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OPIS PRZYPADKU
CASE REPORT

Spontaneous large hematoma of rectus abdominis muscle as complication of plasma coagulation disorders in 32-year-old man with terminal liver cirrhosis

Samoistny duży krwiak mięśnia prostego brzucha jako powikłanie osoczowych zaburzeń krzepnięcia u 32-letniego mężczyzny z marskością wątroby w stadium terminalnym

Grzegorz K. Jakubiak, Mikołaj Pietrzak, Agata Stanek, Grzegorz Cieślar

Katedra i Oddział Kliniczny Chorób Wewnętrznych, Angiologii i Medycyny Fizykalnej, Wydział Nauk Medycznych w Zabrzu, Śląski Uniwersytet Medyczny w Katowicach / Department of Internal Medicine, Angiology and Physical Medicine, Faculty of Medical Sciences in Zabrze, Medical University of Silesia, Katowice, Poland

ABSTRACT

Liver cirrhosis and its complications constitute one of the most important causes of hospitalization in internal medicine departments. Liver cirrhosis is not a homogeneous disease entity, but is the final stage of many different liver diseases, and the most important causes leading to the development of liver cirrhosis are alcoholic liver disease and viral hepatitis. One of the typical complications of liver cirrhosis are symptoms of bleeding diathesis. The most common bleeding complication in patients with liver cirrhosis is bleeding from the upper gastrointestinal tract. The aim of this publication is to present a case report of a 32-year-old man in the terminal stage, hospitalized due to decompensated alcoholic cirrhosis of the liver, who during hospitalization developed a spontaneous large hematoma in the rectus abdominis muscle on the left side. The presented case report shows that in the care of patients with decompensated liver cirrhosis, vigilance must be maintained to recognize bleeding complications, including those with an atypical clinical course.

KEYWORDS

liver cirrhosis, alcoholic liver disease, hemorrhagic diathesis, hematoma

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Address for correspondence: dr n. med. Grzegorz K. Jakubiak, Katedra i Oddział Kliniczny Chorób Wewnętrznych, Angiologii i Medycyny Fizykalnej, Śląski Uniwersytet Medyczny w Katowicach, ul. Stefana Batorego 15, 41-902 Bytom, tel. +48 32 786 16 30, e-mail: grzegorz.jakubiak@sum.edu.pl

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STRESZCZENIE

Marskość wątroby oraz jej powikłania stanowią jedną z najważniejszych przyczyn hospitalizacji na oddziałach internistycznych. Marskość wątroby nie stanowi jednorodnej jednostki chorobowej, lecz jest końcowym stadium wielu różnych chorób wątroby, a najważniejszymi przyczynami prowadzącymi do jej rozwoju są alkoholowa choroba wątroby oraz wirusowe zapalenia wątroby. Jednym z typowych powikłań marskości wątroby są objawy skazy krwotocznej. Najczęstszym powikłaniem krwotocznym u chorych z marskością wątroby jest krwawienie z górnego odcinka przewodu pokarmowego. Celem niniejszej pracy jest przedstawienie opisu przypadku 32-letniego mężczyzny w stadium terminalnym, hospitalizowanego z powodu niewyrównanej alkoholowej marskości wątroby, u którego w trakcie hospitalizacji wystąpił samoistny duży krwiak w obrębie mięśnia prostego brzucha po stronie lewej. Opisany przypadek pokazuje, że w opiece nad chorymi z niewyrównaną marskością wątroby należy zachowywać czujność pod kątem rozpoznania powikłań krwotocznych, również takich o nietypowym przebiegu klinicznym.

SŁOWA KLUCZOWE

marskość wątroby, alkoholowa choroba wątroby, skaza krwotoczna, krwiak

INTRODUCTION

Liver cirrhosis is not a homogeneous disease entity, but a condition that may develop in the course of many different liver diseases. Cirrhosis is characterized by progressive liver fibrosis, the replacement of normal liver parenchyma with connective tissue, the formation of regenerative nodules, progressive loss of liver function, and the development of hypertension in portal circulation [1]. Table I presents a summary of the most important causes leading to the development of liver cirrhosis.

