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Bacterial infections as an important factor of liver cirrhosis decompensation – a case report

Infekcje bakteryjne jako istotny czynnik dekompensacji marskości wątroby – opis przypadku

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Słowa klczowe
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Summary

Liver cirrhosis is associated with a broad spectrum of clinical manifestations. Except the typical symptoms including coagulation disorders, jaundice, and hypoalbuminemia, it is worth to mention the impact of the liver disease on the intestinal barrier. The impairment of the intestinal barrier in liver cirrhosis is an important factor that leads to increased risk of acquiring bacterial infection or hepatic encephalopathy development. In this article, there has been presented a case of 61-years old man with diagnosed liver cirrhosis due to chronic hepatitis C virus infection. This patient was hospitalized several times in 2015 due to various liver cirrhosis complications. The decompensations of his liver function were associated both with infections (urinary tract infections and bacteremia) and with hepatic encephalopathy. Selected biochemical test results, clinical assessment, and MELD scores were shown combined with information regarding infections that occurred.

Streszczenie

Marskość wątroby jest związana z szerokim spectrum manifestacji klinicznych. Poza typowymi objawami, do których należą zaburzenia układu krzepnięcia, żółtaczka i hipoalbuminemia, warto wspomnieć o wpływie choroby wątroby na przepuszczalność bariery jelitowej. Nieprawidłowa funkcja bariery jelitowej u chorych z marskością wątroby jest istotnym czynnikiem, który wpływa na zwiększone ryzyko wystąpienia infekcji bakteryjnej lub rozwoju encefalopatii wątrobowej. W poniższym artykule zaprezentowano przypadek 61-letniego mężczyzny ze zdiagnozowaną marskością wątroby na tle przewlekłego wirusowego zapalenia wątroby typu C. Pacjent ten był kilkukrotnie hospitalizowany z powodu różnych powikłań marskości wątroby. Dekompensacje funkcji wątroby były związane zarówno z zakażeniami (zakażenie układu moczowego oraz bakteriemia), jak i z encefalopatią wątrobową. Zaprezentowano wybrane wyniki badań biochemicznych, ocenę kliniczną oraz wyniki MELD w zestawieniu z informacjami o współistnieniu zakażeń.

Conflict of interest
Konflikt interesów
None
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INTRODUCTION

Liver cirrhosis is the end stage of liver damage that can be caused by various factors including but not limited to viral infections, toxic damage and metabolic diseases. The impairment of liver function does not result only in protein synthesis or bilirubin conjugation, but also is related to higher bacterial infection rate (1). This phenomenon can be explained by intestinal bacterial overgrowth and increased bacterial translocation (2, 3). The most common infections are urinary tract infection and spontaneous bacterial peritonitis, followed by pneumonia, soft tissue, skin infections and bacteraemia (3). Another complication related to liver cirrhosis is hepatic encephalopathy. Its pathology is multifactorial; however among the others it includes the metabolism of ammonia. The production of ammonia it higher, if bacterial overgrowth is present (4). The hyperammonemia influences the induction of encephalopathy

through promotion of cerebral oedema, modulation of the blood-brain barrier and neuroinhibition (4-6). Importantly bacterial infections seem to be underestimated cause of liver function impairment and hepatic encephalopathy. We describe a case report illustrating an association between liver function, complications of liver cirrhosis and multiple bacterial infections.

CASE PRESENTATION

A 61-years old man, Caucasian, was admitted to the Department of Infectious Diseases and Hepatology on 16 Feb 2015 due to liver failure (grade 2 encephalopathy, ascites, jaundice) combined with kidney function impairment, and dyselectrolytemia. The liver cirrhosis was caused by chronic hepatitis C virus infection (HCV genotype 1b). Patient previously had undergone unsuccessful antiviral therapy twice in the past (with PEGylated interferon and ribavirin). During the first hours of the hospitalization he presented severe encephalopathy with coma. Calculated MELD at admission was 26, MELD-Na – 40. In the urine sample examination leucocytes (20-30 in the visual field) and numerous bacteria were found and urinary tract infection (UTI) was diagnosed. The empiric treatment with ceftriaxone and ciprofloxacin was administrated, the culture from next urine sample was negative and there was no possibility to isolate species of bacteria that caused the urinary tract infection. The additional treatment (rifaximin, IV albumin, ornithine, diuretics) was successful and allow patient to recover from coma. In two weeks treatment patient liver function had improved, bilirubin concentration decreased from 99.8 μmol/l to 77.1 μmol/l, creatinine concentration also decreased from 270.1 μmol/l to 126.7 μmol/l (GFR 21.0 ml/min/1.73 m^2 and 53.7, respectively), in the urine sample leucocytes were depleted. Calculated MELD decreased to 20, MELD-Na to 30. The condition of patient worsens again after two weeks of hospitalization. Although liver function remained stable, kidney function worsened (creatinine concentration increased to 332.3 μmol/l, GFR 16.5 ml/min/1.73 m^2), subsequently MELD and MELD-Na rose again to 30 and 40, respectively. Leucocytes were found again in urine sample. Patient recovered from UTI (decrease of leucocyturia, decrease of CRP concentration) after treatment. Again, after resolving UTI, function of the liver measured by MELD increased (MELD 24, MELD-Na 24). Patient was transferred to the Department of Nephrology, Transplantology and Internal Medicine of Medical University of Silesia for qualification for orthotropic liver transplantation (OLTx) on 18 Mar 2015.

