Advanced Alcoholic Cirrhosis: A Case Report

JENNIFER LEFEBVRE, MICHELLE BUTINA

A 43-year-old male presented to a local hospital exhibiting several complications of alcoholic cirrhosis including acute renal failure, several bacterial infections, and edema. Patient complications and other complications resulting from cirrhosis are discussed and related to clinical laboratory findings. The patient was placed on antibiotics and fully recovered from his bacterial infections.

OBJECTIVES: Discuss several complications that can arise from having alcoholic cirrhosis of the liver; describe the distinctive pattern on serum or urine protein electrophoresis indicating cirrhosis.

ABBREVIATIONS: ALT = alanine aminotransferase; AST = aspartate aminotransferase; BUN = blood urea nitrogen; HRS = hepatorenal syndrome; NP = not performed; *S. aureus* = *Staphylococcus aureus*; US = United States; WBC = white blood cell.

INDEX TERMS: alcoholic cirrhosis; alcoholism, cirrhosis.

Clin Lab Sci 2007;20(2):80

***Jennifer LeFebvre MT (ASCP)** is **medical technologist,** Memorial Health University Medical Center, Savannah GA.*

***Michelle Butina MS CLS (NCA) MT (ASCP)** is **assistant professor,** Medical Technology Program, Armstrong Atlantic State Universtity, Savannah GA.*

***Address for correspondence:** Michelle Butina, assistant professor, Medical Technology Program, Armstrong Atlantic State University, 11935 Abercorn Street, Savannah GA 31419. (912) 921-5513. butinami@mail.armstrong.edu*

The peer-reviewed Clinical Practice Section seeks to publish case studies, reports, and articles that are immediately useful, are of a practical nature, or contain information that could lead to improvement in the quality of the clinical laboratory's contribution to patient care, including brief reviews of books, computer programs, audiovisual materials, or other materials of interest to readers. Direct all inquiries to Bernadette Rodak MS CLS(NCA), Clin Lab Sci Clinical Practice Editor, Clinical Laboratory Science Program, Indiana University, Clarian Pathology Laboratory, 350 West 11th Street, 6002F, Indianapolis IN 46202. brodak@iupui.edu

The paper has not been presented at a meeting nor has it been accepted for presentation at a future meeting.

CASE PRESENTATION

A 43-year-old male was admitted into a local hospital with left knee pain and swelling, fever, confusion, three days of urinary incontinence, and abdominal pain. The patient had a history of alcoholic cirrhosis of the liver, acute renal failure, and smoking. Due to his known history of alcoholic cirrhosis and acute renal failure; a hepatic function panel, basic metabolic panel and urinalysis were ordered upon admission. Table 1 includes significant test results.

Because of his swollen and feverish knee, synovial fluid was aspirated for cell count. The cell count revealed many neutrophils, bacteria, and gross pus. Blood cultures were also ordered, eventually growing *Staphylococcus aureus*. Upon culture of the synovial fluid, *S. aureus* was also isolated. Throughout the hospitalization, other samples were obtained and cultured. Urine cultures grew *Klebsiella pneumoniae* and *S. aureus* on two different occasions. Gram-positive cocci organisms were seen microscopically while examining a bile specimen. The patient was treated with Zosyn® and Nafcillin.[1]

OVERVIEW

The liver is one of the most important organs in the body. It plays a major role in protein, lipid, and carbohydrate homeostasis, enzyme and bile synthesis, detoxification and infection control. The human liver weighs approximately 1.4 kg and regulates many metabolic functions.[2] In this particular case, the long history of alcohol abuse resulted in cirrhosis of the liver. Other factors such as hepatitis virus, autoimmune disorders, hemochromatosis, fat deposition, drugs and toxins, and obstructed bile ducts are all known to cause cirrhosis of the liver. Cirrhosis refers to the inflammation and scarring of liver tissue. As the liver tissue becomes progressively damaged, blood is not properly filtered of toxins or byproducts, and proteins are not adequately synthesized.

PATHOGENESIS

Normal liver tissue becomes inflamed in the early stages of cirrhosis. As it progresses, the tissue becomes scarred, nodular, and fibrotic. A nodule is a small mass of tissue or an aggrega-

tion of cells, characterized by their size. The nodules present on the liver can either be described as macronodules (greater than three mm in width), micronodules (diameters between 1.5 m and three m), or both.[2] The scarred and lobular tissue is the result of damaged hepatocytes being replaced by fibrous tissue. Abnormal hepatocytes cannot function properly resulting in interruption of circulation and regulation of critical body functions.[3]

Alcoholic cirrhosis is the leading type of cirrhosis in the United States and is one of the ten leading causes of death in the US.[2] It has been found that women are more affected by alcohol than men. Two to three daily drinks for women and three to four for men over many years have been linked to alcoholic cirrhosis.[4] Some researchers believe that men are more tolerant because they can detoxify alcohol more readily.[5]

CLINICAL CHARACTERISTICS AND COMPLICATIONS

A patient's health, medical history, lifestyle, and compliance with previous physician recommendations all contribute to the symptoms and complications. General symptoms of cirrhosis include edema, ascites (fluid accumulation in the abdominal cavity), jaundice, fatigue, itching, confusion, abdominal pain, bleeding, bruising and weight loss.[6] If these symptoms are overlooked, liver disease can go undetected and further complications can arise. The patient in this case study has sustained several major complications attributed to chronic alcoholic cirrhosis. They were acute renal failure, bacterial infections, and edema.

