Ascites in adults with cirrhosis: Initial therapy

AUTHOR: Bruce A Runyon, MD, FAASLD
SECTION EDITOR: Keith D Lindor, MD
DEPUTY EDITOR: Kristen M Robson, MD, MBA, FACG

All topics are updated as new evidence becomes available and our peer review process is complete.

Literature review current through: **Sep 2023.**
This topic last updated: **Apr 08, 2022.**

INTRODUCTION

Cirrhosis is the most common cause of ascites in the United States, accounting for approximately 85 percent of cases [1]. In addition, ascites is the most common complication of cirrhosis. Within 10 years after the diagnosis of compensated cirrhosis, approximately 58 percent of patients will have developed ascites [2]. (See "Pathogenesis of ascites in patients with cirrhosis".)

Successful treatment of the patient with ascites depends upon an accurate diagnosis regarding the cause of ascites formation [3,4]. A careful history, physical examination, and abdominal paracentesis with appropriate ascitic fluid analysis can usually determine the cause of ascites formation [1,3]. Patients who have a cause for ascites formation other than cirrhosis may not respond to the treatments used in those with cirrhosis. This is particularly true for ascites due to peritoneal carcinomatosis, in which sodium restriction and diuretics cause intravascular volume depletion without mobilization of the ascitic fluid [5]. (See "Evaluation of adults with ascites" and 'Diuretic therapy' below and "Malignancy-related ascites".)

This topic will review the initial therapy of ascites in patients with cirrhosis. The diagnosis and evaluation of patients with ascites, the treatment of refractory ascites, and the management of spontaneous bacterial peritonitis are discussed elsewhere. (See "Evaluation of adults with ascites" and "Ascites in adults with cirrhosis: Diuretic-resistant ascites" and "Spontaneous bacterial peritonitis in adults: Treatment and prophylaxis".)

The management of ascites in adults with cirrhosis is also discussed in practice guidance from the American Association for the Study of Liver Diseases [6]. The discussion that follows is generally consistent with that guidance.

GOALS OF THERAPY

The goals of therapy in patients with ascites are to minimize ascitic fluid volume and decrease peripheral edema, without causing intravascular volume depletion. Although there is no evidence that treatment of fluid overload in patients with cirrhosis improves survival, the following benefits have been noted:

- Patients report that with less fluid they feel much better, have less abdominal discomfort, can move and eat more easily, and have less shortness of breath.
- As fluid is removed, ascitic fluid opsonins become more concentrated, which may protect against spontaneous bacterial peritonitis [7,8].
- Reductions in the risk of cellulitis and of abdominal wall hernia formation or diaphragmatic rupture (and resulting hepatic hydrothorax) associated with tense ascites [9].
- Reduced amount of energy expended heating the ascitic fluid [9].

THRESHOLD FOR INITIATING TREATMENT

Once a patient with cirrhosis develops clinically apparent ascites, it is unlikely to resolve without specific treatment (eg, a sodium-restricted diet and diuretics). Three possible exceptions to this rule are:

- Patients with alcohol-associated cirrhosis who have a huge dietary sodium intake and a large reversible component of liver disease
- Patients with alcoholic hepatitis
- Patients who develop ascites for the first time in the setting of resuscitation for upper gastrointestinal bleeding
- Patients with decompensated hepatitis B cirrhosis

In such patients, ascites may resolve following cessation of drinking and reduction of sodium intake, discontinuation of intravenous fluids, and treatment with antiviral therapy, respectively.

The natural history of small-volume ascitic fluid that is only apparent on imaging with no perceived swelling of the abdomen is finally receiving some attention; it can spontaneously disappear [10]. We only initiate therapy once the ascites is clinically apparent.

THERAPEUTIC APPROACH

The treatment of ascites in patients with cirrhosis includes abstinence from alcohol, restricting dietary sodium, and treating with diuretics (algorithm 1). In addition to treatment with alcohol abstinence, sodium restriction, and diuretics, patients with tense ascites should undergo an initial large-volume therapeutic paracentesis. (See 'Alcohol abstinence' below and 'Dietary sodium restriction' below and 'Diuretic therapy' below and 'Large-volume paracentesis' below.)

Alcohol abstinence is an important component of treatment because it dramatically improves ascites in some patients with alcohol-induced liver disease. Pharmacologic treatment with baclofen may improve alcohol abstinence. (See 'Alcohol abstinence' below.)

Patients with ascites should be restricted to 88 mEq (2000 mg) of sodium per day. Since fluid follows sodium passively, fluid restriction is generally not required for the treatment of ascites, though it may be indicated in patients with ascites and a very low serum sodium (less than 120 mEq/L) (see "Hyponatremia in patients with cirrhosis"). Sodium restriction alone (ie, without the addition of diuretics) will be sufficient treatment only in the small subset of patients whose urinary sodium excretion (in the absence of diuretics) is more than 78 mEq per day (88 mEq intake minus 10 mEq of non-urinary losses).

Most patients with cirrhosis and ascites require both dietary sodium restriction and diuretics. When given the option of a more restrictive diet without diuretics or less restrictive diet with diuretics, most patients choose the latter. Our approach is to prescribe diuretics in combination with sodium restriction to all patients with cirrhosis and clinically detectable ascites [1,3]. The diuretic doses can be tapered or temporarily discontinued if weight loss is rapid. Diuretic therapy typically consists of treatment with spironolactone and furosemide in a ratio of 100:40 mg per day, with doses titrated upward every three to five days as needed (up to 400 mg spironolactone and 160 mg furosemide per day). (See 'Diuretic therapy' below.)

Several medications should be avoided or used with caution in patients with ascites, including angiotensin converting enzyme inhibitors, angiotensin II receptor blockers, and nonsteroidal

antiinflammatory drugs.

In addition, consideration should be given to stopping or not initiating nonselective beta blockers such as propranolol in patients with refractory ascites, as they may shorten survival in such patients. (See 'Medications to avoid or use with caution' below.)

Management of underlying liver disease — In some cases, management of the underlying liver disease can treat reversible components of a patient's hepatic decompensation and lead to improvement or resolution of ascites. This can be seen in patients with cirrhosis due to alcohol-induced liver disease, autoimmune hepatitis, or chronic hepatitis B virus. By contrast, most other causes of cirrhosis are not reversible. It may be more appropriate to consider liver transplantation for definitive treatment of these diseases, rather than protracted medical therapy of ascites [1]. (See "Liver transplantation in adults: Patient selection and pretransplantation evaluation".)

Alcohol abstinence — Patients who are actively consuming alcohol should be encouraged to abstain completely. Even if alcohol is not the only cause of their liver disease (eg, in patients with cirrhosis due to chronic hepatitis C), the alcohol-related component of their liver disease may improve dramatically with abstinence. Alcoholism is commonly the driving force for mortality in patients with chronic hepatitis C virus who come to medical attention [11]. Pharmacologic therapy with baclofen has been shown to improve abstinence in patients with alcohol-associated liver disease.

