OXFORD

Oxford Medical Case Reports, 2022, **2**, 47–49
https://doi.org/10.1093/omcr/omac002
Case Report

Diabetic ketoacidosis-associated gangrenous ischaemic colitis masquerading as acute pancreatitis and differentiated using computed tomography

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Abstract

Diagnosis of an acute abdomen during an episode of diabetic ketoacidosis (DKA) is crucial for providing appropriate treatments and obtaining favourable outcomes, but may be difficult due to its considerable overlap with multiple intra-abdominal diseases in terms of clinical course and laboratory findings. In this study, we presented a case showing signs of an acute abdomen with sharp rises in serum pancreatic biochemical markers during the treatment of DKA with pyelonephritis. Contrast-enhanced computed tomography (CT) was performed to confirm the onset of acute pancreatitis; however, pneumatosis intestinalis and poor enhancement of the rectal wall were detected, indicating the presence of rectal infarction. Hartmann's procedure was immediately performed, and histological examination of the resected specimen revealed gangrenous ischaemic colitis. The present case highlights DKA as a risk factor of ischaemic colitis and the role of contrast-enhanced CT in the differential diagnosis of an acute abdomen in hyperglycaemic crisis.

INTRODUCTION

Ischaemic colitis occurs when there is a transient or sustained compromise in blood flow that fails to meet the metabolic needs of the colon [1]. This leads to mucosal ulceration, inflammation, haemorrhage and eventually bowel infarction [1]. Different types of ischaemic colitis, including transient colitis, chronic ulcerative colitis with or without stricture, and gangrenous colitis, are differentiated based on their clinical course and severity [2]. Gangrenous colitis has a high mortality rate of up to 40% and requires urgent surgical intervention [1]. Diabetic ketoacidosis (DKA) plays a critical role in the development of ischaemic colitis due to mesenteric collapse as it results in a serious reduction of fluid volume [2, 3]. We herein describe a patient with gangrenous ischaemic colitis who presented with an acute abdomen during an episode of DKA that was difficult to differentially diagnose from acute pancreatitis, which is also a common comorbidity of DKA [4].

CASE REPORT

A 61-year-old man who had been treated for type 2 diabetes mellitus (T2DM), hypertension, dyslipidaemia and spinocerebellar degeneration with orthostatic dysregulation, dysarthria, neurogenic bladder and faecal incontinence at a local clinic was referred to our department for inadequate glycaemic control with impaired consciousness (Glasgow Coma Scale E2V1M4). On admission to the emergency room, he had a body temperature of 36.1°C, blood pressure of 84/56 mm Hg, pulse of 104 bpm and respiration rate of 24 breaths/min. Physical examination showed clear lungs and no cardiac murmurs on chest auscultation, non-palpable liver and spleen, decreased bowel sounds, no abdominal distension, mass or bruit, and no oedema in the extremities. Laboratory examination showed high levels of fasting plasma glucose (1500 mg/dl) and glycated haemoglobin (HbA_{1c} 10.6%). DKA was diagnosed based on the detection of metabolic acidaemia (pH 7.209) with an increased anion gap (27.7 mEq/L) due to high serum ketone levels (4.04 mmol/L). Urinalysis revealed that the urine was macroscopically cloudy, and urinary occult blood, ketone, and sugar were strongly positive. Leukocytosis (27.1×10^9/L) with predominant neutrophils (92.0%), high serum C-reactive protein levels (4.54 mg/dl), and pyuria with leukocytes ≥10/high power field in the urinary sediment were suggestive of concomitant pyelonephritis. Biochemistry analysis indicated a marked increase in serum blood urea nitrogen (BUN) (139.2 mg/dL), creatinine (5.10 mg/dL), potassium (7.7 mEq/L), creatinine kinase (CK 3480 IU/L) with predominant CK-MM isozyme (BB 0%, MB 2%, MM 98%), and myoglobin levels (10 899 IU/L) representing

Received: August 17, 2021. **Revised:** December 13, 2021. **Accepted:** January 3, 2021