Acute renal failure is common in patients with cirrhosis. In the majority of patients the renal failure is due to prerenal azotemia (reduced renal blood flow) and tubular necrosis.[7] The main causes of prerenal failure in cirrhosis patients include intravascular volume depletion (hemorrhage, gastrointestinal losses, shock), severe sepsis, and hepatorenal syndrome (HRS).[7] The decreased renal blood flow leads to decreased glomerular filtration which results in insufficient filtering of blood, hormone secretion, excretion of toxins and waste products, and acid-base imbalances. The patient's renal failure was demonstrated by his incontinence upon admission, as well as his elevated blood urea nitrogen (BUN) and serum creatinine levels (Table 1). Upon admission and throughout hospitalization, his serum BUN and creatinine levels remained high. Another factor confirming kidney damage was the presence of protein in his urine. In healthy kidneys, the glomerulus does not allow large molecules, such as protein to be filtered. However, this patient had extremely high percentages of protein in his urine, as indicated in the results of his urine protein electrophoresis (Table 2 and Figure 1).

Many cirrhotic patients suffer from bacterial infections that can ultimately lead to death. There are many factors contributing to the increased number of bacterial infections including deficient complement production, prolonged hospitalization, and complications of cirrhosis leading to a weakened immune system.[8] The most common infections include those of the urinary tract, respiratory tract, peritoneum of the abdomen (peritonitis), and blood (bacteremia).[8] As stated

Table 1. Patient laboratory results throughout hospital stay

Test name	Admission Day 1	Day 10	Discharge Day 17	Normal range
BUN	49.26	18.20	21.77	2.9-7.1 mmol/L
Creatinine	415.48	362.44	335.92	61.9-114.9 umol/L
WBC	38.2	19.0	9.7	4.8-10.8 K/CUMM
Bilirubin	106.02	343.71	218.88	1.7-5.1 umol/L
AST	244	51	3116	12-38 U/L
ALT	52	16	985	10-45 U/L
Albumin	19	12	19	35-48 g/L
Protein (serum)	70	80	55	60-80 g/L
Protein (urine)	30	88	NP	Negative-Trace
Urinalysis (bacteria)	Many	NP	NP	None

earlier, the patient suffered from several bacterial infections including a urinary tract infection and bacteremia.

Another complication of alcoholic cirrhosis is edema, an accumulation of excess fluid. The damaged liver fails to produce adequate levels of albumin, which is necessary to maintain the colloid osmotic pressure of intravascular fluid.[2] Hypoalbuminemia leads to fluid leakage from the capillary beds allowing the fluid to collect in the surrounding tissues. Fluid accumulation in the abdomen (ascites) and legs is common in patients with cirrhosis.[6] Table 1 displays the consistently decreased serum albumin levels of this patient resulting in his edema.

Blood clotting abnormalities, mental disturbances, portal hypertension, and medication sensitivity are other complications often seen with alcoholic cirrhosis. Clotting factors, proteins that are essential for hemostasis, are not being synthesized by the liver resulting in prolonged bleeding. Toxemia, toxins in the bloodstream, due to renal and hepatic damage can inhibit certain coagulation factors, thus exacerbating bleeding problems. Another factor that could affect bleeding is the theory that alcohol has a direct affect on bone marrow megakaryocytes, leading to platelet functional abnormalities and thrombocytopenia. Neurological complications are due to the build up of toxins in the blood that eventually become deposited in the brain. A cirrhotic liver slows the flow of blood through the portal vein (vein that carries blood from the intestine and spleen to the liver) causing the pressure to increase, known as portal hypertension. Lastly, medication sensitivity is due to the diminished ability of the liver to filter medications from the blood.

RELEVANT LABORATORY RESULTS

After reviewing the patient's medical history and evaluating his current medical situation, many laboratory tests were ordered to evaluate his multiple conditions. His serum BUN and creatinine values remained elevated, indicating acute renal failure. Significant liver damage contributed to increased aspartate aminotransferase (AST) and alanine aminotransferase (ALT), as shown in Table 1. Both have high concentrations in the liver and if elevated can indicate liver damage.

Serum and urine protein electrophoresis can identify the amount of specific protein levels present. These tests were ordered due to the decreased serum albumin levels and normal total protein levels, indicating an elevation in other serum proteins. Table 2 and Figure 2 illustrate the electrophoretic pattern of serum hypoalbuminemia, hypergammaglobulinemia, and a beta-gamma bridge. The beta-gamma bridging was caused by fast moving gamma globulins, preventing distinct bands from appearing between the gamma and beta globulin fractions, and therefore forming a bridge between the two.[2] The electrophoresis pattern and the serum hypoalbuminemia demonstrate a classic finding for active alcoholic cirrhosis patients.

