Case Report

Sudden Unexpected Death from Ischemic Colitis Following Post-traumatic Hospitalization: A Case Report

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Ischemic colitis (IC) is a serious gastrointestinal condition that can be fatal in older adults with multiple health problems. We report the case of an 81-year-old man who developed IC after a long period of hospitalization for multiple traumatic injuries. Factors such as limited mobility, impaired bowel function, advanced age, cardiovascular/metabolic disorders, and side effects of medications collectively increased vulnerability to colonic ischemia. Despite routine monitoring and stable vital signs, the patient's condition suddenly deteriorated, resulting in death. Autopsy revealed transmural hemorrhagic necrosis of the sigmoid colon and pan-peritonitis, illustrating how IC can progress silently. Clinicians should be aware of gastrointestinal complications in bedridden older patients or those taking drugs that affect bowel movements. From a forensic perspective, this case demonstrates that death may be linked to the downstream effects of trauma rather than to the injuries themselves, emphasizing the importance of careful postmortem examination. Preventive care, close monitoring of bowel function, and early recognition of warning signs are essential for reducing the risk of fatal outcomes in high-risk populations.

Key Words: Ischemic colitis; Sudden death; Trauma; Hospitalization; Autopsy

Introduction

Ischemic colitis (IC) is a condition in which the blood supply to the colon is reduced, causing inflammation and necrosis, and was first described by Boley et al. in 1963 [1]. Clinically, IC is not a rare condition; however, most cases improve with conservative treatment. Therefore, they are rarely the subject of medicolegal autopsies.

We recently encountered a case of sudden death due to IC during hospitalization after a traffic accident. We report this case, underscoring both its clinical challenges and relevance in forensic medicine.

Case Report

An 81-year-old man was admitted to our hospital after a pedestrian traffic accident involving a cargo truck. He had a history of hypertension, diabetes mellitus, benign prostatic hyperplasia, dementia, dyslipidemia, and surgery for lumbar spinal stenosis. He was diagnosed with multiple traumatic injuries, including fractures of the cervical, thoracic, and lumbar vertebrae, sternum, and ribs, as well as dislocation of the hip joint. Hip dislocation was reduced, and other injuries were managed conservatively. Over the course of his acute hospitalization, he gradually stabilized, and after a period of recovery, he was transferred to a rehabilitation hospital for long-term supportive care after 1 month.

At the rehabilitation hospital, he experienced recurrent episodes of abdominal discomfort, and plain radiographs obtained at that time revealed findings consistent with paralytic ileus. The patient was conservatively managed with bowel rest and hydration. In addition to the gastrointestinal complaints, the patient developed neuropsychiatric disturbances, including insomnia, hallucinations, and episodes of aggressive behavior. Despite these issues, follow-up laboratory tests and routine daily monitoring of vital signs revealed no remarkable abnormalities. However, nearly 3 months after the accident, the nursing staff found him unresponsive. Immediate cardiopulmonary resuscitation was performed, but the patient could not be resuscitated.