Among patients with alcoholic cirrhosis, abstinence may lead to improvement of hepatic histology in some patients with marked fibrosis and inflammation [12,13], a reduction in or even normalization of the portal pressure [14], and disappearance of or easier to treat ascites [15]. Perhaps the most common setting in which patients with refractory ascites revert back to diuretic-responsive ascites is alcohol-associated liver disease with total abstinence from alcohol. (See "Management of alcohol-associated steatosis and alcohol-associated cirrhosis".)

Pharmacologic therapy for alcohol dependence may improve abstinence. Baclofen was shown in a randomized trial with patients with alcoholic liver disease to reduce alcohol craving and consumption [16], though trials in other populations have had mixed results [17,18]. We have had good results with the use of baclofen in patients with alcohol-associated liver disease, so our practice is to use baclofen to aid with abstinence, particularly in patients who have been admitted with alcoholic hepatitis [19]. The starting dose is 5 mg three times per day for three days. The dose is then increased to 10 mg three times per day. The dose can be increased further as needed until the craving for alcohol is extinguished. We will use doses up to 95 mg

daily in five divided doses. The pills can be carried by the patient and taken when the patient feels a craving for alcohol [20].

Management of other liver diseases — Patients with decompensated cirrhosis due to autoimmune hepatitis may have a reversible component with glucocorticoid therapy with or without azathioprine. (See "Management of autoimmune hepatitis".)

Patients with decompensated cirrhosis due to hepatitis B can have dramatic improvement with effective antiviral treatment, and there is evidence that cirrhosis can reverse with successful treatment [21].

Medications to avoid or use with caution — In patients with cirrhosis, lower arterial blood pressure is associated with lower survival rates [22], so medications that decrease blood pressure might decrease survival. In addition, propranolol may decrease survival in patients with refractory ascites [23]. We stop or avoid initiating angiotensin converting enzyme inhibitors (ACEIs), angiotensin II receptor blocker (ARBs), and beta blockers in patients with progressive cirrhosis whose blood pressure is declining, and we do not use beta blockers in patients with refractory ascites. Other classes of antihypertensive drugs should be used only if the blood pressure is above goal.

Prostaglandin inhibitors, such as nonsteroidal anti-inflammatory drugs (NSAIDS), should also be avoided since they can reduce urinary sodium excretion and induce azotemia [24].

Avoidance of angiotensin inhibition — Systemic blood pressure tends to progressively fall with increasing severity of cirrhosis, particularly in patients with decompensated liver disease. The progressive decrease in systemic blood pressure is associated with reductions in renal perfusion and glomerular filtration rate and increases in the activity of the renin-angiotensin system [4]. This leads to elevated serum levels of vasoconstrictors such as vasopressin, angiotensin, and aldosterone. These vasoconstrictors support the patients' blood pressure by compensating for the vasodilatory effects of nitric oxide [6]. Because arterial pressure is directly related to survival [22], we suggest that ACEIs and ARBs be avoided in patients with ascites, a position that is also supported by the American Association for the Study of Liver Diseases and the European Association for the Study of the Liver [4,6].

Occasional patients with cirrhosis also have proteinuric chronic kidney disease due to diabetes mellitus or other conditions. In patients who do not have cirrhosis, ACEIs and ARBs can slow the rate of progression of renal disease and are widely used. The goal blood pressure in such patients is less than 130:80 mmHg. However, there are no clinical trials of angiotensin inhibition to slow the progression of chronic proteinuric kidney disease in patients with decompensated cirrhosis. Furthermore, such patients often have systolic pressures below 100 mmHg and, due

to the increased activity of the renin-angiotensin-aldosterone system, are at risk for a marked reduction in blood pressure and worsening renal function with angiotensin inhibitor therapy. As a result, we typically do not use ACEIs and ARBs in patients with proteinuric chronic kidney disease who have decompensated cirrhosis. (See "Antihypertensive therapy and progression of nondiabetic chronic kidney disease in adults" and "Treatment of diabetic kidney disease".)

If patients with ascites are started on an ACEI or an ARB, they must have their blood pressure and renal function monitored closely.

Propranolol — Although propranolol has been shown to prevent variceal hemorrhage in patients with large varices, studies suggest it may worsen survival in patients with refractory ascites [23]. We have seen this drug reduce blood pressure in patients with ascites and make the fluid more diuretic-resistant. Patients with ascites who are started on a beta blocker should have their blood pressure and renal function monitored carefully. The risks versus benefits of beta blockers must be carefully weighed in each patient with advanced cirrhosis. In general, we do not use propranolol in patients with refractory ascites. (See "Ascites in adults with cirrhosis: Diuretic-resistant ascites", section on 'Discontinuing beta blockers'.)

Cirrhosis cures hypertension — As patients with cirrhosis decompensate with accumulation of ascites, their systemic vascular resistance and mean arterial pressure (MAP) silently decrease. Many patients, especially those with obesity and cirrhosis due to nonalcoholic steatohepatitis, are hypertensive before they decompensate (hence the phrase "cirrhosis cures hypertension"). When MAP is ≤82 mmHg, two-year survival is only 20 percent; whereas when MAP >82 mmHg, two-year survival is 70 percent [22]. It has been this author's (BAR) experience that the MAP threshold (82 mmHg) applies to all patients (ie, those with spontaneous or iatrogenic hypotension, resulting from antihypertensive medication use). When MAP is ≤82 mmHg, medications that lower blood pressure must be stopped. If they are continued, worsening hypotension, hyponatremia, and azotemia are expected.

Too often, clinicians who start beta blockers or other antihypertensive medications do not follow these patients for the long term and may not realize that antihypertensive medications are not tolerated once ascites develops, especially diuretic-resistant ascites.

Older practice guidelines regarding variceal bleeding recommended beta blocker therapy without plans for discontinuation (ie, no stopping rules). However, attention has been drawn to the adverse effects of beta blockers in patients with ascites and the need for guidance regarding discontinuing therapy [25]. Updated practice guidance and guidelines have added thresholds for drug discontinuation but chose blood pressure <90/60 mmHg rather than MAP <82 mmHg [26,27]. There are no data to support a threshold <90/60 mmHg, which equates to a

MAP of 70 mmHg. For example, MAP <60 mmHg was associated with a higher mortality rate. In a retrospective study including 183 patients with cirrhosis who required intensive care unit admission, patients with nadir MAP <60 mmHg during the first hospital day had a higher mortality rate compared with MAP ≥60 mmHg (43 versus 7 percent) [28]. MAP <60 mmHg is dangerously close to 70 mmHg, the threshold used by practice guidelines. Many hospitals may still have local recommendations based on older practice guidelines that lead to excessive use of beta blockers. This can lead to iatrogenic hypotension with resulting hyponatremia, azotemia, and mortality.

