (abdominal compartment syndrome) จากการมีเลือดออกในช่องท้อง ซึ่งรักษาโดยการผ่าตัดอีกครั้งหนึ่ง หลังจากนั้นผู้ป่วยเกิดภาวะตับวาย, ไตวาย, และการหายใจล้มเหลว ผู้ป่วยเสียชีวิตจากการมีเลือดออกมากในระบบทางเดินหายใจ

Abdominal compartment syndrome (ACS) is a condition in which increased intra-abdominal pressure causes derangements of various organ functions. Although the entity is still somewhat controversial, its clinical relevance has been increasingly mentioned.[1,5] The effects of increased intra-abdominal pressure on the renal, respiratory, cardiovascular, and gastrointestinal systems have been shown in clinical and experimental models.[2-10] Decreased hepatic blood flow has also been observed in animal studies.[11] Here, we present a case of fulminant hepatic failure following ACS.

Case presentation

A 30 year-old male was stabbed in the abdomen with a kitchen knife. He arrived at the hospital emergency room with a blood pressure of 130/80 mmHg, and pulse 92/min. Exploratory laparotomy revealed 1 cm through and through wounds of the stomach, 3rd part of the duodenum, and infrarenal inferior vena cava. All wounds underwent primary repair. During the three hours of operation, six units of blood and 3,500 ml of crystalloid solution were administered. The systolic blood pressure was above 100 mmHg at all time. After the operation the patient was admitted to the intensive care unit and was ventilated with a volume controlled respirator.

During the first 10 hours postoperation, the hemodynamics, airway pressure, blood chemistry, and blood gas values were within normal limits. The urine output was 50-100 ml/hour. After that, the abdomen became progressively distended with a drop of the hematocrit from 34% to 26%. The blood pressure was still in normal range, the central venous pressure (CVP) was 12 cmH_2O, and the urine output decreased to 5-20 ml/hour. His respiration became progressively difficult to manage with rising of the peak airway pressure. Three units of blood were subsequently transfused but these only raised the hematocrit value to 28%. At 14 hours postoperation, the abdomen was remarkably tense, the blood pressure was 140/90 mmHg, pulse 100/min, the CVP was 18 cmH_2O, the peak airway pressure was 65 cmH_2O, and the patient became anuric. At this time, the diagnosis of intra-abdominal bleeding with an ACS was made and the patient was brought to the operating room for a relaparotomy.

At operation, blood gushed out when the abdominal cavity was entered owing to a very high intra-abdominal pressure. Approximately 2,500 ml of blood was recovered from the peritoneal cavity. No active arterial bleeding was found. Some venous oozing from the retroperitoneal area was noted. All anastomoses were intact. The liver, spleen, stomach, and intestine appeared normal. Immediately after the abdomen was open, the airway pressure decreased to normal resulting in a dramatic improvement of ventilation. Urine flow reappeared 40 minutes later and subsequently increased to 100-200 ml/hour. The operation took 100 minutes and the abdomen was closed without tension.

Immediately after the operation, persistent hypoglycemia developed. The lowest plasma glucose detected was 27 mg/dL. The plasma glucose was in the range of 50-150 mg/dL in spite of a continuous infusion of 20 percent dextrose solution and intermittent infusion of 50 percent glucose solution. Twenty-four hours later, laboratory tests revealed TB 3.47 mg/dL, DB 1.81 mg/DL, SGOT 9,910 units/L, SGPT 3,281 units/L, AP 274 units/L, PT 23.8 seconds (control 12.5 seconds), albumin 3.2 gm/dL, globulin 1.7 gm/dL, BUN 35 mg/dL, and Cr 3.7 mg/dL. During the 2nd and 3rd day, the patient developed progressive jaundice with rising BUN and Cr levels and the urine flow was about 20-30 ml/hour. At the end of the 3rd day, the laboratory results revealed TB 8.94 mg/dL, DB 7.96 mg/dL, SGOT 664 units/L, SGPT 999 units/L, AP 230 units/L, PT 22.2 seconds (control 11.6 seconds), albumin 3.3 gm/dL, globulin 1.6 gm/dL, BUN 50 mg/dL, and Cr 5.9 mg/dL. The 3rd laparotomy was carried out to rule out intra-abdominal sepsis owing to progressive multiorgan failure. At operation, no fluid collection or evidence of intra-abdominal infection was found. The liver was congested. Both kidneys were tense. A liver biopsy was performed, and the pathologic sections revealed centrilobular hemorrhagic necrosis as is seen in hemorrhagic shock. During the post-operative period the patient received intensive supportive therapy for multi-organ failure including total parenteral nutrition, hemodialysis, and respiratory support.

At the end of the second week of admission, the laboratory tests revealed TB 30 mg/dL, DB 27.6 mg/dL, SGOT 104 units/L, SGPT 72 units/L, AP 328 units/L, PT 18.9 seconds (control 12.9 seconds), BUN 97 mg/dL, and Cr 3.3 mg/dL. The HBsAg was negative. The antiHBc and antiHCV were positive. On day 17th of admission, while the patient's general condition was improving, he developed a fatal hemoptysis. An autopsy revealed congested liver, but the other intra-abdominal findings appeared normal. The cause of hemoptysis could not be specifically defined.

Discussion

Although the intra-abdominal pressure of this patient had not been measured, the diagnosis of ACS was made from classic clinical pictures of tense abdomen, high peak airway pressure and anuria, all of which were corrected by decompressive celiotomy. The cause of the ACS in this subject was intra-abdominal bleeding from veins of the retroperitoneal area. Clearly, if the patient had been operated earlier, ACS should not have occurred. Massive intra-abdominal bleeding resulting in ACS has been previously reported.[2]

Progressive deterioration of the hepatic function, leading to acute liver failure, may occur in complicated trauma patients without ACS. However, such a serious event is usually associated with certain predisposing factors i.e. prolonged shock, severe liver injuries, and sepsis. In this case report, the above mentioned factors were not present. Although a certain amount of

blood had been lost, and this might have been responsible for hepatic ischemia, the effects of such hemorrhage should not have resulted in fulminant hepatic failure as occurred in this patient. The rate of bleeding was relatively slow and the intravascular volume could be easily and effectively replaced. The patient experienced no episodes of profound prolonged hypotension. The systolic blood pressure was almost always above 100 mmHg and the CVP was always in high or supra normal levels. We believed that the increased intra-abdominal pressure in this patient must have compounded the detrimental effects of blood loss on the liver, resulting in severe ischemic hepatitis and fulminant hepatic failure.

Increased intra-abdominal pressure has been shown to cause decreased visceral blood flow, including hepatic arterial, portal venous, and hepatic microcirculatory blood flow.[11,12] Although significant clinical effects of ACS on a previously normal liver are uncommon, the fatal fulminant hepatic failure in our patient should alert surgeons for it's potential risks in critically injured patients.

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