

'Edema Everywhere'

Jennifer L. Shin, B.Sc. (OT2) and Eni J. Keszthelyi, M.Sc. (OT2)

Case Presentation

A 58-year-old man with a history of Type 2 diabetes mellitus was admitted for the evaluation of gross, generalized edema (anasarca).

Seven months prior: The patient presented to his family physician with a petechial rash over the lower extremities. A skin biopsy was performed which demonstrated non-specific inflammation. The rash resolved upon treatment with a topical steroid ointment.

Four months prior: The patient developed leg ulcers, which were treated with prednisone 75 mg po bid for presumed lower extremity vasculitis. His steroid therapy was complicated by the development of generalized edema, including abdominal distention. His weight increased by 23 kg. Furosemide (40 mg po daily) was prescribed for the management of his edema and resulted in significant improvement.

Three months prior: The patient was admitted to hospital for the management of increasing peripheral edema, skin breakdown involving the lower extremities, and ascites. A number of laboratory and radiologic investigations were performed (Table 1). Prednisone was discontinued. Topical silver sulfadiazine ointment and occlusive dressings were applied to the lower extremity ulcers. With aggressive diuresis over a 7-day hospital stay, the patient's weight decreased from 92.1 kg to 78.1 kg. Discharge medications included Novolin 30/70 24 units sc bid, spironolactone 100 mg po daily, furosemide 80 mg po daily, atenolol 50 mg po daily, quinapril 20 mg po daily, and metformin 500 mg po tid. Home nursing care was arranged to help with the management of his ulcers.

Table 1
Laboratory Investigations Performed
Three Months Prior to Current Admission

Variable	Value	Range
Sodium	133	135 - 145 mmol/L
Potassium	3.6	3.5 - 5.0 mmol/L
Chloride	91	95 - 108 mmol/L
Creatinine	144	40 - 130 µmol/L
Glucose	5.1	3.9 - 6.9 mmol/L
BUN	15.6	3.0 - 7.0 mmol/L
Hemoglobin	134	115 - 165 g/L
HbA1c	0.096	<0.06 of Total Hb
WBC count	8.2	3.5 - 12.0 x 10 ⁹ /L
Platelet count	26	150 - 450x10 ¹² /L
PT	13.2	9 - 13 s
PTT	26.4	26 - 36 s
AST	60	< 40 U/L
LDH	886	50 - 240 U/L
ALP	161	35 - 110 U/L
Total Bilirubin	18	<18 mol/L
Albumin	26	35 - 50 g/L
Urine glucose	positive	negative
Urine protein	negative	negative
Abdominal Ultrasound	5-6 L ascitic fluid, liver poorly visualized, multiple gallstones, slight splenomegaly; kidneys 11.7cm (R) and 11.6 cm (L).	
2D-Echocardiogram and ECG	Grade III/IV LV function with hypokinesia in apex and septum; normal sinus rhythm and rate; Q waves in V ₁ through V ₃ ; no ST segment or T wave abnormalities.	
CXR	Atelectasis in lung bases	

Two months prior: The patient was re-admitted to hospital on three separate occasions for the management of his recurrent ascites, edema, and diabetes. There was worsening of his renal function (creatinine = 233 mmol/L). The patient also complained of abdominal discomfort, generalized weakness, and fatigue. As part of the edema workup, he had an outpatient nephrology assessment. Urinalysis was normal: there was no proteinuria, no hematuria, and no urinary sediment. It was felt that renal disease was an unlikely cause for his anasarca.

He was subsequently admitted to a university-affiliated General Internal Medicine Clinical Teaching Unit.

Current admission: Inquiry into past medical history revealed significant alcohol consumption, 40 oz/week (~1180 g/week) of rum for 20 years, but he had been abstinent for the past 1.5 years. In addition, longstanding, poorly controlled Type 2 diabetes mellitus (DM) and associated complications, including renal insufficiency (creatinine range 140-200 mmol/L) and non-proliferative diabetic retinopathy, were revealed. Past medical history also included prior myocardial infarction, hypertension, and hyperlipidemia. There was no prior history of congestive heart failure or dyspnea.