TREATMENT AND PROGNOSIS

Currently, there is no cure for alcoholic cirrhosis. Complications, symptoms, and further progression can be treated or prevented with proper care and monitoring. Abstaining from alcohol is critical to stop the progression of the disease, while exercise may delay it.

Table 2. Patient electrophoresis results

	Serum	Normal range	Urine	Normal range
Albumin	25.2	55%-63%	22.2	0%
Alpha-1	5.0	1.9%-4.7%	5.5	0%
Alpha-2	6.1	8.5%-15%	6.0	0%
Beta	11.8	9%-17.6%	19.9	0%
Gamma	51.9	9%-22.3%	46.5	0%

Figure 1. Urine protein electrophoresis pattern

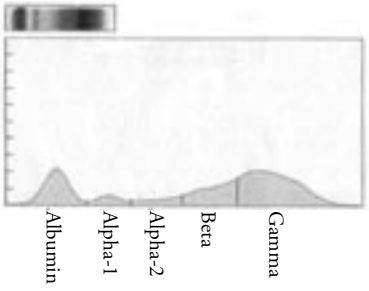
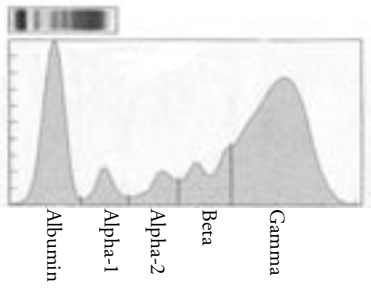


Figure 2. Serum protein electrophoresis pattern



Treatment and prevention of ascites and edema may include dietary changes, most notably sodium intake or prescribing diuretics that aid in fluid balance. Portal-hypertension can be controlled by blood pressure medications such as beta-blockers, angiotensin-converting enzyme inhibitors, or calcium channel blockers.[6]

CASE CONCLUSION

This patient's case demonstrated a typical but serious case of alcoholic cirrhosis, with many major complications. Laboratory results displayed abnormal kidney and liver functions, as well as infections of numerous body sites. The patient was treated for the bacterial infections and his liver and metabolic panels were continually monitored during his hospitalization.

If a patient's condition progresses to the stage that a liver transplant is necessary, it is encouraging to know that 80 percent to 90 percent of patients survive the surgery.[9] Immunosupressant drugs such as cyclosporine and tacrolimus greatly improve the survival rate by suppressing the immune system and preventing it from attacking the newly transplanted liver.[4] These same drugs have also made finding a compatible liver easier, because an exact human leukocyte antigen match is not always necessary. Early diagnosis, monitoring, and abiding by the physician's recommendations can all increase a patient's chance of surviving with alcoholic cirrhosis of the liver.

It is important to remember that treating patients not only involves treating the current clinical problems, but also attacking the primary causal problem. In this case, alcohol was the underlying source of all of the patient's complications. Refraining from drinking in the future may prolong his life and eliminate further complications.

REFERENCES

1. Dalovisio J, Tice A, Wynn M, and others. Evaluation of the efficacy and safety of outpatient parenteral antimicrobial therapy for infections with methicillin-sensitive staphylococcus aureus. South Med J 2005;98(6):590-5.
2. Bishop M, Fody E, Schoeff L. Clinical chemistry: principles, procedures, correlations. 5th ed. Baltimore: Lippincott Williams & Wilkins; 2005.
3. Worman HJ. Cirrhosis. New York; Columbia University Department of Medicine:2000. Available from http://cpmcnet.columbia.edu/dept/gi/disliv.html. Accessed 2006 Feb 19.
4. National Digestive Diseases Information Clearinghouse. Cirrhosis of the liver. Bethesda; National Institutes of Health. 2003. Available from http://digestive.niddk.nih.gov/ddiseases/pubs/cirrhosis/. Accessed 2005 Aug 24.
5. American Liver Foundation. Cirrhosis: many causes. New York;2003-2004. Available from www.liverfoundation.org/db/articles/1059. Accessed 2005 Aug 23.
6. Magee K. What is cirrhosis? Boise; Healthwise:2004. Available from http://health.yahoo.com/ency/healthwise/aa67653/_aa67656. Accessed 2005 Aug 26.
7. Moreau R, Lebrec D. Acute renal failure in patients with cirrhosis: perspectives in the age of MELD. Perspect Clin Hepatol 2003;37:233-43.
8. Brann O. Infectious complications of cirrhosis. Curr Gastroenterol Rep 2001;3(4):285-92.
9. Arn R, Koorey D, McCaughan G, and others. An active liver transplant programme for hepatocellular carcinoma in cirrhotic patients: is it justified? Clin Transplant 1999;13(6):531-5.