Avoidance of NSAIDs — A general component of the therapy of patients with cirrhosis and ascites is avoidance of the use of NSAIDs. These agents inhibit the synthesis of renal prostaglandins, leading to renal vasoconstriction, a lesser response to diuretics, and the possible precipitation of acute renal failure [29]. NSAIDs can also precipitate upper gastrointestinal bleeding in the setting of cirrhosis [30]. (See "NSAIDs: Electrolyte complications" and "NSAIDs: Acute kidney injury".)

Dietary sodium restriction — A mainstay in the treatment of ascites is to restrict sodium intake to 88 mEq (2000 mg) per day (including all foods, liquids, and medications) [1,6]. In motivated patients who receive appropriate education, this can be done without the purchase of special food (figure 1 and table 1). (See "Patient education: Low-sodium diet (Beyond the Basics)".)

The development of ascites in a patient with portal hypertension is the consequence of avid renal retention of sodium and water. Patients with cirrhosis whose urinary excretion of sodium is significantly less than dietary intake will accumulate sodium and water, eventually leading to ascites and perhaps peripheral edema. (See "Pathogenesis of ascites in patients with cirrhosis".)

Removal of ascites and edema requires the induction of negative sodium balance. In addition to cosmetic benefits and the possible relief of abdominal discomfort, fluid removal also may produce a modest reduction in portal pressure, presumably mediated by the reduction in plasma volume [31]. It may also decrease the risk of ascitic fluid infection by increasing ascitic fluid opsonic activity [7,8]. As liver function deteriorates, urinary sodium excretion falls progressively and can approach zero [15,32,33]. In this setting, the induction of negative sodium balance requires the combination of sodium restriction and diuretics. (See 'Diuretic therapy' below.)

Education about the importance of dietary sodium restriction is a central component of the management of ascites. The patient and the cook (if the patient is not the cook) should meet with a dietitian for an education session. In our experience, most patients do not receive this

education session and are therefore much less likely to follow the diet. Those who understand and follow the diet usually do not require readmission for fluid overload; by comparison, noncompliant patients often require multiple hospitalizations.

Fluid restriction — Fluid restriction has traditionally been recommended in patients with cirrhosis and ascites who have hyponatremia. However, such an approach is not supported by the available data and will make an already thirsty patient more uncomfortable, just to correct an asymptomatic laboratory abnormality. We only limit fluid intake when the plasma sodium is less than 120 mEq/L, which is an uncommon finding (1 percent in a multicenter review of 997 consecutive patients) [34], or the patient has neurologic symptoms that might be due to hyponatremia. (See "Hyponatremia in patients with cirrhosis".)

For fluid restriction to raise the serum sodium, the total fluid intake should be less than the urine volume, which is often quite low and therefore difficult to achieve in patients with cirrhosis who have hyponatremia. Sucking on ice chips or lollipops may be helpful in patients with severe thirst.

Diuretic therapy — Most patients with clinically apparent ascites will require diuretic therapy in addition to dietary sodium restriction. Diuretic therapy typically consists of treatment with spironolactone and furosemide in a ratio of 100:40 mg per day, with doses titrated upward as needed (up to 400 mg spironolactone and 160 mg furosemide per day). Concerns with diuretic therapy include overly rapid removal of fluid and electrolyte imbalances.

Dietary sodium restriction and diuretic therapy is effective in approximately 90 percent of patients with cirrhosis and ascites. In patients who appear to be diuretic-resistant, it is important to exclude lack of adherence with dietary sodium restriction. (See 'Diuretic resistance' below.)

Diuretic regimen — The most successful therapeutic regimen for cirrhotic ascites is the combination of oral spironolactone and furosemide, beginning with 100 mg and 40 mg, respectively [35]. However, in small patients (patients who weigh approximately 50 kg or less) with a small volume of ascites, we use lower doses (eg, 50 mg of spironolactone and 20 mg of furosemide). If a clinical response is not evident after three to five days, or if the degree of weight loss is less than desired, the doses can be increased by 100 mg and 40 mg, respectively. This can be repeated if needed. The maximum recommended doses are spironolactone 400 mg per day and furosemide 160 mg per day [1,6].

The long half-life of spironolactone makes single-daily dosing most appropriate. Giving both drugs once per day in the morning also maximizes adherence and minimizes nocturia. (See "Loop diuretics: Dosing and major side effects".)

It is important not to treat fluid overload from cirrhotic ascites with intravenous furosemide, as is often done in patients with heart failure. With heart failure, clinicians may use intravenous furosemide if there is concern that oral furosemide is being poorly absorbed. However, this is not appropriate in patients with cirrhosis because oral furosemide is absorbed well in these patients [36]. In patients with cirrhosis, giving intravenous furosemide often causes an acute reduction in renal function that cannot be explained by the diuresis [37] and may lead to crescendo azotemia that may then masquerade as hepatorenal syndrome. We generally suggest that intravenous furosemide be avoided for the treatment of ascites unless there is no other option (eg, soft tissue or pulmonary edema in a patient taking nothing by mouth). However, a single dose of intravenous furosemide (80 mg) may help identify patients who are diuretic-resistant. Patients who are diuretic resistant will typically secrete <50 mEq of urine sodium over eight hours following a dose of intravenous furosemide [38].

At least one randomized trial demonstrated that concomitant treatment with both spironolactone and furosemide can mobilize moderate ascites more rapidly than sequential diuretics (ie, spironolactone alone followed by adding furosemide) [39]. In addition, using a combination of spironolactone and furosemide in a ratio of 100:40 mg usually maintains normokalemia. Single-agent spironolactone, especially at higher doses, regularly causes hyperkalemia in patients with cirrhosis and ascites. The concomitant use of furosemide usually leads to better urine sodium excretion as well as normokalemia. However, patients with parenchymal renal disease or who have undergone liver transplantation may require lower doses of spironolactone to prevent hyperkalemia. In such patients, we use a ratio of spironolactone to furosemide that is less than 100:40 (eg, 100:80 or 100:120). It is a matter of trial and error to achieve natriuresis without hyperkalemia. In some cases, we cannot use any spironolactone, especially when the glomerular filtration rate is very low or the patient develops hyperkalemia.

The only setting in which we begin with spironolactone monotherapy is in patients with profound hypokalemia (most often seen in the setting of severe alcoholic hepatitis). Furosemide is added once the potassium normalizes and potassium replacement is no longer needed.

Increasingly, older adults are being diagnosed with cirrhosis and ascites, including patients >90 years of age. These patients have silently sustained a decrease in glomerular filtration rate with aging and are intolerant of high-dose spironolactone. The usual starting doses of diuretics in these patients are 25 mg of spironolactone with 20 mg of furosemide orally daily. If that is not enough, 50 mg of spironolactone with 40 mg of furosemide daily can be given. The risks of higher doses must be weighed against the benefits. Older patients are more prone to falls than younger patients. It is preferable to tolerate a little fluid overload in these fragile patients rather

than pushing to euvolemia or hypovolemia. It has been said, "Better wet and wise than dry and with dementia" [40]. This recommendation is even more relevant in older adults.