Medications on admission included: Novolin 30/70 26 units sc bid, furosemide 80 mg po daily, spironolactone 100 mg po daily, nitroglycerine 0.3-0.6 mg sl prn, and oxazepam 30-60 mg hs prn.

At the time of admission, the patient was alert, oriented, and in no apparent distress. Vital signs were: heart rate 114 beats/minute, blood pressure 120/70 mmHg, and respiratory rate 18 breaths/min. Oxygen saturation was normal. JVP was 3 cm above the sternal angle. The patient was not in any respiratory distress. Chest examination revealed fine inspiratory bilateral crackles at the lung bases. Precordial examination revealed normal first and second heart sounds, with no extra sounds or murmurs. There was bilateral pitting edema to the ankles.

Abdominal examination revealed bulging flanks, with dullness to percussion at the flanks, 5 cm of shifting dullness, and a positive fluid wave. There was no palpable splenomegaly and Castell's sign was negative. There was no jaundice or scleral icterus. However, there were spider nevi present on the trunk, as well as loss of axillary hair bilaterally. No other peripheral stigmata of chronic liver disease were apparent.

Lower extremity leg ulcers were noted bilaterally. On the right side, there were multiple weeping ulcers with surrounding cellulitis with areas of tissue necrosis. Multiple ulcers were also observed on the left side. A number of laboratory investigations were performed (Table 2).

Table 2
Results of Laboratory Investigations
at Current Admission

Variable	Value	Range
Sodium	133	135 – 145 mmol/L
Potassium	4.7	3.5 – 5.0 mmol/L
BUN	16.7	3.0 – 7.0 mmol/L
Chloride	97	95 – 108 mmol/L
Creatinine	189	40 – 130 mmol/L
Glucose (fasting)	10.7	< 6.1 mmol/L
Urate	817	Males: 238 – 506 µmol/L
Total Cholesterol	3.23	high ≤ 6.21 mmol/L
LDL	1.94	high ≤ 4.14 mmol/L
Triglycerides	1.17	< 2.26 mmol/L
Total Iron	9	4 – 30 µmol/L
Transferrin	1.78	1.88 – 3.41 g/L
Iron saturation	0.21	0.12 – 0.57 %
TSH	5.39	0.50 – 4.7 mIU/L
HbA1c	.056	< 0.06
AST	34	< 40 U/L
ALT	38	< 40 U/L
LDH	718	50 – 240 U/L
ALP	417	35 – 110 U/L
Total Bilirubin	27	< 18 mol/L
Albumin	31	35 – 50 g/L
Hemoglobin	117	115 – 165 g/L
WBC count	9.3	3.5 – 12.0 x 10 ⁹ /L
Platelets	79	150 – 450 x 10 ¹² /L
PT	17	9 – 13 s
PTT	42	26 – 36 s
INR	1.35	0.9 – 1.1
24 hour Urine Function (protein)	0.076	negligible
Abdominal Ultrasound	Coarse, nodular liver; small mobile calculi in gallbladder; kidneys: 9.8 cm (R) 10.1 cm (L); common bile duct not well seen; normal intrahepatic ducts; Doppler revealed normal flow and direction in hepatic veins	
2D-Echocardiogram and EKG	Grade II/IV LV function with hypokinesia in apex and septum; normal sinus rhythm and rate; Q waves in V ₁ through V ₃ ; reduced voltages	
Paracentesis	2.2 L cloudy fluid; WBC, 400x10 ⁶ /L; 96% lymphocytes, 4% neutrophils; RBC, 10, 000 x 10 ⁶ /L	
Cryocrit	Negative	
Immunoglobulin survey	Trivial spikes within polyclonal increases of IgG 20.3 mmol/L, IgA 9.34 mmol/L, IgM 2.7 mmol/L	
Blood Smear	Hypersegmentation of neutrophils (PMN)	
Viral Serology	Hepatitis B and C Negative	
Liver Biopsy	Moderate to severe alcoholic cirrhosis	