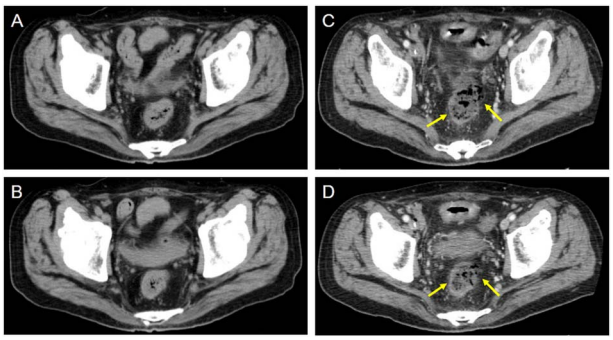

Figure 1. Computed tomography of the intrapelvis showed no apparent abnormalities on admission (A and B), but revealed pneumatosis intestinalis and poor enhancement in the rectal wall on Day 2 of hospitalization (C and D, *arrow*).

renal failure with electrolyte abnormalities and rhabdomyolysis. The electrocardiogram reflected the patient's hyperkalaemic status with a marked high-peaked T wave in leads $V_{2–4}$ and wide QRS complexes in all leads. Non-contrast-enhanced computed tomography (CT) analysis on arrival revealed no significant findings in the head, chest, abdomen or intrapelvis (Fig. 1A and B).

Treatment with continuous intravenous infusion of isotonic saline (0.9% saline solution) and regular insulin was initiated after the diagnosis of DKA, but haemodialysis was not performed. Piperacillin/tazobactam was also intravenously administered after blood and urine cultures were obtained. Streptococcus species were detected with a bacterial count of $\geq 10^5$ colony-forming units/ml in the urine culture test. Streptococcus, a weak pathogen, accounts for only 1–2% of causative bacteria of urinary tract infections. However, the patient had poorly controlled DM and neurogenic bladder, which could have contributed to the development of streptococcal-related pyelonephritis [5]. The results of multiple blood cultures were negative for bacteraemia. For DKA, plasma glucose and β-hydroxybutyrate levels in capillary blood were corrected to 378 mg/dl and 0.1 mmol/L, respectively, 18 hours after the initiation of treatment. Serum potassium levels were dropped to 5.7 mEq/L, which almost normalized the electrocardiogram findings at that time. However, the treatment failed to ameliorate renal failure as serum BUN and creatinine remained high, i.e. 128.3 mg/dl and 5.22 mg/dl, respectively. Impaired consciousness and haemodynamic instability continued, and diffuse distension and rigidity of the abdomen were newly observed. Intestinal peristalsis remained diminished, and no melena was observed. Serum pancreatic biochemical markers that were normal at the time of admission (amylase 98 IU/L) rose remarkably (amylase 1026 IU/L, pancreatic type amylase 1013 IU/L, and lipase 1201 IU/L). Contrast-enhanced CT was performed to elucidate coexisting acute pancreatitis, which revealed pneumatosis intestinalis and poorly enhanced bowel walls of the rectum (Fig. 1C and D), but no evidence of severe pancreatitis was found. There were no mesenteric artery and venous occlusion due to emboli and thrombosis.

The patient underwent Hartmann's procedure, including segmental resection of the rectum and sigmoid colostomy formation. The resected rectum was clearly demarcated from the non-necrotic area and the necrotic mucosa was a circumferential lesion (Fig. 2A). Histological examination of the specimen revealed typical findings of gangrenous ischaemic colitis, such as coagulative mucosal necrosis, submucosal haemorrhage and necrotising muscle layer infiltrated with numerous inflammatory cells including neutrophils (Fig. 2B–E). The patient recovered uneventfully after surgery and subsequent rehabilitation practices, and he has then maintained proper glycaemic control (HbA_{1c} 5.9–6.3%) with oral glucose-lowering agents and normal renal function for 2 years after being discharged.

DISCUSSION

Six risk factors including hypoalbuminaemia (<3.5 g/dl), older age (≥60 years), haemodialysis, hypertension, DM and use of constipation-inducing medication are associated with the development of ischaemic colitis [2]. Of these, the patient had four risk factors: older age (61 years), hypoalbuminaemia (2.9 g/dl) and a history of hypertension and T2DM. His abnormal bowel habits including recurrent constipation and diarrhoea due to spinocerebellar degeneration might have increased the visceral intraluminal pressure, leading to worsened bowel wall ischaemia.