An autopsy was performed on the third day after death. Externally, decomposed discoloration was observed on the abdomen. Upon opening the abdominal cavity, approximately 200 mL of hemorrhagic purulent exudate was identified (Fig. 1A). Examination of the gastrointestinal tract revealed hemorrhagic necrosis of the sigmoid colon (Fig. 1B, C), and a large amount of feces was found inside. In addition, marked atherosclerosis was identified in the abdominal aorta (Fig. 1D) and both common iliac arteries. The heart showed mild cardiomegaly and coronary atherosclerosis
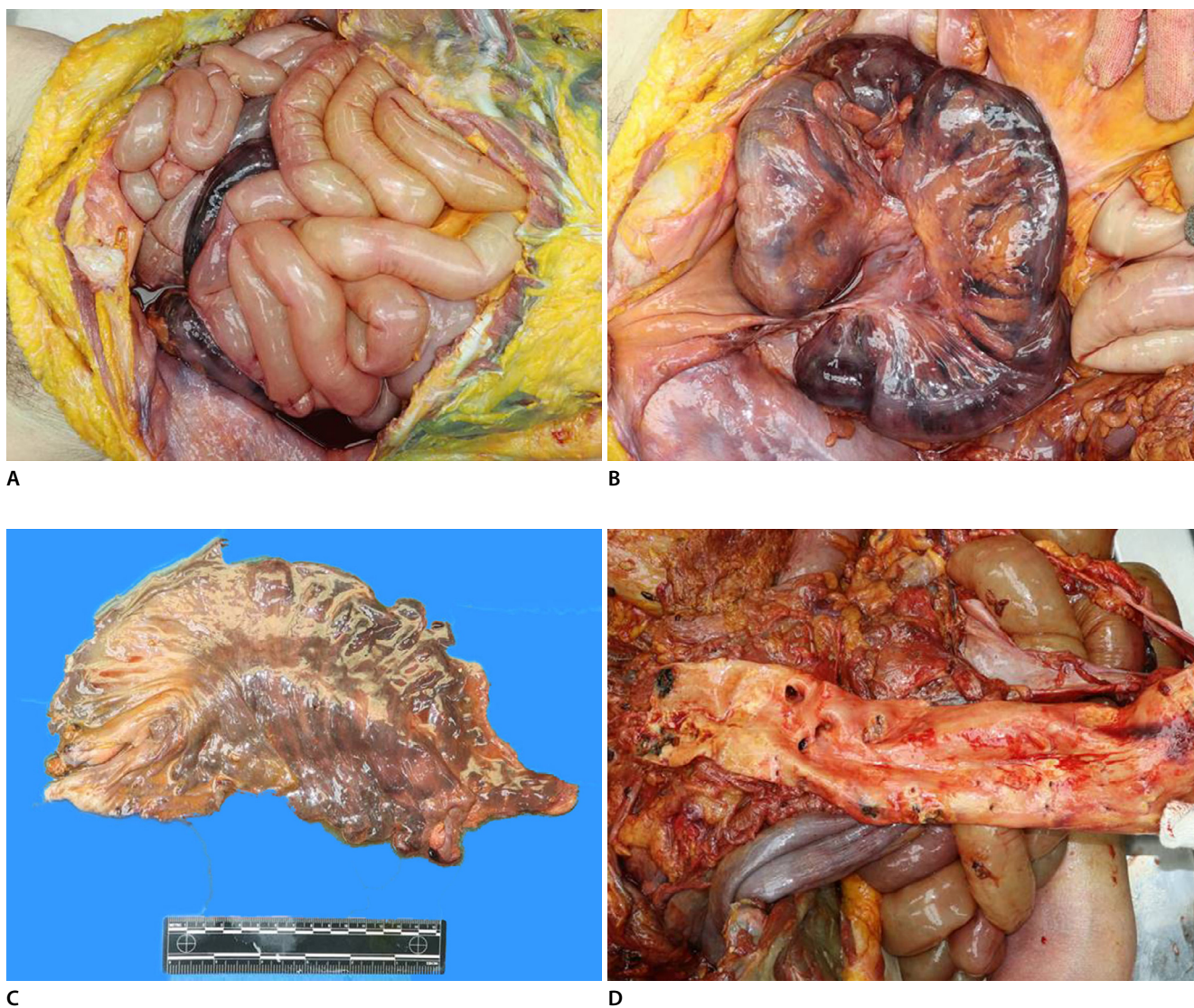

Fig. 1. Gross examination revealed purulent exudate in the abdominal cavity (**A**) and hemorrhagic necrosis of the sigmoid colon (**B**, **C**). Additionally, marked atherosclerosis was identified in the abdominal aorta (**D**).

with calcification in the left anterior descending coronary artery.

Microscopic examination revealed a transmural hemorrhagic infarction of the sigmoid colon (Fig. 2A), accompanied by acute suppurative inflammation extending to the serosa (Fig. 2B). Acute nonspecific inflammation was also noted in the mesentery and diaphragm; therefore, these findings were diagnostic of IC complicated by pan-peritonitis. Hepatocyte necrosis with cholestasis was identified, and benign nephrosclerosis and diabetic nephropathy were observed.

In the toxicological analysis, tramadol, acetaminophen, quetiapine, valproic acid, bisoprolol, donepezil, haloperidol, and risperidone were within the therapeutic range.

Overall, the cause of death was determined to be IC of the sigmoid colon, complicated by panperitonitis.

Discussion

The colon is richly supplied by the branches of the superior and inferior mesenteric arteries, and its extensive collateral circulation generally protects it from ischemia. IC is the most common form of intestinal ischemia, with an estimated incidence ranging from 4 to 44 cases per 100,000 person-years and occurring predominantly in older adults [2,3]. The disease most frequently involves watershed zones, including the splenic flexure (Griffith's point) and the rectosigmoid junction (Sudeck's point), where the blood supply is relatively tenuous [4].

The pathophysiology of IC is multifactorial. Non-occlusive mechanisms account for most cases and are typically triggered by systemic hypotension, hypovolemia, or vasoconstrictive medications [5,6]. Occlusive etiologies such as arterial thromboembolism or advanced atherosclerosis are less common but are often associated with more severe outcomes [7]. Histologically, IC is characterized by mucosal and submucosal necrosis, congestion, and hemorrhage, with progression to transmural infarction in advanced disease. Clinically, patients may present with acute abdominal pain, hematochezia, or bloody diarrhea; however, the spectrum of the disease ranges from mild, self-limiting episodes to fulminant gangrenous colitis requiring urgent colectomy [8,9].