It is important to avoid hypokalemia in patients with cirrhosis and ascites because potassium depletion can enhance renal ammonia production and possibly precipitate hepatic coma [41]. A transcellular cation exchange, in which potassium moves out of cells (to partially replete extracellular potassium stores) in exchange for hydrogen, is thought to be responsible for this effect, since the ensuing intracellular acidosis stimulates ammonia synthesis in the proximal tubular cells, which can contribute to hyperammonemia. Our goal is to maintain the plasma potassium concentration above 3.4 mEq/L but below 5 mEq/L. (See "Hypokalemia-induced kidney dysfunction", section on 'Increased ammonia production'.)

Interestingly, spironolactone (an aldosterone antagonist) alone is more effective than furosemide alone in patients with cirrhosis [35,42]. The surprising finding that a normally weak diuretic may be more effective than a loop diuretic in cirrhotic ascites may be related to differences in the mechanism of drug action [42]. Loop and thiazide diuretics must enter the tubular lumen to be effective. Most of these drugs are highly protein-bound; as a result, they enter the tubular lumen by secretion in the proximal tubule, not by glomerular filtration. This secretory process appears to be impaired in cirrhosis, perhaps due to competitive or toxic inhibition by retained compounds such as bile salts. The net effect is that diuretic entry into the lumen, and therefore the natriuretic effect, may be limited [43].

Spironolactone is occasionally associated with painful gynecomastia. As a result, amiloride (10 to 40 mg per day), another potassium-sparing diuretic that directly closes the aldosterone-sensitive luminal sodium channels in the collecting tubules, has also been used in the treatment of ascites. However, amiloride appears be less effective than spironolactone in patients with cirrhosis [44].

Patients who are forewarned about the risk of gynecomastia are usually willing to continue spironolactone despite its occurrence. Eplerenone is a newer, highly selective mineralocorticoid receptor antagonist. In contrast to spironolactone, eplerenone is not associated with an increased risk of gynecomastia and has been associated with resolution of gynecomastia after discontinuing spironolactone [45]. However, it is far more expensive than spironolactone, and no study has compared the efficacy of spironolactone with that of eplerenone in patients with ascites. In our experience, eplerenone seems to be less effective than spironolactone in the treatment of ascites. (See "Secondary pharmacologic therapy for heart failure with reduced ejection fraction".)

Rate of fluid removal — The rate at which fluid can safely be removed in cirrhosis depends upon the presence or absence of peripheral edema. When a diuresis is induced, the fluid is initially lost from the vascular space; the ensuing fall in intravascular pressure allows the edema fluid to be mobilized to replete the plasma volume. Fluid mobilization can be rapid (in some cases, exceeding 2 kg per day without detectable intravascular volume depletion) in patients with peripheral edema [46].

By comparison, patients who only have ascites without edema can mobilize ascitic fluid solely via the peritoneum. The maximum rate at which this can occur is only 300 to 500 mL per day; more rapid fluid removal with diuretics (weight loss >0.75 kg per day) can lead to plasma volume depletion and azotemia [46,47]. If more rapid fluid removal is required, an initial large-volume paracentesis should be performed. (See 'Large-volume paracentesis' below.)

Patient monitoring — One of the most important concepts in the treatment of patients with cirrhosis and ascites is feedback for the clinician. When a decision is made to start or to alter the treatment regimen, the clinician needs to know if the decision was a good one. Patients must be reevaluated frequently with daily weights in the hospital or at home.

For patients who are admitted to the hospital for the treatment of ascites, the first clinic visit should be one to two weeks following discharge. A study in heart failure showed that a seven-day clinic return helped prevent readmission within 30 days of discharge [48]. Patients sent home with a remote appointment are regularly readmitted with worsening fluid overload, overdiuresis, or electrolyte imbalances before they are due back in the clinic. If distant travel is a problem, "telephone medicine" can permit adjustment of diuretic doses based upon weights, faxed laboratory results, and symptoms [49].

Patients with a component of alcohol injury to their liver disease may become less sodium avid with abstinence and healing of the reversible component of their liver injury. Some may be able to be tapered off diuretics, and some revert from diuretic resistance to diuretic sensitivity. Clues that the patient may no longer need diuretics include orthostatic symptoms, azotemia, and the absence of ascites and edema. Once patients lose their sodium avidity, we begin tapering the diuretics, reducing to a final dose of 50 mg of spironolactone plus 20 mg of furosemide before discontinuing altogether.

Diuretics should be stopped if patients develop uncontrolled or recurrent encephalopathy, a serum sodium less than 120 mEq/L despite fluid restriction (such patients typically have very low urine sodium output and pre-hepatorenal syndrome), or a serum creatinine greater than 2.0 mg/dL (180 micromol/L). These patients may need alternative therapies, such as serial paracenteses. In patients with poor renal perfusion, the addition of midodrine may allow for the

reinstitution of diuretics. (See "Ascites in adults with cirrhosis: Diuretic-resistant ascites", section on 'Initial treatments' and "Ascites in adults with cirrhosis: Diuretic-resistant ascites", section on 'Options if initial treatments fail' and "Ascites in adults with cirrhosis: Diuretic-resistant ascites", section on 'Oral midodrine'.)

Diuretic resistance — Dietary sodium restriction and diuretic therapy is initially effective in approximately 90 percent of patients with cirrhosis and ascites [50]. However, in patients with no reversible component to their liver injury, the ascites usually worsens over time and progresses from being diuretic-sensitive to being diuretic-resistant. In patients who appear to be diuretic-resistant, it is important to exclude lack of adherence with dietary sodium restriction. This is done by testing urinary sodium excretion. Patients who are diuretic-resistant will have inadequate excretion, whereas those who are not adherent to the sodium restriction will gain weight despite what would otherwise be adequate excretion. (See "Ascites in adults with cirrhosis: Diuretic-resistant ascites", section on 'Diagnosis'.)

Large-volume paracentesis — If the patient has tense ascites or if the patient or the clinician is in a hurry to decompress the abdomen, a 4 to 5 L paracentesis is more rapid than diuretic therapy. Removal of less than 5 liters of fluid does not appear to have hemodynamic or hormonal consequences, and post-paracentesis colloid infusion does not appear to be necessary [51]. For larger paracenteses, albumin (6 to 8 g/L of fluid removed) can be administered [52]. A meta-analysis demonstrated a survival advantage with albumin infusion after paracentesis involving mean volumes of 5.5 to 15.9 liters [53]. (See "Ascites in adults with cirrhosis: Diuretic-resistant ascites", section on 'Therapeutic paracentesis'.)