Table I. Most important pathological processes leading to liver cirrhosis [2,3]

Causes of liver cirrhosis
Alcoholic liver disease
Chronic hepatitis C
Nonalcoholic fatty liver disease (NAFLD)
Chronic hepatitis B
Hemochromatosis
Wilson's disease
Primary biliary cirrhosis
Primary sclerosing cholangitis
Autoimmune hepatitis
Idiopathic or cryptogenic cirrhosis

In the initial stages of the disease, patients usually do not have typical clinical symptoms, but the characteristics of liver cirrhosis can only be detected by additional tests, especially fibrosis assessment by elastography. When typical features of portal hypertension and clinical symptoms of liver failure occur, decompensated cirrhosis can be discussed. The pathophysiology of liver cirrhosis and its complications is extremely complex. The most important features of liver failure include hyperbilirubinemia, hypoalbuminemia, prolonged prothrombin time, and hyperammonaemia. As a result, jaundice, peripheral oedema, and fluid accumulation in the serous cavities of the body, the development of hepatic encephalopathy, and bleeding can occur. As a result of the development of portal hypertension, splenomegaly, platelet sequestration, and varices of the oesophagus and anus occur [3,4,5].

The aim of this publication is to present a case report of a 32-year-old terminally ill patient with decompensated alcoholic cirrhosis complicated by progressive liver failure, who spontaneously developed a large hematoma in the rectus abdominis muscle as a result of plasma coagulation disorders.

CASE REPORT

Anamnesis and physical examination

A 32-year-old man was admitted to the Clinic as part of the emergency internal medicine service. In the anamnesis, he complained of vomiting, diarrhoea, yellowing of the skin, and progressive weakness for several days. At that time, the patient had not been treated for chronic diseases. He had acute pancreatitis about two years ago. The patient admitted that he smoked cigarettes and abused alcohol. Physical examination revealed, among other things, jaundice and moderate oedema of the distal sections of the legs.

Laboratory tests

The laboratory tests at admission revealed characteristic features of liver damage and failure. Significant hyperbilirubinemia (14.45 mg/dL), increased gamma-glutamyl transferase activity (416.0 U/L), and aspartate aminotransferase activity (209.7 U/L) were observed with a normal alanine aminotransferase activity value (38.1 U/L). Alkaline phosphatase activity was only slightly increased (183.0 U/L). Amylase and lipase activities in the blood were normal. There was also an increased concentration of C-reactive protein (CRP) in the blood (40.69 mg/L). The serum creatinine concentration was normal. The blood sodium concentration was slightly decreased (131 mmol/l) with a normal blood potassium concentration. The peripheral blood morphology parameters included: leukocytosis ($19.0 \times 10^3/\mu L$) with

a shift toward neutrophilia (80.6%), a reduced number of red blood cells (3.65 × 10^6/μL) with a normal haemoglobin concentration (13.7 g/dL) and normal haematocrit value (41.6%). The mean corpuscular volume (MCV) value was significantly increased (114.0 fL). The platelet count was normal (183 × 10^3/μL). Moreover, the laboratory tests performed on the following days revealed hypoalbuminemia (2.0 g/dL). On admission, the prothrombin time was increased to 21.6 seconds (INR 2.2), and on the following days of hospitalization the prothrombin time was further prolonged.

Diagnostic imaging and endoscopy

Due to the lack of a diagnosis that would explain the entire clinical picture presented by the patient, on the first day of hospitalization, computed tomography (CT) of the abdominal cavity and pelvis was performed with contrast enhancement, mainly to exclude the mechanical cause of jaundice. The CT scan described features of liver cirrhosis with the presence of free fluid in the peritoneal cavity, post-inflammatory changes in the pancreas, and cholelithiasis with thickening of the gallbladder wall. The patient was consulted surgically and indications for surgical intervention were excluded. A chest radiograph was performed, which revealed the presence of a small amount of fluid in the pleural cavities.
Gastroscopy revealed oesophageal varices without obvious signs of imminent bleeding, portal gastropathy, gastritis, and enlargement of the ampulla of Vater with the presence of two polyps in the duodenum.