The day following admission, paracentesis was performed and 6.5 L of cloudy ascitic fluid was removed. Laboratory analysis of the ascitic fluid was performed and revealed the following: WBC, $437 \times 10^6/L$ (5% PMN, 50% lymphocytes, 40% macrophages); albumin, 3 g/L; amylase, 12 U/L; LDH, 64 U/L; and protein, 17 g/L. Culture of the ascitic fluid was negative for bacteria. The serum/ascites albumin gradient was 28 g/L. In addition, an ultrasound-guided percutaneous liver biopsy was performed.

Compression therapy for the ulcers was initiated with 1:20 sodium hypochlorite compresses. The ulcers were debrided. A culture of the ulcers was negative. The patient's weight decreased from 89 kg to 73 kg over his 8 days of hospitalization.

In summary, the patient is a 58-year-old male with a background history of chronic alcohol abuse and longstanding, poorly controlled Type 2 DM. He initially received steroid therapy for the management of chronic lower extremity ulcers. This treatment was complicated by the development of generalized edema, including ascites. The patient now presents with persistent lower extremity ulcerations, and progressively worsening ascites. Clinical examination revealed the presence of ascites and stigmata of chronic liver disease, including spider nevi and the loss of axillary hair. He also complained of significant generalized weakness and fatigue. Diagnostic and therapeutic paracentesis revealed transudative ascites with a serum/ascites albumin gradient of 28 g/L. Cell count and culture of the ascites fluid did not reveal evidence of spontaneous bacterial peritonitis.

What is the differential diagnosis to explain these clinical findings?

Differential Diagnosis

Generalized Edema. Edema indicates expansion of the extracellular fluid compartment. This is caused by an imbalance between capillary and interstitial fluid hydrostatic and oncotic pressures. Gross, generalized edema is termed anasarca (Table 3). It can be characterized by periorbital puffiness, pitting edema of the extremities, trunk and face puffiness, pleural effusions, and ascites.¹⁹ Edema can be categorized by its nature, extent, and location. Serial assessment of the amount of accumulated fluid can be done by daily measurements of the patient's abdominal girth or weight. One kg of weight gain equals 1 L of fluid accumulation.³

Table 3
Causes of Generalized Edema

- Congestive heart failure
- Nephrotic syndrome and other causes of kidney disease
- Nutritional disorders
- Cirrhotic liver disease

Edema due to congestive heart failure: Edema due to congestive heart failure can be caused by defective systolic

emptying of the heart or impaired left ventricular relaxation. A reduction in the effective circulating volume triggers activation of the sympathetic nervous system and the renin-angiotensin-aldosterone (RAA) system leading to the retention of salt and water. The patient has a past history of an anterior myocardial infarction; however, he has not experienced dyspnea or paroxysmal nocturnal dyspnea. He does have orthopnea, but it was felt to be related to ascites and diaphragmatic embarrassment. JVP was normal, and the echocardiogram revealed only moderately impaired left ventricle (LV) function. These findings make cardiac causes of his edema less likely.

Edema due to nephrotic syndrome: This syndrome is characterized by proteinuria leading to a lower colloid oncotic pressure and net fluid movement into the interstitium. The decrease in effective circulating volume activates the RAA system, which promotes further salt and water retention. The patient did not have proteinuria within the nephrotic syndrome range (defined as urine protein $> 3.5g/day$), nor was there evidence of severe hypoalbuminemia (defined as serum albumin $< 20 g/L$). However, he did have mild hypoalbuminemia (31 g/L; normal albumin range is 35-50 g/L).