Liver transplantation and shunts — Patients with liver disease severe enough to be complicated by ascites should be considered for liver transplantation and referred for evaluation unless they have medical or psychosocial contraindications. Other methods to control ascites, such as serial therapeutic paracentesis and transjugular intrahepatic portosystemic shunts (TIPS) are usually reserved for patients with refractory ascites. Peritoneovenous shunts (LeVeen or Denver) or surgical portosystemic shunts have very limited indications, even in those with refractory ascites. (See "Liver transplantation in adults: Patient selection and pretransplantation evaluation" and "Ascites in adults with cirrhosis: Diuretic-resistant ascites".)

Other treatments — Other potential treatments include:

- Chronic albumin therapy – For patients with decompensated cirrhosis, long-term administration of albumin has been studied in Europe, although chronic albumin therapy is not standard practice [54,55]. In a randomized, open-label trial including 440 patients

with cirrhosis and uncomplicated ascites, transplant-free survival rates at 18 months were higher in patients given albumin infusions (40 grams twice weekly for two weeks, then weekly) in addition to standard care (diuretics and large volume paracentesis, as needed) compared with patients receiving standard care alone (77 versus 66 percent; HR 0.62, 95% 0.40-0.95) [54]. Additional trials in the United States are needed to determine if albumin infusion should be incorporated into clinical practice and if the potential benefits outweigh the risks and barriers to its use (eg, availability, cost).

- Sodium-glucose co-transporter 2 inhibitors – Sodium-glucose co-transporter 2 (SGLT2) inhibitors are used for hyperglycemia in patients with type 2 diabetes mellitus. SGLT2 inhibitors reduce blood glucose by increasing urinary glucose excretion. In a case series including three patients with cirrhosis, type 2 diabetes mellitus, and fluid retention, SGLT2 inhibitor therapy was associated with reduction in ascites and peripheral edema [56]. Clinical trials are needed to explore the use of SGLT2 inhibitors in patients with ascites related to cirrhosis. (See "Sodium-glucose cotransporter 2 inhibitors for the treatment of hyperglycemia in type 2 diabetes mellitus".)

MANAGEMENT OF COMPLICATIONS

Patients with ascites may develop complications such as spontaneous bacterial peritonitis, umbilical hernias, and hepatic hydrothorax. The management of these complications is discussed elsewhere. (See "Spontaneous bacterial peritonitis in adults: Treatment and prophylaxis", section on 'Treatment' and "Hepatic hydrothorax", section on 'Management' and "Cirrhosis in adults: Overview of complications, general management, and prognosis", section on 'Umbilical hernias'.)

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Cirrhosis" and "Society guideline links: Portal hypertension and ascites".)

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given

condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topics (see "Patient education: Cirrhosis (The Basics)" and "Patient education: Low-sodium diet (The Basics)" and "Patient education: Fluid in the belly (ascites) (The Basics)")
- Beyond the Basics topics (see "Patient education: Cirrhosis (Beyond the Basics)" and "Patient education: Low-sodium diet (Beyond the Basics)")

SUMMARY AND RECOMMENDATIONS

- **Goals of therapy –** Once a patient with cirrhosis develops clinically apparent ascites, it is unlikely to resolve without specific treatment. Treatment of ascites in patients with cirrhosis is aimed at the underlying cause of the hepatic disease and at the sodium and water retention (algorithm 1). The goals of therapy in patients with ascites are to minimize ascitic fluid volume and decrease peripheral edema, without causing intravascular volume depletion. (See 'Goals of therapy' above and 'Therapeutic approach' above.)
- **Alcohol abstinence –** Patients who are actively consuming alcohol should be encouraged to abstain completely. Baclofen can assist in maintaining abstinence by reducing alcohol craving. Even if alcohol is not the only cause of their liver disease (eg, in patients with cirrhosis due to chronic hepatitis C), the alcohol-related component of their liver disease and their ascites may improve dramatically with abstinence. (See 'Alcohol abstinence' above.)
- **Medications to avoid or use with caution –** In patients with cirrhosis, lower arterial blood pressure is associated with lower survival rates, so medications that decrease blood pressure might decrease survival. In addition, propranolol may decrease survival in patients with refractory ascites.

We stop or avoid initiating angiotensin converting enzyme inhibitors (ACEIs), angiotensin II receptor blocker (ARBs), and beta blockers in patients with progressive cirrhosis whose blood pressure is declining, and we do not use beta blockers in patients with refractory ascites. Prostaglandin inhibitors, such as nonsteroidal antiinflammatory drugs (NSAIDS), should also be avoided since they can reduce urinary sodium excretion and induce azotemia. (See 'Medications to avoid or use with caution' above.)

- **Dietary restriction –** We suggest an 88 mEq (2000 mg) per day sodium restriction (**Grade 2C**). Sodium restriction alone (ie, without the addition of diuretics) will be sufficient treatment only in the small subset of patients whose urinary sodium excretion (in the absence of diuretics) is more than 78 mEq per day. Education about the importance of dietary sodium restriction is a central component of the management of cirrhotic ascites (figure 1 and table 1). (See 'Dietary sodium restriction' above.)

  Although fluid restriction is frequently ordered, its role is unclear, particularly since adherence is difficult to achieve. Thus, we generally only recommend fluid restriction in patients whose plasma sodium concentration is <120 mEq/L. (See "Hyponatremia in patients with cirrhosis".)

- **Diuretic therapy –** In patients who require diuretic therapy, we suggest combination therapy with spironolactone and furosemide rather than sequential therapy (spironolactone followed by the addition of furosemide) (**Grade 2B**). We typically begin with spironolactone 100 mg and furosemide 40 mg once daily in the morning. In small patients with a small volume of ascites, we use lower doses (eg, 50 mg of spironolactone and 20 mg of furosemide). If a clinical response is not evident after three to five days, or if the degree of weight loss is less than desired, the doses can be increased by 100 mg and 40 mg, respectively. This can be repeated if needed. The maximum recommended doses are spironolactone 400 mg per day and furosemide 160 mg per day. (See 'Diuretic regimen' above.)

  In patients with parenchymal renal disease, we use a ratio of spironolactone to furosemide that is less than 100:40 (eg, 100:80 or 100:120). It is a matter of trial and error to achieve natriuresis without hyperkalemia. In some cases, we cannot use any spironolactone, especially when the glomerular filtration rate is very low or the patient develops hyperkalemia.

  Older adults also do not tolerate the usual ratio of diuretics. Starting doses of diuretics in these patients are 25 mg of spironolactone with 20 mg of furosemide orally daily. If that is not enough, 50 mg of spironolactone with 40 mg of furosemide daily can be given.

We suggest that intravenous furosemide be avoided unless there is no other option (eg, soft tissue or pulmonary edema in a patient taking nothing by mouth) (**Grade 2C**). Oral furosemide is absorbed well in the setting of cirrhosis, and intravenous furosemide may cause an acute reduction in renal function and can lead to crescendo azotemia that may then masquerade as hepatorenal syndrome. (See 'Diuretic resistance' above.)