Large hematoma of the rectus abdominis muscle

On the twelfth day of hospitalization, the patient reported abdominal pain in the mid-abdominal area radiating to the back. Physical examination revealed no abdominal pain on palpation and no symptoms of peritonitis. Painkillers and antispasmodics were administered with partial improvement. Due to the persistence of pain on the next day, it was decided to repeat the abdominal and pelvic CT with contrast enhancement. The performed examination revealed a hematoma measuring 35 × 75 × 125 mm in the rectus abdominis muscle on the left side. It should be noted that the patient did not suffer any injury during hospitalization. Moreover, he did not undergo any invasive procedures, including intra-abdominal skin injections or paracentesis. Figures 1 and 2 present CT scans showing the hematoma.

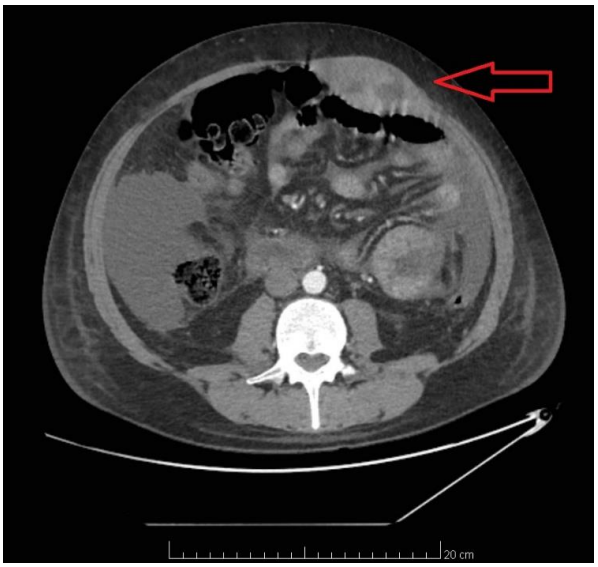

Fig. 1. Hematoma in rectus abdominis muscle on left side visible in CT image (cross-section).

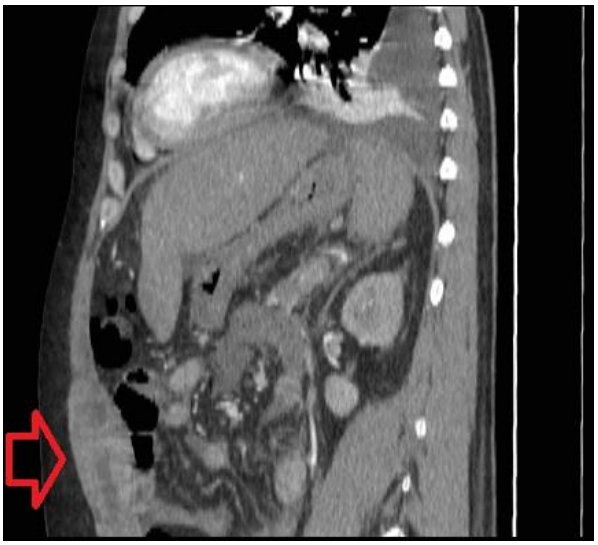

Fig. 2. Hematoma in rectus abdominis muscle on left side visible in CT image (longitudinal section).

On the day the hematoma was diagnosed, the prothrombin time was 30.6 seconds (INR 3.2). The peripheral blood count performed on the same day showed a haemoglobin concentration of 12.7 g/dL. Then, it was decided to transfuse fresh frozen plasma (a total of four units were transfused). Despite this, in the following days of hospitalization, the prothrombin time was further prolonged, to 31.3 seconds (INR 3.3) and 68.0 seconds (INR 7.7), respectively. In the following

days of hospitalization, the haemoglobin concentration dropped to 11.1 g/dL and 7.0 g/dL.

Further course of hospitalization

Despite the typical full treatment for decompensated liver cirrhosis, the patient's clinical condition deteriorated systematically. Consciousness disorders and peripheral oedema increased. Laboratory tests showed progressive signs of liver failure, renal failure, and anaemia. On the fifteenth day of hospitalization, the patient was pronounced dead.

