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A CASE OF ACUTE MESENTERIC ARTERY ISCHEMIA INDUCED BY ALCOHOLISM

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Abstract: *Acute Mesenteric Ischemia, known as one of the vascular diseases of the intestine, is one of the significant causes of acute abdominal pain that is not considered initially but has a high mortality rate [50-70%] in acute abdominal cases. A 55-year-old male patient was presented to the emergency room of our hospital with abdominal pain starting at night and was admitted to the surgery service. His radiological examination results were normal, however, widespread gangrenous areas were detected in terminal ileum during the surgery. Necrotic bowel segment was resected. Accompanying pathological conditions were not detected in etiology of the patient. However, the patient had been using alcohol regularly for 20 years, which was an interesting characteristic of the patient.*

Keywords*: Mesenteric artery, ischemia, alcohol*

Introduction

Acute Mesenteric Ischemia (AMI) is a disorder known as the "disease of the elderly" and, although it is uncommon, it is difficult to make a diagnosis for it without any clinical suspicion [1]. 2/3 of the cases are acute thromboembolic occlusion of the superior mesenteric artery, while 1/3 of them are non-occlusive mesenteric ischemia and venous mesenteric thrombosis [2]. AMI which is generally associated with cardiovascular system diseases [CVS: ischemic heart disease, hypertension, congestive heart failure, arrhythmia etc.], diabetes mellitus [DM], chronic obstructive pulmonary disease [COPD] and other additional diseases [such as kidney disease, liver disease, coagulopathies, cerebral vascular diseases] is a 50-70% mortal disease due to the masking by accompanying pathological conditions, lack of any specific clinical symptoms, delayed diagnosis and delayed surgical intervention [1,3,4]. This paper presents a case without any pathological condition and with accompanying alcoholism.

Case Presentation

Our case was a 55-year-old male patient who was admitted to the hospital with the complaint of severe abdominal pain starting at night. The medical history of the patient included no condition other than

using alcohol regularly for the past 20 years. Physical examination showed extensive tenderness in the abdomen, defense and rebound in the whole abdomen and hyperactive bowel sounds. In the radiological examination of the patient, whole abdominal ultrasonography and upper and lower abdominal tomography scans were normal and abdominal free fluid was not detected. Loss of density compatible with diffuse liver steatosis as well as calcifications in aorta and its branches were monitored. In biochemical and haematological examinations, leukocyte count was detected as 20.60 K/uL [reference range: 4-11 K/uL], haemoglobin as 17.7% [reference range: 11.5-18%] MCH as 32.40 Pg [reference range: 27-32 Pg], thrombocyte as 348.00 10^9/L [reference range: 140-150 10^9/L], urea as 41 mg/dl [reference range:13-43 mg/dl], creatinine as 1.16 mg/dl [reference range: 0.6-1.2 mg/dl], direct bilirubin as 0.29 mg/dl [reference range: 0.0-0.5 mg/dl], total bilirubin as 0.93 mg/dl [reference range: 0.3-1.2 mg/dl] and C-reactive protein as [CRP] 14.2 mg/dl [reference range: 0-6 mg/dl]. Furthermore, serum CPT and GGT values were found to be high [56.20 U/L and 72 U/l; reference ranges 0-40 and 0-55 U/L respectively]. Since acute abdominal manifestations were diagnosed through physical examination, laparotomy was performed and approximately 300 cc gangrene fluid was detected. In the exploration of terminal ileum, gangrene and false membranes were present at the segment starting 10 cm proximal up to about 80 cm proximal of the ileocaecal valve [Figures 1].

Figure 1. Necrotic ileal intestinal segment.

Gangrenous segment was resected. During the preparation for anastomosis, the nutrition of an approximately 20 cm segment of the small bowel was impaired in the proximal end. The resection was expanded to include this segment. The continuity was maintained with end-to-end anastomosis. 2x100 ml Human Albumin was administered to the patient with 2.35 g/dl albumin. 1.16 mg creatinine value of the patient was detected to be 0.93 mg/dl on the day of discharge. Urea level increased up to 60 mg/dl post-operatively and decreased to normal levels on the 5th day. Direct and total bilirubin increased very little on the first day of post-operation and was within normal levels during and following operation. Changes in CRP were interesting; it was 14.2 mg/dl during admission and went up to 152 mg/dl a day later. It fell back to 44.9 mg/dl on the 2nd day of post-operation. As the patient developed no complications on the 5th day of post-operation, he was discharged with advice and instructions. Small bowel and terminal ileum with acute ischemic necrosis was monitored in the pathology report of the patient.