- **Monitoring –** When a decision is made to start or to alter the treatment regimen, the clinician needs to know if the decision was a good one. Patients must be reevaluated frequently with daily weights in the hospital or at home. For patients who are admitted to the hospital for the treatment of ascites, the first clinic visit should be one to two weeks following discharge. (See 'Patient monitoring' above.)

- **Patients with suspected diuretic resistance –** Dietary sodium restriction and diuretic therapy is effective in approximately 90 percent of patients with cirrhosis and ascites. In patients who appear to be diuretic-resistant, it is important to exclude lack of adherence with dietary sodium restriction. (See 'Diuretic resistance' above.)

ACKNOWLEDGMENT

The UpToDate editorial staff thank Dr. José Such, MD, PhD for his contributions as author to prior versions of this topic review.

REFERENCES

1. Runyon BA, Montano AA, Akriviadis EA, et al. The serum-ascites albumin gradient is superior to the exudate-transudate concept in the differential diagnosis of ascites. Ann Intern Med 1992; 117:215.
2. Ginés P, Quintero E, Arroyo V, et al. Compensated cirrhosis: natural history and prognostic factors. Hepatology 1987; 7:122.
3. Runyon BA, AASLD Practice Guidelines Committee. Management of adult patients with ascites due to cirrhosis: an update. Hepatology 2009; 49:2087.
4. European Association for the Study of the Liver. Electronic address: easloffice@easloffice.eu, European Association for the Study of the Liver. EASL Clinical Practice Guidelines for the management of patients with decompensated cirrhosis. J Hepatol 2018; 69:406.

5. Pockros PJ, Esrason KT, Nguyen C, et al. Mobilization of malignant ascites with diuretics is dependent on ascitic fluid characteristics. Gastroenterology 1992; 103:1302.
6. Biggins SW, Angeli P, Garcia-Tsao G, et al. Diagnosis, Evaluation, and Management of Ascites, Spontaneous Bacterial Peritonitis and Hepatorenal Syndrome: 2021 Practice Guidance by the American Association for the Study of Liver Diseases. Hepatology 2021; 74:1014.
7. Runyon BA, Van Epps DE. Diuresis of cirrhotic ascites increases its opsonic activity and may help prevent spontaneous bacterial peritonitis. Hepatology 1986; 6:396.
8. Runyon BA, Antillon MR, McHutchison JG. Diuresis increases ascitic fluid opsonic activity in patients who survive spontaneous bacterial peritonitis. J Hepatol 1992; 14:249.
9. Dolz C, Raurich JM, Ibáñez J, et al. Ascites increases the resting energy expenditure in liver cirrhosis. Gastroenterology 1991; 100:738.
10. Tonon M, Piano S, Grbec M, et al. What is the evolution of grade 1 ascites in patients with cirrhosis? Hepatology 2015; 62:360A.
11. Said A, Williams J, Holden J, et al. The prevalence of alcohol-induced liver disease and hepatitis C and their interaction in a tertiary care setting. Clin Gastroenterol Hepatol 2004; 2:928.
12. Powell WJ Jr, Klatskin G. Duration of survival in patients with Laennec's cirrhosis. Influence of alcohol withdrawal, and possible effects of recent changes in general management of the disease. Am J Med 1968; 44:406.
13. Veldt BJ, Lainé F, Guillygomarc'h A, et al. Indication of liver transplantation in severe alcoholic liver cirrhosis: quantitative evaluation and optimal timing. J Hepatol 2002; 36:93.
14. REYNOLDS TB, GELLER HM, KUZMA OT, REDEKER AG. Spontaneous decrease in portal pressure with clinical improvement in cirrhosis. N Engl J Med 1960; 263:734.
15. Runyon BA. Historical aspects of treatment of patients with cirrhosis and ascites. Semin Liver Dis 1997; 17:163.
16. Addolorato G, Leggio L, Ferrulli A, et al. Effectiveness and safety of baclofen for maintenance of alcohol abstinence in alcohol-dependent patients with liver cirrhosis: randomised, double-blind controlled study. Lancet 2007; 370:1915.
17. Addolorato G, Caputo F, Capristo E, et al. Baclofen efficacy in reducing alcohol craving and intake: a preliminary double-blind randomized controlled study. Alcohol Alcohol 2002; 37:504.
18. Garbutt JC, Kampov-Polevoy AB, Gallop R, et al. Efficacy and safety of baclofen for alcohol dependence: a randomized, double-blind, placebo-controlled trial. Alcohol Clin Exp Res

2010; 34:1849.

19. Yamini D, Lee SH, Avanesyan A, et al. Utilization of baclofen in maintenance of alcohol abstinence in patients with alcohol dependence and alcoholic hepatitis with or without cirrhosis. Alcohol Alcohol 2014; 49:453.
20. Heydtmann M, Macdonald B, Lewsey J, et al. The GABA-B agonist baclofen improves alcohol consumption, psychometrics and may have an effect on hospital admission rates in patients with alcoholic liver disease. hepatology 2011; 56:1091a.
21. Malekzadeh R, Mohamadnejad M, Rakhshani N, et al. Reversibility of cirrhosis in chronic hepatitis B. Clin Gastroenterol Hepatol 2004; 2:344.
22. Llach J, Ginès P, Arroyo V, et al. Prognostic value of arterial pressure, endogenous vasoactive systems, and renal function in cirrhotic patients admitted to the hospital for the treatment of ascites. Gastroenterology 1988; 94:482.
23. Sersté T, Melot C, Francoz C, et al. Deleterious effects of beta-blockers on survival in patients with cirrhosis and refractory ascites. Hepatology 2010; 52:1017.
24. Boyer TD, Zia P, Reynolds TB. Effect of indomethacin and prostaglandin A1 on renal function and plasma renin activity in alcoholic liver disease. Gastroenterology 1979; 77:215.
25. Ge PS, Runyon BA. The changing role of beta-blocker therapy in patients with cirrhosis. J Hepatol 2014; 60:643.
26. Garcia-Tsao G, Abraldes JG, Berzigotti A, Bosch J. Portal hypertensive bleeding in cirrhosis: Risk stratification, diagnosis, and management: 2016 practice guidance by the American Association for the study of liver diseases. Hepatology 2017; 65:310.
27. de Franchis R, Baveno VI Faculty. Expanding consensus in portal hypertension: Report of the Baveno VI Consensus Workshop: Stratifying risk and individualizing care for portal hypertension. J Hepatol 2015; 63:743.
28. Peng J, Russell S, Shamseddeen, H . Nadir mean arterial pressure during the first day of admission to the intensive care unit (ICU) is associated with ICU mortality. Hepatology 2019; 70:659A.
29. Arroyo V, Ginés P, Rimola A, Gaya J. Renal function abnormalities, prostaglandins, and effects of nonsteroidal anti-inflammatory drugs in cirrhosis with ascites. An overview with emphasis on pathogenesis. Am J Med 1986; 81:104.
30. de Lédinghen V, Mannant PR, Foucher J, et al. Non-steroidal anti-inflammatory drugs and variceal bleeding: a case-control study. J Hepatol 1996; 24:570.
31. García-Pagán JC, Salmerón JM, Feu F, et al. Effects of low-sodium diet and spironolactone on portal pressure in patients with compensated cirrhosis. Hepatology 1994; 19:1095.