Nutritional edema: Nutritional edema is caused by a diet that is severely deficient in protein intake or following protein loss in the gastrointestinal tract. This promotes development of edema secondary to hypoalbuminemia. Examples include Beriberi heart disease or multiple peripheral arteriovenous fistulas. Starvation can promote edema through hypoalbuminemia, hypokalemia, and other caloric deficits.¹ The patient did not have any severe nutritional disorders.

Edema due to liver disease: Edema due to liver disease is multifactorial in nature. The patient had stigmata of chronic liver disease and a significant past history of alcohol consumption, suggesting liver disease as a possible cause for his ascites.

The serum ascites: albumin gradient of 28g/L indicates the presence of portal hypertension (serum ascites:albumin gradient = serum albumin - serum ascites; gradient $> 11g/L$ shows portal hypertension). Two hypotheses for the development of ascites in cirrhosis include the underfill theory and the overfill theory. In the underfill theory, portal hypertension and hypoalbuminemia lead to transudation of sodium and water into the peritoneum, causing a decrease in effective circulating volume. This results in subsequent salt and water retention by the kidneys. In the overfill theory, liver disease primarily leads to the renal retention of sodium and water which secondarily "overflows" into the peritoneal cavity. A combined theory postulates that liver disease results in peripheral vasodilatation, causing a reduction in effective circulating volume, central vasoconstriction of the afferent arteriole of the kidney, and secondary angiotensin II-mediated reabsorption of sodium in the proximal convoluted tubules.²

Edema due to metabolic causes. The patient's TSH was normal, ruling out myxedema (as seen in hypothyroidism) as a cause of his edema.

Cirrhosis. Cirrhosis is an irreversible process of chronic injury to the hepatic parenchyma. It is the end result of hepatocellular regeneration and fibrous scarring that constitute the major responses of the liver to a variety of longstanding or repetitive insults.^{4, 5, 6} After cardiovascular disease and cancer, cirrhosis of the liver is the third leading cause of death in patients aged 45- to 65-years in the Western world.⁷ Cirrhosis may be classified by its etiologic causes or pathologic features into a variety of subtypes (Table 4).^{2, 4}

Table 4
Subtypes of Cirrhosis

1. Alcoholic
2. Biliary
3. Cardiac
4. Metabolic (Hemochromatosis)
5. Inherited (Wilson's Disease)
6. Drug- related (α -methyl-Dopa)
7. Viral (Hepatitis B and C virus)
8. Cryptogenic (no identified cause)
9. Other

Cirrhosis results in three major sequelae: portal hypertension and its complications, hepatocellular failure, and/or the development of hepatocellular carcinoma. The cirrhotic patient may present with mild, compensated disease, or with severe end-stage complications. The clinical and laboratory features of cirrhosis are extensive (Table 5).

Table 5
Clinical and Laboratory Features of Cirrhosis

- * Ascites
- * Jaundice
- * Splenomegaly
- * Spider angiomas, palmar erythema,
Fetor hepaticus, Dupuytren's contracture, caput medusae
Generalized muscle wasting
Bruising
Hepatic encephalopathy
- * Loss of axillary and pubic hair
Males: testicular atrophy, gynecomastia

* asterisk indicates features of cirrhosis identified in this patient

Bleeding from esophageal varices is a severe complication of cirrhosis. Esophageal varices are the result of porto-systemic shunting due to portal hypertension in the cirrhotic liver. The presence of coagulopathy may also contribute to bleeding.

Complications of ascites. Complications of ascites include the hepatorenal syndrome and spontaneous bacterial peritonitis (SBP). Hepatorenal syndrome is defined as functional renal failure. The kidneys are histologically normal but there is a decline in the glomerular filtration rate with an increase in serum creatinine, marked reduction in urinary sodium excretion, and the development of oliguria. SBP refers to infection of the ascites fluid. Presentation of SBP may include fever and abdominal pain; however, some patients may experience no signs and symptoms other than a change in mentation. Laboratory evaluation of the ascites fluid did not reveal the presence of SBP in this patient.