Discussion

Some scientists point out that the age of developing Acute Mesenteric Ischemia is 65 and above, however, they have not been able to indicate its incidence due to lack of available data. They also state in a study that mortality rates of patients increased even when they underwent surgery, as a result of the reduction of mesenteric blood flow after the initial 24 hours and the onset of symptoms [1,2]. Furthermore, various publications suggest that there is a “golden time” of 7-8 hours throughout the initiation of pain and arterial obstruction and that normal physical examination findings show that mesenteric artery occlusion must be considered in the presence of abdominal pain [5].

Doppler Ultrasonography was reported to have no value in the diagnosis of non-occlusive mesenteric ischemia and to not make a thorough diagnosis of ischemia even in the presence of severe arterial stenosis. However, computed tomography of the abdomen is considered to be useful in intestinal ischemia associated with non-occlusive mesenteric ischemia [5]. We believe that mortality was prevented in our case by the performance of surgery within 24 hours following the onset of symptoms. In Acute Mesenteric Ischemia cases, a specific biochemical diagnosis method that can give clinicians a

clue into detecting prognosis is not available yet. Leukocytosis level is generally between 12.000-20.000/mm^3; about 50% of the patients have metabolic acidosis which is a late symptom indicating the bowel infarction, and about 25% of them have hyperamylasemia [3,6]. Intestinal necrosis, pre-renal azotemia, hypoxemia and bacteremia are accompanied by increase in serum lactate, phosphate and alkaline phosphatase levels [5]. In an experimental study on rabbits which were infected with acute mesenteric ischemia, serum creatinine and inorganic phosphorus levels were detected to increase starting from the 2nd hour and remain high for 24 hours. In the same study, it was considered that creatinine kinase level could be more precise and a sign of poor prognosis could be present. Moreover, it is not clearly known whether the changes in biochemical values such as inorganic phosphorus and creatinine kinase were an early diagnosis factor or a prognostic factor [7]. Ischemia in organs usually leads to decreased bioactive agents and ATP, to apoptosis and necrosis, and eventually to functional loss of organs and acceleration of the reperfusion of blood flow. Thus, endothelial cell damage increases and also an inflammatory response occurs. Due to the deficiency of complements such as IgM and CRP [C-reactive protein] identified for ischemic reperfusion, complement supplementation is sought [8]. In many cases, leukocyte count, blood urea nitrogen [BUN], creatinine, liver enzyme and amylase levels were found high; in cases resulting in mortality, preoperative leukocytosis, high liver enzyme level and high BUN-creatinine level were statistically insignificant. However, bilirubin level was found significant in more than half of the cases examined for mortality. There are differences between publications in terms of the relationship of biochemical data and mortality findings. In some publications, the presence of leukocytosis is usually deemed a risky factor in terms of mortality, while sGOT [Aspartate transaminase / Glutamic oxaloacetic transaminase] and BUN values are regarded significant in terms of mortality in elderly patients [4]. In our case, a significant leukocytosis was present and bilirubin levels were normal. During admission of the patient, liver enzyme levels increased slightly, however, these values showed a significant increase on the 5th day of post-operation. A significant increase was not observed in creatinine values.

The changes in CRP were interesting; it was 14.2 mg/dl during the admission and went up to 152 mg/dl a day later. According to our clinical experience, the long duration between the initiation of complaints and the application to the hospital in acute abdominal or inflammatory cases may cause a significant increase in this value. However, the initiation of our patient's abdominal pain and his application to the hospital were within one day, and it was an interesting finding . Since we usually miss the initial phase in such inflammatory cases or do not have the chance to check the CRP at the beginning of clinical findings, we have not had the opportunity to evaluate it compared to other cases. This can be explained through a study on rats assessing mesenteric ischemia, where intestinal ischemia caused an accumulation of CRP, C_3 and Ig M within the 1st hour and in the 3rd hour following reperfusion [8].