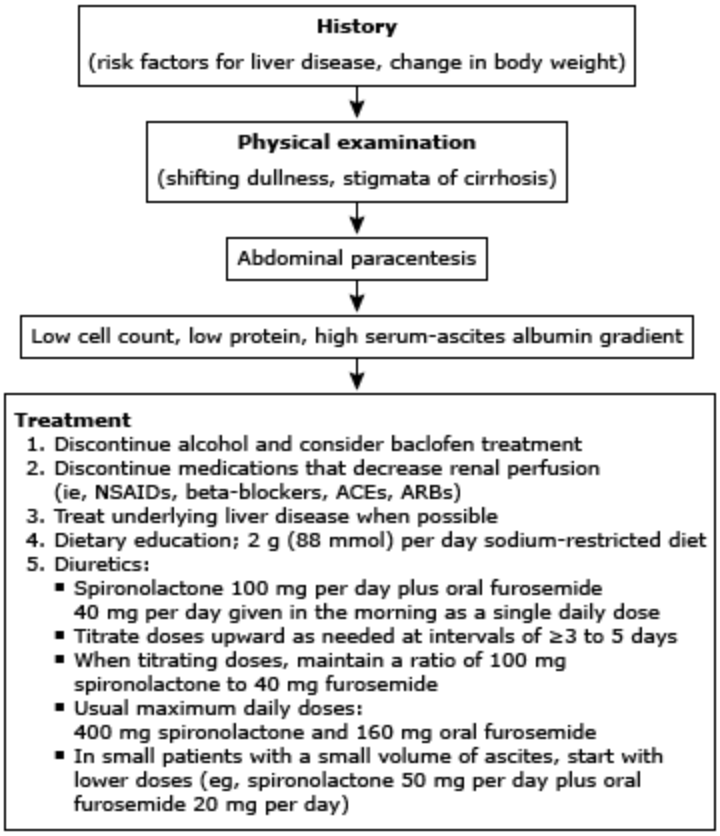
32. Wensing G, Lotterer E, Link I, et al. Urinary sodium balance in patients with cirrhosis: relationship to quantitative parameters of liver function. Hepatology 1997; 26:1149.
33. Arroyo V, Bosch J, Gaya-Beltrán J, et al. Plasma renin activity and urinary sodium excretion as prognostic indicators in nonazotemic cirrhosis with ascites. Ann Intern Med 1981; 94:198.
34. Angeli P, Wong F, Watson H, et al. Hyponatremia in cirrhosis: Results of a patient population survey. Hepatology 2006; 44:1535.
35. Fogel MR, Sawhney VK, Neal EA, et al. Diuresis in the ascitic patient: a randomized controlled trial of three regimens. J Clin Gastroenterol 1981; 3 Suppl 1:73.
36. Sawhney VK, Gregory PB, Swezey SE, Blaschke TF. Furosemide disposition in cirrhotic patients. Gastroenterology 1981; 81:1012.
37. Daskalopoulos G, Laffi G, Morgan T, et al. Immediate effects of furosemide on renal hemodynamics in chronic liver disease with ascites. Gastroenterology 1987; 92:1859.
38. Spahr L, Villeneuve JP, Tran HK, Pomier-Layrargues G. Furosemide-induced natriuresis as a test to identify cirrhotic patients with refractory ascites. Hepatology 2001; 33:28.
39. Angeli P, Fasolato S, Mazza E, et al. Combined versus sequential diuretic treatment of ascites in non-azotaemic patients with cirrhosis: results of an open randomised clinical trial. Gut 2010; 59:98.
40. Therapie innerer Krankheiten, 9th ed, Paumgartner G, Brandt T, Greten H, et al (Eds), Springer-Verlag, Berlin 2013.
41. Artz SA, Paes IC, Faloon WW. Hypokalemia-induced hepatic coma in cirrhosis. Occurrence despite neomycin therapy. Gastroenterology 1966; 51:1046.
42. Pérez-Ayuso RM, Arroyo V, Planas R, et al. Randomized comparative study of efficacy of furosemide versus spironolactone in nonazotemic cirrhosis with ascites. Relationship between the diuretic response and the activity of the renin-aldosterone system. Gastroenterology 1983; 84:961.
43. Pinzani M, Daskalopoulos G, Laffi G, et al. Altered furosemide pharmacokinetics in chronic alcoholic liver disease with ascites contributes to diuretic resistance. Gastroenterology 1987; 92:294.
44. Angeli P, Dalla Pria M, De Bei E, et al. Randomized clinical study of the efficacy of amiloride and potassium canrenoate in nonazotemic cirrhotic patients with ascites. Hepatology 1994; 19:72.
45. Dimitriadis G, Papadopoulos V, Mimidis K. Eplerenone reverses spironolactone-induced painful gynaecomastia in cirrhotics. Hepatol Int 2011; 5:738.

46. Pockros PJ, Reynolds TB. Rapid diuresis in patients with ascites from chronic liver disease: the importance of peripheral edema. Gastroenterology 1986; 90:1827.
47. Boyer TD. Removal of ascites: what's the rush? Gastroenterology 1986; 90:2022.
48. Hernandez AF, Greiner MA, Fonarow GC, et al. Relationship between early physician follow-up and 30-day readmission among Medicare beneficiaries hospitalized for heart failure. JAMA 2010; 303:1716.
49. Runyon BA. Treatment of patients with cirrhosis and ascites. Semin Liver Dis 1997; 17:249.
50. Stanley MM, Ochi S, Lee KK, et al. Peritoneovenous shunting as compared with medical treatment in patients with alcoholic cirrhosis and massive ascites. Veterans Administration Cooperative Study on Treatment of Alcoholic Cirrhosis with Ascites. N Engl J Med 1989; 321:1632.
51. Peltekian KM, Wong F, Liu PP, et al. Cardiovascular, renal, and neurohumoral responses to single large-volume paracentesis in patients with cirrhosis and diuretic-resistant ascites. Am J Gastroenterol 1997; 92:394.
52. Bernardi M, Caraceni P, Navickis RJ. Does the evidence support a survival benefit of albumin infusion in patients with cirrhosis undergoing large-volume paracentesis? Expert Rev Gastroenterol Hepatol 2017; 11:191.
53. Bernardi M, Caraceni P, Navickis RJ, Wilkes MM. Albumin infusion in patients undergoing large-volume paracentesis: a meta-analysis of randomized trials. Hepatology 2012; 55:1172.
54. Caraceni P, Riggio O, Angeli P, et al. Long-term albumin administration in decompensated cirrhosis (ANSWER): an open-label randomised trial. Lancet 2018; 391:2417.
55. Romanelli RG, La Villa G, Barletta G, et al. Long-term albumin infusion improves survival in patients with cirrhosis and ascites: an unblinded randomized trial. World J Gastroenterol 2006; 12:1403.
56. Montalvo-Gordon I, Chi-Cervera LA, García-Tsao G. Sodium-Glucose Cotransporter 2 Inhibitors Ameliorate Ascites and Peripheral Edema in Patients With Cirrhosis and Diabetes. Hepatology 2020; 72:1880.

