中文題目:缺血性腸壞死合併急性成人呼吸窘迫症候群

英文題目:Ischemic bowel disease with ARDS

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Background: The mortality rate of ischemic bowel disease is high and emergent management will be necessary. The pathophysiology of ischemic bowel disease is reduced blood flow to the intestine, which cause ischemia and necrosis if no reperfusion for a long period. The primary etiology is: vasculopathy, which included mesenteric artery emboli (53%), mesenteric artery thrombosis (26%), NOMI (non-occlusive mesenteric ischemia) (12%), and mesenteric venous thrombosis (9%). The other etiology may be secondary to extrinsic vascular compression or tumor. Volvulus, strangulation, and adhesion may also cause intestinal ischemia. We will present a young male patient who was brought to our hospital due to cardiac arrest.

Case report: This is a 40-year-old man with underlying disease of parenchymal liver disease with suspected liver cirrhosis without regular OPD follow up. According to his mother's statement, this patient had complained of abdominal cramping pain with vomiting since last night and the symptoms became more severe this morning. And then he was brought to local medical department. Because of consciousness loss with low blood pressure, he was transferred to our hospital emergently. Cardiac arrest was noted during the period of transferring, and CPCR started immediately on the ambulance. At our ER, there was no spontaneous breathing and no pulsation. Bilateral pupil size 5.0/5.0 without light reflex, seizure episode was noted at ER and fresh blood 550 ml from NG tube. After CPCR and intubation with mechanical ventilator support, ROSC was noted and brain CT scan was arranged for differential diagnosis of consciousness change, which showed no ICH. For critical condition, he was admitted to our ICU. After admission, marked high blood ammonia concentration was noted, besides, electrolytes imbalance with hypocalcemia and hyperphosphatemia were found. We arranged prophylaxic antibioites use with ceftriaxone. For abdominal fullness with muscle guarding, abdominal CT was performed and revealed diffuse bowel necrosis and pneumatosis intestinalis with portal vein gas accumulation, which considered ischemic bowel disease. Otherwise, ARDS pattern was also noted. Serious arterial blood gas showed severe metabolic acidosis. Although intensive sodium bicarbonate solution supplementations were given, the vital signs did no become stable. After GI consultation, surgical intervention was suggested, but dilated pupils. After discussing with GS doctor, surgical intervention with diffuse bowel resection was considered, but even if extremly high dose of inotropic agent use with dopamine, levophed, bosmin, profound shock was still noted and PEA was noted again and we started CPCR and EKG still showed asystole post CPCR 1 hour. After discussing with family, we stopped CPCR and declared expired.

Discussion: The risk factors of ischemic bowel disease included congestive heart failure, peripheral vascular disease, coronary artery bypass surgery or other vascular surgeries, recent heart attack, colon cancer, diabetes, hemodialysis, shock, strangulated hernia, oral contraseptives and pregnancy, polcythemia, amyloidosis, radiation, trauma, and old age. But our patient had no above risk factors. The morphologic and metabolic changes during ischemia is increased transcapillary filtration, intestinal edema, net flux of fluid into the bowel lumen, and then increased mucosal permeability of luminal bacteria, which cause cellular death and transmural necrosis. Diagnostic testing included of marked leukocytosis if transmural infarction, elevated serum amylase, metabolic acidosis (serious and late finding. Otherwise, plain abdominal film & CT scan may revealed thickened bowel wall, thumbprinting, adynamic ileus, and gasless abdomen. Pneumatosis intestinalis and portal vein gas will be found only in severe cases.

Conclusion: The treatment included of replacement of lost fluid, correct hemocon-centration & acidosis, prevent infection, CVP, Swan-Ganz catheter, cardiac function optimized, 30 minutes for assessing the viability of bowel and resection of obviously necrotic bowel. The mortality rate was about 71% (59~93%) in the past 15 years, and up to 90% if bowel wall infarction. Survivors usually have significant long-term morbidity because of the reduced intestinal mucosal surface available for absorption.