Alcoholic liver disease. Three main forms of evolving liver injury due to alcohol abuse can be described: fatty infiltration, alcoholic hepatitis, and alcoholic cirrhosis. In the majority of patients with chronic alcoholism, the fatty liver is benign and damage is potentially 100% reversible following a period of abstinence. However, alcoholic liver cirrhosis, representing end-stage disease, will develop in 10 to 20% of patients who continue to drink.⁸ Variable factors associated with the development of alcoholic liver disease include the amount and duration of alcohol intake, genetic and metabolic factors, and the patient's underlying nutritional status. Not all individuals who abuse alcohol develop significant liver damage, however, a linear correlation exists between the duration and dose of alcohol use, and the development of alcoholic liver disease. The most common cirrhosis subtype, alcoholic liver cirrhosis, is more likely if alcohol consumption is greater than 160 g/day.⁵ Alcoholic cirrhosis is the most severe form of alcohol-related liver injury.⁹

Most prominent in this patient was the presence of gross, generalized edema and ascites. Of clinical importance, the patient did not exhibit any signs and symptoms characteristic of hepatic encephalopathy, which includes: the presence of asterix, changes in behaviour, or alterations in the level of consciousness. Although the patient quit drinking 1.5 years ago, he admits to a 20-year history of drinking up to 1180 g/week of rum.

Clinical Diagnosis: Alcoholic liver disease

Pathological Discussion

An ultrasound-guided percutaneous liver biopsy was performed. A 1.2 cm core of white liver tissue was excised and embedded *in toto*. Light microscopy of the biopsy sample revealed extensive areas of regenerative micronodule formation with interspersed fibrous septa and large areas of extinction (Figure 1). These findings were consistent with the patient's clinical findings indicative of alcoholic liver disease. Using a modified version of the standard Laennec scoring system for chronic liver disease, a diagnosis of severe cirrhosis with no evidence of recent activity was made.

There are a number of factors that may influence the ease with which a diagnosis of cirrhosis can be made from a liver a biopsy. The amount and quality of the tissue samples, the size and positioning of contained nodules, the type of biopsy needle and associated procedures used (i.e. biopsy with laparoscopy,

transjugular biopsy) will influence the ease of making the diagnosis by reviewing pathologists. A number of other criteria are useful in attaining a tentative diagnosis of cirrhosis:¹⁰

- liver consistency at biopsy
- ease of specimen fragmentation
- abnormal structural changes (reticulin)
- hepatocellular changes such as regenerative hyperplasia, pleomorphism, large-cell or small-cell dysplasia, and excess copper-associated protein.

Examination of the biopsy sample demonstrated the two requisite features for the diagnosis of cirrhosis: diffuse parenchymal injury *and* widespread fibrosis. Histopathologically, parenchymal injury gives the appearance of structurally abnormal nodules of regenerating hepatocytes surrounded by interconnecting bands of fibrous tissue/scarring.^{2, 4, 7, 11} At least one nodule must be completely surrounded by fibrous tissue to support the diagnosis of cirrhosis.^{5,6} Cirrhotic livers can be micronodular (all or most nodules < 3 mm in diameter), macronodular (nodules > 3 mm), or mixed. Fibrosis results from an imbalance between collagen production and degradation. It is a common finding in end-stage chronic liver diseases; however, fibrosis alone is not necessarily synonymous with cirrhosis.

Ultrasound may also be helpful for the diagnosis of cirrhosis, but only if ascites is concomitantly present. Increased echogenicity and dense, irregularly distributed reflective regions characterize areas of cirrhosis. Ultrasound examination of this patient's liver revealed a coarse, nodular liver, which was small in size. In addition, a large amount of ascitic fluid was present.

