
Lethal non-occlusive mesenteric ischemia after pelvic fracture due to falling down

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Abstract: An 85-year-old female who had fallen down and bumped her left hip into the floor 3 days earlier could not move due to the pain. She was transferred to a local medical facility and received a diagnosis of hypoglycemia, anemia, rhabdomyolysis, minor traumatic subarachnoid hemorrhage and pelvic fracture. She had no remarkable medical history. On arrival, she had subcutaneous hemorrhaging on her face and left hip. She complained of epigastralgia, but a physical examination of her abdomen revealed no suggestive findings. Whole-body CT revealed only pelvic fracture. She initially received a diagnosis of gastric ulcer due to stress concerning epigastralgia. She underwent massive infusion and transfusion; however, her oliguria did not improve. On the second hospital day, her epigastralgia continued. Her respiratory function deteriorated, so she received tracheal intubation with mechanical ventilation. As her oliguria did not respond to furosemide, continuous hemofiltration was also initiated. Vasopressor administration was also commenced for hypotension in order to maintain her blood pressure. However, her general condition did not improve, and her abdomen became distended. We suspected delayed intestinal injury due to trauma and performed enhanced CT on the fourth hospital day, which revealed segmental, enlarged, non-enhanced small bowel, corresponding to non-occlusive mesenteric ischemia (NOMI). Her family did not agree to surgical resection for the necrotic bowel because of her elderly age. Unfortunately, conservative therapy failed to obtain a successful outcome. Critical-care physicians should pay attention to complications of NOMI, especially when critical ill patients complain of epigastralgia.

Keywords: non-occlusive mesenteric ischemia; pelvic fracture; epigastralgia

INTRODUCTION

Acute mesenteric ischemia is a rare disease and classified into three categories: acute superior mesenteric artery (SMA) occlusion, mesenteric venous thrombosis (MVT) and non-occlusive mesenteric ischemia (NOMI). According to an Acosta report, the ratios of acute SMA occlusion (embolus/thrombus ratio = 1.4), MVT and NOMI were approximately 68%, 16%, and 16% [1]. NOMI is characterized by gastrointestinal ischemia with normal vessels that leads to increased permeability, bacterial translocation and further mucosal hypo perfusion due to macrovascular vasoconstriction, hypo perfusion of the tips of the villi and shunting [2]. It is very common in critically ill and perioperative patients but also occurs in patients with pancreatitis, burns, renal failure and sepsis [2]. The mortality rate due to NOMI remains high (50-70%) [1-4]. However, NOMI after blunt trauma has never been reported. We herein report a fatal case of pelvic fracture due to fall accompanying NOMI.

CASE PRESENTATION

An 85-year-old female who had fallen down and bumped her left hip into the floor 3 days earlier could not move due to the pain. When she did not attend a local meeting in her community as she regularly had, a

neighbor called the police, and they found the patient unconscious in her home. She was transferred to a local medical facility and received a diagnosis of hypoglycemia, anemia, rhabdomyolysis, minor traumatic subarachnoid hemorrhage and pelvic fracture (**Figure 1**). She underwent tracheal intubation and infusion of glucose and was transferred to our department.

She had no remarkable medical history. On arrival, her Glasgow Coma Scale score was E4VTM6. She had a blood pressure of 118/84 mmHg, a heart rate of 100 beats per minute (BPM), a SpO₂ of 96% under room air and a body temperature of 35.4 °C. As she could obey commands, the tracheal tube was removed. On a physical examination, she had subcutaneous hemorrhaging on her face and left hip. She complained of epigastralgia, but a physical examination of her abdomen revealed no suggestive findings. A venous gas analysis revealed a pH of 7.29, PCO₂ of 40 mmHg, HCO₃⁻ of 19 mmol/l and a base excess level of -6.5 mmol/l.

The main results of a biochemical analysis of the blood were as follows: white blood cell count of 11,100/μl, hemoglobin level of 7.6 g/dl, platelet count

of $29.7 \times 10^4/\mu\text{l}$, total protein level of 5.2 g/dl, aspartate aminotransferase level of 348 IU/L, alanine aminotransferase level of 250 IU/L, blood urea nitrogen level of 48.8 mg/dl, creatinine level of 2.22 mg/dl, creatinine phosphokinase level of 7835 IU/l, c-reactive protein level of 7.0 mg/dl, activated partial thromboplastin time of 37.8 (27.5) seconds and international normalized ratio of prothrombin time of 1.40. Whole-body computed tomography (CT) revealed only pelvic fracture with retroperitoneal hematoma. The bowel did not have any abnormal lesions.