Although most patients recover with conservative management, prognosis worsens considerably in cases of transmural ischemia or perforation. Right-sided colonic involvement, advanced age, cardiovascular comorbidities, elevated serum lactate levels, and diagnostic delays are considered poor prognostic factors [10,11]. Importantly, IC has forensic significance because it may cause unexpected sudden deaths. In such instances, a detailed postmortem examination,
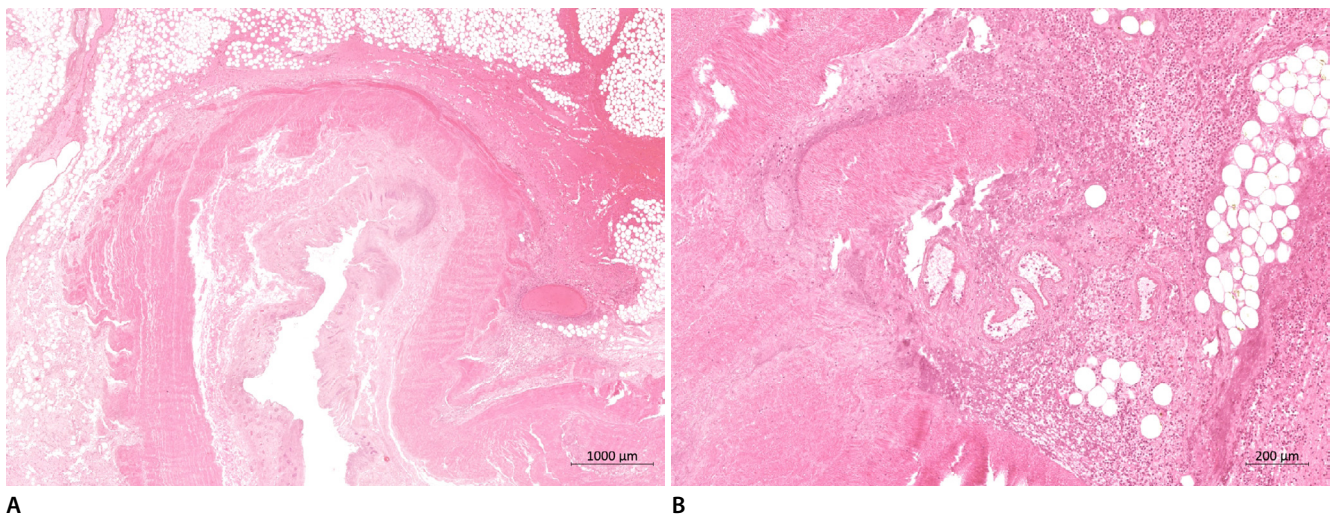
Fig. 2. Microscopic findings in the sigmoid colon. The low-power view shows transmural hemorrhagic infarction (**A**, H&E, ×10). Acute nonspecific suppurative inflammation is identified in the outer muscle proper and serosa (**B**, H&E, ×100).

including gross pathology and histology, provides an essential clinicopathological correlation to establish IC as the cause of death [12].

The present case highlights the complex interplay among multiple risk factors for the development of fatal ischemic colitis. The patient sustained multiple rib, sternal, vertebral, and hip injuries due to the accident, necessitating prolonged immobilization and transfer to a rehabilitation hospital. Extended bed rest significantly restricted mobility, impaired bowel motility, and worsened constipation, which are factors that predispose patients to colonic ischemia [13]. These circumstances were compounded by advanced age and major comorbidities, including hypertension, dementia, and poorly controlled diabetes mellitus, all of which are established risk factors for vascular compromise and impaired tissue resilience [13]. Furthermore, a review of the rehabilitation hospital records confirmed repeated episodes of ileus and stool retention, suggesting that colonic dysfunction had progressed before the fatal event.

Medication history also contributes to the risk profile. Toxicological analysis has demonstrated that therapeutic levels of analgesics, antihypertensives, and antipsychotics are associated with adverse effects such as constipation, gastrointestinal dysmotility, and reduced physical activity, which further increases the risk of IC in vulnerable patients [14,15]. Thus, although drug toxicity per se was excluded as a cause of death, the side-effect burden of the prescribed medications must be considered an important aggravating factor in the pathogenesis.

The suddenness of clinical deterioration is another notable aspect. In elderly and bedbound patients, abdominal pain and warning signs of colonic ischemia may be blunted or nonspecific. Even when vital signs appear stable shortly before death, fulminant IC may progress silently to peritonitis and septic shock, leading to abrupt collapse [16]. Therefore, the absence of prior alarm signals in this case is consistent with the well-documented insidious and unpredictable course of IC in frail elderly patients.

From a forensic perspective, the determination of causality is crucial. Although IC was not a direct traumatic injury, it developed in a clinical context that was clearly precipitated by the consequences of the accident, namely, multiple fractures, immobility, and prolonged rehabilitation. Without the accident and its sequelae, the patient would have been far less likely to enter a high-risk state culminating in IC and death. Accordingly, the death may be regarded as a complication or an indirect consequence of the accident, rather than a natural event. This distinction has significant medicolegal implications, particularly when assessing the degree of responsibility attributable to the driver who caused the traffic accident.

In conclusion, this case demonstrates that ischemic colitis, while fundamentally a disease of vascular insufficiency, may arise as a fatal complication in elderly patients with a combination of immobility, comorbidities, side effects of medication, and pre-existing vascular disease. Forensic pathologists must go beyond identifying the immediate cause of death and consider how trauma and its sequelae indirectly contribute to fatal outcomes. Clinicians should recognize that even in the absence of direct abdominal injury, the chain of causation may extend from accidents to immobilization, bowel dysfunction, ischemic colitis, and ultimately death.

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