Alcoholic liver cirrhosis is most commonly associated with micronodule formation. Furthermore, disease progression may convert micronodule formation into a nonspecific macronodular pattern. This process of conversion takes more than 2 years to develop and represents regeneration following abstinence.^{5, 7}

In order to distinguish alcoholic hepatitis from other causes of liver disease, certain characteristic features in the biopsy sample were closely examined. Morphological changes induced by alcoholic hepatitis include ballooning of liver cells and the presence of Mallory bodies, perivenular fibrosis and usually, but not always, hepatocellular steatosis. The biopsy specimen did not show evidence of hepatocyte ballooning or Mallory bodies, and less than 1% of all hepatocytes contained evidence of large droplet steatosis. Fatty change is usually a marker of recent alcohol consumption and can be used to determine the timeframe of disease activity. Absence of significant steatosis in this biopsy corroborates the patient's assertion that he has remained abstinent from alcohol over the past 1.5 years. There were few inflammatory cells (lymphocytes or neutrophils), bile ducts were seen in normal numbers, and there was no evidence of cholestasis. The iron stain was negative indicating that the patient does not have any of the inheritable iron-depositing disorders, such as hemochromatosis or thalassaemia, and he does not have cirrhosis with siderosis. In addition, there were no ground glass cells, thereby ruling out a diagnosis of hepatitis B virus infection.^{6,10}

Pathological Diagnosis: Alcoholic liver cirrhosis

Prognosis of Alcoholic Liver Cirrhosis

Alcoholic liver cirrhosis has a less favourable outcome in the elderly and in women. Cirrhosis is generally believed to be an irreversible process. However, with abstinence, and appropriate, timely therapy, the destructive process may be delayed. A number of disease features have been characterized which are known to have considerable prognostic value in determining patient outcome (Table 6).

Table 6
Features Associated with Improved Prognosis in Cirrhosis

- Alcoholic cirrhosis etiology offers a more favourable prognosis than cryptogenic (unidentified) cirrhosis
- Reversible precipitating factor
- Improvement within 1 month of hospital therapy
- Absence of jaundice
- Diuretic responsive, minimal ascites
- Large liver size
- Serum albumin > 25 g/L; absence of hyponatremia
- Absence of bruising and coagulopathy
- Absence of histological hepatic changes other than fatty infiltration

In general, abstinence from alcohol consumption increases survival in patients with both compensated and decompensated liver disease. Patients with alcoholic liver cirrhosis and ascites who continue to drink heavily have the worst prognosis.¹² Prognosis may also be related to age. In one study, one-year mortality among patients over 60 years of age was 50%, compared with a mortality of 7% in patients under the age of 60 years.¹³ Other prognostic indicators include amount of alcohol consumed, ratio of serum transaminases (AST/ALT), serum bilirubin and creatinine levels, size of esophageal varices, and severity of portal hypertension.¹⁴ Liver failure is the most common cause of death in patients with alcoholic liver cirrhosis (observed in 27-51% of cases). Other significant causes of mortality include gastrointestinal hemorrhage (9-47% of cases), renal failure (1-8% of cases), hepatoma formation (5-16% of cases), and infections (3-17% of cases).^{14, 15, 16}

Treatment Options

Various treatment options are available for the management of patients with cirrhosis and ascites. These approaches include dietary modification, pharmacologic regimens, and surgical interventions.

Dietary sodium restriction: Restricting salt intake will help to reduce the edema. A reasonable goal is a 40 mmol/day sodium diet.

Spirolactone: Spirolactone antagonizes the actions of aldosterone and is an effective diuretic agent in the management of ascites. It is potassium-sparing, thus limiting the development of hypokalemia. Potassium depletion in cirrhotic patients can contribute to the development of hepatic encephalopathy.

Patients who fail to respond to a combination of sodium restriction and diuretic therapy are said to be diuretic-resistant.