The CK isozyme CK-BB is found in the mucosa and muscularis throughout the gastrointestinal tract [6, 7]. After intestinal infarction, serum CK-BB levels immediately rise, peak at 6 hours, and rapidly drop within 24 hours, even in the presence of progressive irreversible bowel infarction [6]. In this case, serum CK-BB isozyme was not detected at the time of admission; therefore, bowel infarction was likely to be initiated after hospitalization. Clinically, ischaemic colitis frequently occurs in the descending and sigmoid colon rather than in the rectum as the three arteries (right and left iliac arteries, and the superior rectal artery) supply blood to the rectum. This characteristic rectal circulation might have contributed to the slow progression of ischaemic colitis before the intestine became gangrenous during his hyperglycaemic crisis.

Acute pancreatitis occurs in 11% of adults with DKA, whereas non-specific elevations in serum amylase and lipase levels are observed in 40% of DKA patients [4, 8]. High serum amylase levels are also associated with bowel infarction, and the magnitude of hyperamylasaemia is related to the degree of infarction [9]. Therefore, bowel infarction and acute pancreatitis may show similar clinical picture, and it is difficult to clearly distinguish between the two based solely on pancreatic biochemical marker values [9]. This case supports the finding that contrast-enhanced CT imaging should be considered in DKA patients to exclude acute pancreatitis

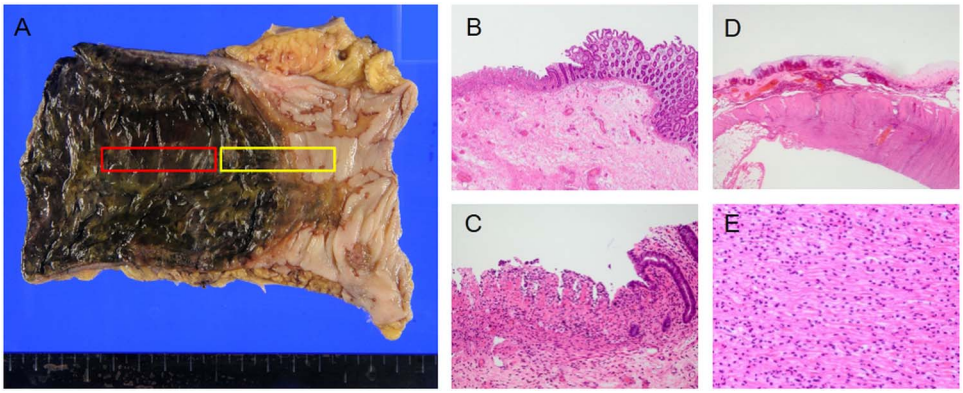

Figure 2. (A) Gross appearance of the resected specimen revealed an entire circumferential necrotic lesion of the rectal mucosa. The lesion had a relatively clear border and several linear and map-like mucosal ulcerations in the non-necrotic region. Histopathological examination of the resected rectum revealed (B) a lesion with clear demarcation from the area of unaltered crypt architecture, and (C) sloughing of the epithelium with loss of ductal epithelial cells (ghost-like appearance) (see yellow square in Fig. 2A). The examination also revealed (D) mucosal coagulative necrosis and submucosal haemorrhage, and (E) necrotising muscle layer infiltrated with numerous inflammatory cells including neutrophils (see red square in Fig. 2A). Haematoxylin and eosin staining: original magnification: (B) ×40, (C)×100, (D)×20 and (E)×400.

when serum amylase levels rise more than three times the normal upper limits [10].

In conclusion, clinical course and laboratory findings in DKA patients may be non-specific to distinguish severe ischaemic colitis from other intra-abdominal diseases, including acute pancreatitis. Evaluation of contrast-enhanced CT imaging may provide useful information for prompt diagnosis and appropriate treatment of intestinal ischaemia in order to achieve a favourable outcome.

ACKNOWLEDGEMENTS

The authors thank Dr Yuichi Kataoka (Department of Emergency and Critical Care Medicine, Kitasato University School of Medicine, Sagamihara, Japan) for his collaboration.

CONFLICT OF INTEREST STATEMENT

None declared.

ETHICAL APPROVAL

The present case report meets ethical guidelines and adheres to the local legal requirements.

CONSENT

Written informed consent was obtained from the patient for the publication of this case report.

GUARANTOR

Hiroaki Iwasaki.

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