Patient had begun qualification for OLTx, and underwent coronary angiography on 28 Apr 2015. Two days later he was admitted to the Department of Infectious Diseases due to liver function decompensation (worsening of jaundice and ascites followed by encephalopathy that revealed after admission – stage 2). Due to fever > 38°C infection was suspected. Blood, urine, and ascitic fluid samples were collected for culture, biochemical and morphology examination. Leukopenia and fever over 38°C allowed to diagnose systemic inflammatory response syndrome (SIRS). *Klebsiella pneumoniae* ESBL was found in urine culture and *Enterobacter cloacae* ESBL in blood culture. The presence of SIRS and the infectious agent combined with bilirubin concentration of 84.8 μmol/l (more than 3 times upper limit level) allowed to diagnose severe sepsis. Patient was treated initially with empiric antibiotic therapy (ceftriaxone), after receiving microbiological tests results – targeted antibiotic therapy with imipenem was applied. The treatment was successful, the observed biochemical results improved (creatinine concentration 215.1 μmol/l to 127.9 μmol/l, GFR 27.3 to 49.8, bilirubin concentration 84.8 μmol/l to 55.3 μmol/l, MELD 23 to 18, MELD-Na 30 to 22). Patient recovered form sepsis and was discharged on 15 May 2015.

The next two hospitalizations that took place on August 2015 and on October 2015 were not related to infections, but to hepatic encephalopathy. There were no sign of infection at admission, and no nosocomial infection during hospitalizations. Calculated MELD during first of these hospitalizations was 18 and 15 (at admission and discharge, respectively). During second hospitalization MELD was 17 and 14 (at admission and discharge, respectively). Figure 1 shows the changes of MELD during all hospitalizations. Additionally the dates with beginning of infection were highlighted. Patient eventually underwent orthotropic liver transplantation in the end of 2015. However the procedure was complicated by stroke.

DISCUSSION

Increased MELD score at admission could be related to the greater risk of infection. As the infection occurs, it worsens the liver function, and the increase of MELD score is observed. The presented patient is an

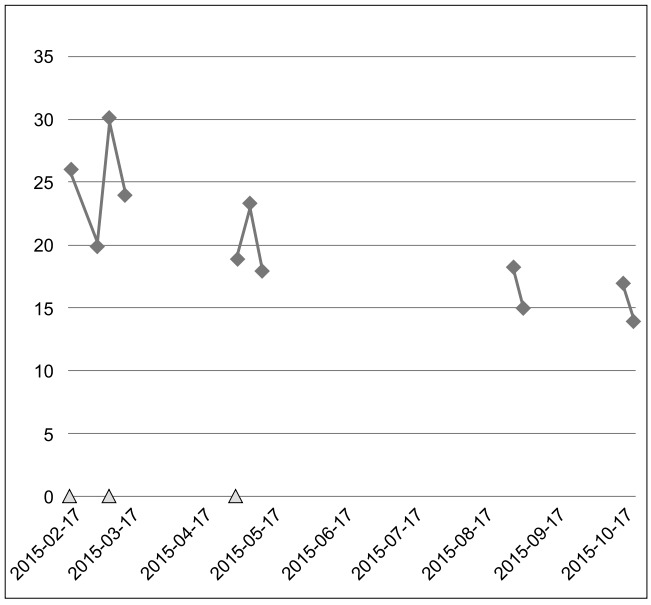

Fig. 1. MELD score in time with infection occurrence
Grey triangle points the date of infection occurence

example that higher MELD at admission was related to infection. When the infection resolved liver function improved, and MELD score decreased. Due to vicious cycle between liver injury and failing intestinal barrier, it may be hard to establish, which factor was the first: was it bacterial infection that worsened liver function and increased MELD score or was the liver failure the reason for intestine barrier failure and subsequently infection occurrence due to bacterial translocation from intestine? Obviously, it was a single patient and pointing out the significance of MELD score as a predictor of infection in liver cirrhosis will require further studies.

CONCLUSIONS

The MELD score of the patient varied during hospitalizations in our department. Each time the infection occurred MELD score before the diagnosis of infection was higher or equal to 19. The occurrence of infection worsened the clinical state of the patient, and while liver function had worsened, the increase of MELD was noted. The last two hospitalizations were due to hepatic encephalopathy. Calculated MELD at admission was lower than in case of admissions of the patient with infection, and equalled 18 and 17, respectively.

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