In these cases, repeated large volume paracentesis is beneficial. In addition to cosmetic and comfort benefits, it also lowers intra-abdominal pressure, resulting in a reduction in portal pressures, increased cardiac output, and a reduction in activation of neurohumoral effector mechanisms. The need to administer albumin in conjunction with large volume paracentesis remains controversial.¹⁷

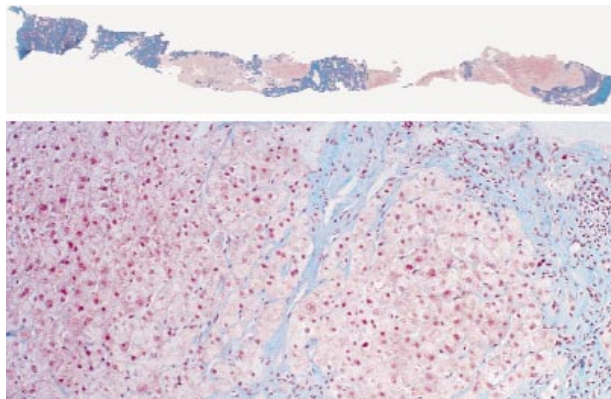


Figure 1.

Transjugular intrahepatic portosystemic shunt (TIPS): TIPS is a surgical procedure that lowers intrahepatic and portal venous pressures. It has primarily been used to control variceal bleeding but may also be used to manage patients with diuretic-resistant ascites. This radiologic intervention involves the creation of a tract within the liver parenchyma, which maintains patency between the portal and hepatic venous systems. Insertion of metallic stents redirect portal blood flow, thereby reducing portal venous hypertension and decompressing esophageal varices.¹⁸ The maximum benefit of TIPS on renal function and urinary sodium excretion is often delayed for several weeks following the procedure. Patient mobility is higher in patients who undergo TIPS, but TIPS also includes risks which include the precipitation of hepatic encephalopathy, stent infection, and stent thrombosis. Complications of TIPS insertion include intraperitoneal hemorrhage, jugular venous thrombus formation, fever, bacteremia, renal failure, and hepatic encephalopathy.

Peritovenous shunts (PVS): PVS are surgically implanted subcutaneous catheter systems that divert ascites fluid from the peritoneal cavity to the venous system. Two available PVS systems are the Denver and Le Vein shunts.¹⁸ The Le Vein Shunt involves the surgical insertion of a catheter incorporating a one-way pressure valve threaded under the skin to transfer fluid from the peritoneal cavity into the central circulation at the junction of the jugular vein or the superior vena cava.

Treatment of variceal bleeding: Beta-blockers have been demonstrated to reduce the risk of a first variceal bleed in patients with endoscopically visualized varices. Band ligation is the treatment of choice for the prevention of recurrent variceal bleeding.

Liver transplantation: In patients with decompensated end stage liver disease, orthotopic liver transplantation may be considered. Liver transplantation remains the only treatment that significantly improves the survival of patients with alcoholic liver disease who remain abstinent.¹⁸

Patient's Outcome

The diagnosis of severe alcoholic liver cirrhosis was discussed with the patient. Treatment included abstinence from alcohol and diuresis with furosemide and spironolactone. Although the patient has been abstinent from alcohol for 1.5 years and is committed to remaining abstinent, he did exhibit several features indicative of a poor prognosis: his ascites was minimally responsive to diuretic therapy; he had low serum albumin measurements and there was the presence of coagulopathy. There were also poor prognostic features on liver biopsy (multiple nodule formation and fibrosis, large areas of extinction, and distortion of the liver architecture).

On the day of discharge, an additional 2.0 L of ascitic fluid was removed. The discharge medications included Novolin 30/70 (30 units sc qam, 26 units sc qhs), furosemide 120 mg po daily, spironolactone 100 mg po daily, silver sulfadiazine topical ointment, and compression wrap of the lower extremity ulcers.

This patient is at increased risk for esophageal varices due to his existing portal hypertension. It was strongly recommended that he undergo a diagnostic esophago-gastro-duodenoscopy to determine whether varices were present. β -adrenergic blockade for the prevention of variceal bleeds may be considered. In addition, the possibility of TIPS insertion if the ascites did not respond to therapy was discussed with the patient.

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