Acute Mesenteric Artery Ischemia is known to mostly occur concomitantly with COPD, DM and CVS diseases [1, 3, 4]. In a study, however, JAK-2 V617F gene mutation positivity and associated Hepatitis-B compatible with myeloproliferative diseases were detected [9]. In another study including 18 cases operated due to Acute Mesenteric Ischemia, alcohol association was observed in the youngest case [10]. In a study on dogs, mesenteric ischemia developed in the group given ethanol alcohol through a catheter, while no change was observed in the control group [11]. A case study reports that the use of a drug named propranolol caused acute mesenteric ischemia in a patient with hyperthyroidism [12]. It is known that alcohol consumption has many adverse effects such as atherosclerosis, myocardial infarction and stroke. Although mesenteric ischemia directly associated with alcohol consumption has not been

reported, alcohol is known to have a direct toxic effect on small intestinal mucosa, decrease the intestinal permeability against toxic substances, lead to the thrombotic occlusion of arterioles by suppressing fibrinolysis and induce ischemia by mesenteric vasoconstriction. The same study reports that an 80-year-old patient using alcohol chronically developed alcohol-associated colonic ischemia during his treatment and was taken to an urgent operation [13]. A study reports that non-alcoholic steatohepatitis may predispose to prothrombotic [14].

Our case is a 55-year-old patient with chronic alcohol use. The lack of any predisposing factor in his etiology and his young age suggest that his long-term use of alcohol may have caused the mesenteric artery ischemia. If the diagnosis was delayed any longer, the 55-year-old patient could have lost the chance for treatment. Alcoholism perhaps may take part in Padua Prediction Score predicts risk of venous thromboembolism in acutely ill hospitalized medical patients [15]. In conclusion, given the increasing use of alcohol in our country and all over the world, acute mesenteric ischemia should be considered in patients with acute abdominal pain and alcohol use history, thus mortality can be reduced by performing early surgical intervention. Acute mesenteric ischemia can be seen as a predisposing factor in alcoholism.

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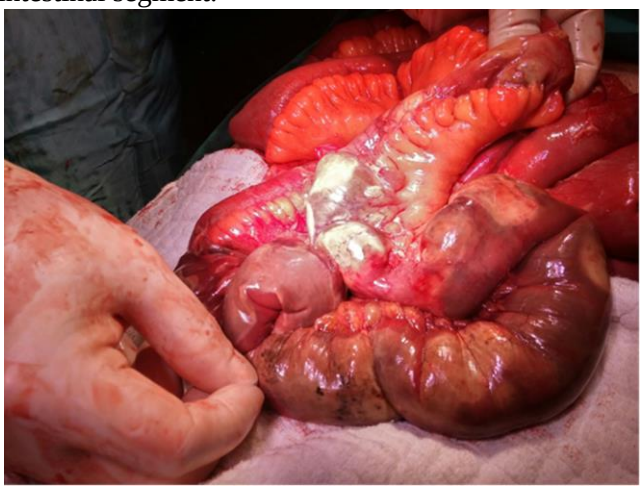
Figure 1. Necrotic ileal intestinal segment.



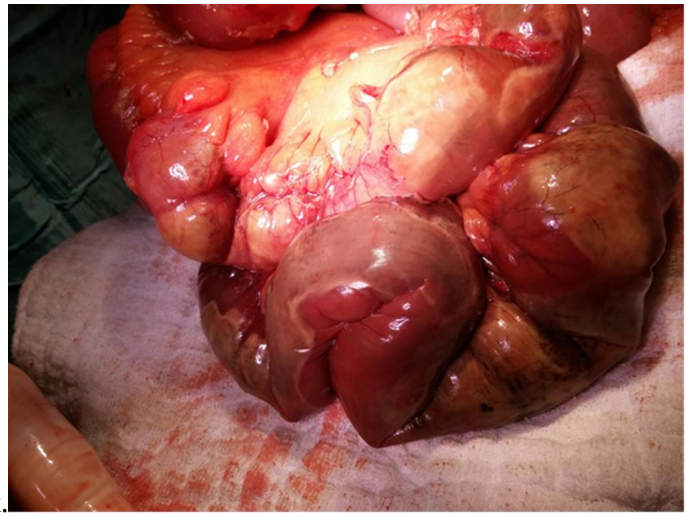


Figure 2. Necrotic ileal intestinal segment.