She initially received a diagnosis of gastric ulcer or acute gastric mucosal lesion due to stress concerning epigastralgia. After admission to the intensive-care unit, she underwent massive infusion and transfusion; however, her oliguria did not improve. On the second

hospital day, her epigastralgia continued. Her respiratory function deteriorated, so she received tracheal intubation with mechanical ventilation. As her oliguria did not respond to furosemide, continuous hemofiltration was also initiated. Vasopressor administration was also commenced for hypotension in order to maintain her blood pressure. However, her general condition did not improve, and her abdomen became distended. We suspected delayed intestinal injury due to trauma and performed enhanced CT on the fourth hospital day, which revealed segmental, enlarged, non-enhanced small bowel, corresponding to NOMI (Figure 2). Her family did not agree to surgical resection for the necrotic bowel because of her elderly age. Unfortunately, conservative therapy failed to obtain a successful outcome.

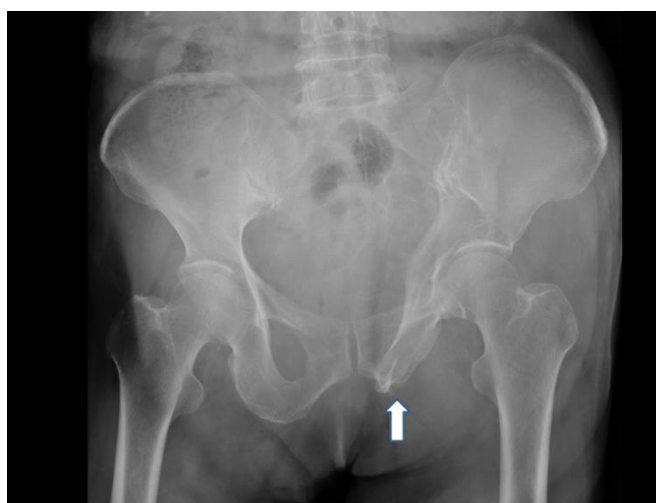


Fig 1: Radiography of the pelvis. Roentgen reveals left pelvic fracture.

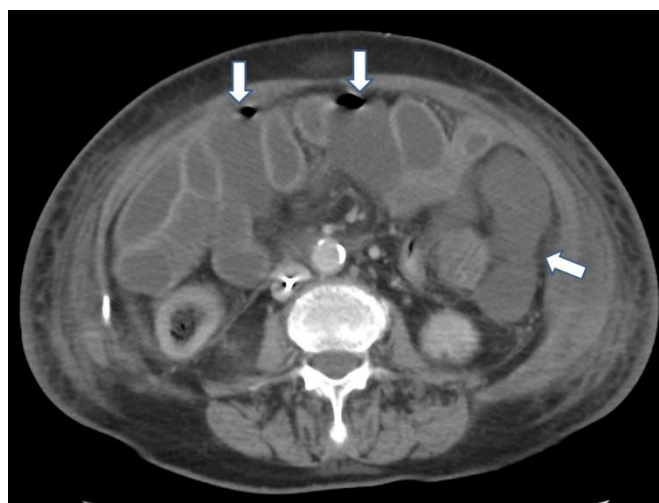


Fig 2: Enhanced CT on the fourth hospital day. CT shows segmental, enlarged, non-enhanced small bowel (white arrow), corresponding to non-occlusive mesenteric ischemia.

DISCUSSION

Hypoperfusion of peripheral mesenteric arteries has a number of different potential causes [3]. The risk of developing NOMI increases with age, probably due to arteriosclerosis in the peripheral

arteries [3,5]. A low cardiac output underlying cardiac disease or insufficiency circulation can lead to peripheral hypo perfusion. The activation of the sympathetic system results in an increased cardiac output and further vasoconstriction of the peripheral

mesenteric vessels. In addition, vasopressors induce smooth muscle contraction of the arteriolar wall. The increased renal blood flow caused by furosemide then leads to a diminished mesenteric perfusion, probably due to the furosemide-related activation of the renin-angiotensin-aldosterone system with subsequently increased levels of angiotensin II. Other causes of mesenteric vasospasms are various forms of shock, septicemia, dehydration and hypotension following dialysis. Our case probably had signs of ischemic mesenteric artery due to insufficient circulation due to hemorrhaging from her pelvic fracture, dehydration due to restriction of drinking or eating based on painful immobilization after falling down and the activation of the sympathetic system, in addition to her high age. Furthermore, the infusion of furosemide and vasopressors and the induction of hemodialysis may have deteriorated her mesenteric ischemia [3]. Because these treatments for critical ill patients are standard therapy, even in elderly patients, critical-care physicians should pay attention to complications of NOMI, especially when critical ill patients complain of epigastralgia.

Conflict of interest: The authors declare no conflicts of interest in association with this study.

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