DISCUSSION

Plasma coagulation disorders are a typical element of the clinical picture of liver failure in the course of liver cirrhosis, and the prolongation of prothrombin time is a sensitive marker of these disorders [6,7]. Nevertheless, it should be noted that the issue of coagulation disorders in patients with liver cirrhosis is difficult. On the one hand, patients are predisposed to hemorrhagic complications owing to the impaired synthesis of coagulation factors in the liver, hyperfibrinolysis, and often coexisting thrombocytopenia, but on the other hand, in patients with liver failure, not only is the synthesis of coagulation factors impaired, but also endogenous anticoagulant synthesis is limited. Additionally, increased intravascular coagulation occurs [8], which can result in thromboembolic complications, an example of which is portal vein thrombosis, often diagnosed in patients with liver cirrhosis [9]. Therefore, in clinical practice, blood coagulation disorders in patients with liver cirrhosis are corrected if symptoms of bleeding diathesis occur [10]. In the case of the patient described in this publication, no other symptoms of bleeding diathesis were observed before the hematoma was diagnosed, thus fresh frozen plasma was transfused only after the hematoma was diagnosed. It should be noted that despite the transfusion of four units of fresh frozen plasma, a further significant increase in prothrombin time was observed. Hence, it can be assumed that even if fresh frozen plasma had been transfused earlier, the course of events would not have changed. Moreover, the patient's general clinical condition deteriorated so rapidly as the disease progressed that death was inevitable.

The presented case report demonstrates that in the care of patients with end-stage liver disease, great vigilance should be exercised in recognizing symptoms suggesting bleeding because in this population of patients not only gastrointestinal bleeding, which is the most typical, may occur, but also other bleeding complications requiring more detailed diagnostics since their clinical picture may not be obvious, as in the presented case report. Stravitz et al. [11] presented the results of a retrospective study that analysed data from 1,770 people hospitalized for acute liver failure between 1998 and 2016. Bleeding symptoms occurred in 187 patients (11%), of which 173 episodes were spontaneous and 22 episodes were a complication of an invasive procedure. In the study group, bleeding from the upper gastrointestinal tract accounted for 84% of spontaneous bleeding cases, and 20 people experienced intracranial bleeding, with half of them occurring spontaneously and half after intracranial pressure monitor placement. According to the research conducted by Cho et al. [12], haemorrhagic complications occur in 12.2% of critically ill patients with liver cirrhosis hospitalized in the intensive care unit. The most common symptoms occur in the upper gastrointestinal tract, followed by bleeding in the respiratory system. Several cases of spontaneous muscle hematomas have been described to date in patients with liver cirrhosis, mainly in the iliopsoas muscle [13,14,15]. A spontaneous retroperitoneal hematoma was also reported in patients with liver cirrhosis awaiting for liver transplant [16]. Sugiyama et al. [17] published a review in 2009 in which they collected case reports available in the literature, showing that spontaneous intramuscular hematoma in patients with liver cirrhosis is a serious condition with a very unfavourable prognosis. In our case, the hematoma was diagnosed two days before the patient's death, which confirms the conclusions presented in the cited article. It is worth emphasizing that the strength of our case report is the fact that during hospitalization the patient underwent two CT scans, proving that the hematoma developed in the last days before death.

CONCLUSIONS

Haemorrhagic complications are an important issue in the care of patients with cirrhosis due to the frequent occurrence of plasma coagulation disorders and thrombocytopenia. Although the main haemorrhagic complication of liver cirrhosis is gastrointestinal bleeding, physicians should also consider other forms of clinical haemorrhagic complications in their routine clinical practice, including intramuscular bleeding. Clinicians should be vigilant, especially when anaemia is observed without an established cause.

Further research is necessary to better understand the pathophysiology and epidemiology of intramuscular bleeding in patients with liver cirrhosis.

Author's contribution

Study design – G.K. Jakubiak, M. Pietrzak, G. Cieślar, A. Stanek
Data collection – G.K. Jakubiak, M. Pietrzak
Manuscript preparation – G.K. Jakubiak
Literature research – G.K. Jakubiak
Final approval of the version to be published – G.K. Jakubiak, M. Pietrzak, G. Cieślar, A. Stanek

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