Topic 1256 Version 42.0

GRAPHICS

Evaluation and initial therapy of a patient with cirrhosis and ascites



ACE: angiotensin-converting enzyme inhibitors; ARB: angiotensin II receptor blockers; NSAID: non-steroidal anti-inflammatory drug.

Reference:

1. *Runyon B. Management of adult patients with ascites due to cirrhosis: Update of 2012. AASLD Practice Guideline (February, 2013).*

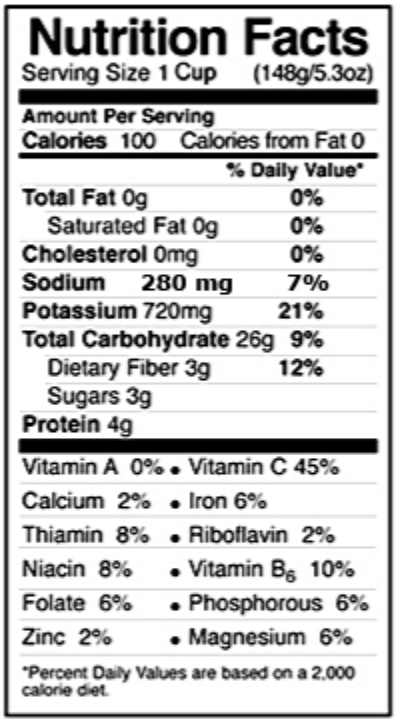
Graphic 77152 Version 6.0

Low-sodium diet

A low sodium diet is one that includes less than 2 grams (2000 milligrams) of sodium each day. The main source of sodium in the diet is table salt, which is often added when foods are processed, prepared, or just before eating. One teaspoon of salt contains about 2300 mg of sodium.

Sodium also occurs naturally in some foods. To determine the amount of sodium that a food contains, consult a reference book; one suggestion is Bobbie Mostyn's *Pocket Guide to Low Salt Foods*. Many websites also provide nutrient data (eg, www.nutrition.gov). Low sodium cookbooks are also available.

On food labels, the amount of sodium in food is listed on the nutrition label:



Nutrition Facts

Serving Size 1 Cup (148g/5.3oz)

Amount Per Serving	
Calories 100	Calories from Fat 0
	% Daily Value*
Total Fat 0g	**0%**
Saturated Fat 0g	**0%**
Cholesterol 0mg	**0%**
Sodium 280 mg	**7%**
Potassium 720mg	**21%**
Total Carbohydrate 26g	**9%**
Dietary Fiber 3g	**12%**
Sugars 3g	
Protein 4g	

Vitamin A 0%	• Vitamin C 45%
Calcium 2%	• Iron 6%
Thiamin 8%	• Riboflavin 2%
Niacin 8%	• Vitamin B_6 10%
Folate 6%	• Phosphorous 6%
Zinc 2%	• Magnesium 6%

*Percent Daily Values are based on a 2,000 calorie diet.

1 serving (1 cup) contains 280 mg of sodium

Decrease the amount of salt you eat gradually. Many people find that they do not miss salt after a few weeks of eating less.

Suggestions to decrease sodium include the following:

- Do not add salt to food while cooking or before eating. Teach family members to taste food before adding salt.
- Avoid eating at fast food restaurants. If this is not possible, choose restaurants that offer fruits or vegetables without sauces or dressings. Ask that no salt be used to prepare food, when possible.
- Do not use salt substitutes unless a healthcare provider approves. Herb and spice combinations that do not contain salt can be used to flavor foods.
- Water softeners remove calcium and add sodium to drinking water. Do not drink softened water. When purchasing bottled water, check the label to ensure that it does not contain sodium.
- Look at labels for over the counter medications. Avoid products that contain sodium carbonate and sodium bicarbonate. Sodium bicarbonate is baking soda.
- Look for food labels that say "no salt added", "low salt", or "low sodium". Item that are labeled low salt/sodium must have less than 140 mg of sodium per serving (check the serving size!)

Graphic 51602 Version 2.0

Low-sodium diet (continued)

Foods to choose	Foods to avoid
Breads:	
Whole-grain breads, English muffins, bagels	Biscuits, prepared mixes (pancake, muffin, cornbread)
Cereals:	
Cooked hot cereals (not instant), such as oatmeal, cream of wheat, rice, or farina; puffed wheat; puffed rice; shredded wheat	Instant hot cereals, many boxed cold cereals
Crackers and snack foods:	
All unsalted crackers and snack foods, unsalted peanut butter, unsalted nuts or seeds	Salted crackers and snack items (chips, pretzels, popcorn), regular peanut butter, prepared dips/spreads, salted nuts or seeds
Pasta, rice, and potatoes:	
Any type of pasta; potatoes; white or brown rice	Macaroni and cheese mix; rice, noodle, or spaghetti mixes; canned spaghetti; frozen lasagna; instant potatoes; seasoned potato mixes
Dried beans and peas:	
Any dried beans or peas without seasoning	Beans or peas prepared with ham, bacon, salt pork, or bacon grease; all canned beans
Meats and alternatives:	
Fresh or frozen beef, poultry, and fish; low-sodium canned tuna and salmon; eggs	Salted, smoked, canned, spiced, and cured meat, poultry, or fish; bacon; ham; sausage; lunch meats; hot dogs; breaded frozen meat, fish, or poultry; frozen dinners; pizza
Fruits and vegetables:	
Any fresh, frozen, or canned fruit, any fresh or frozen vegetables without sauce, canned vegetables without salt, low-salt tomato sauce/paste	Regular canned vegetables and vegetable juices, regular tomato sauce and tomato paste, olives, pickles, relishes, sauerkraut, frozen vegetables in butter or sauces, crystallized and glazed fruit, maraschino cherries, fruit dried with sodium sulfite
Dairy products:	
Milk, cream, sour cream, non-dairy creamer, yogurt, low-	Buttermilk, Dutch processed chocolate milk, processed cheese slices and spreads, processed cheese, cottage cheese, aged or natural cheese

sodium cottage cheese, low-sodium cheese	
Fats and oils:	
Plant oils (olive, canola, corn, peanut), unsalted butter or margarine	Prepared salad dressings, bacon, salt pork, fat back, salted butter or margarine
Soups:	
Salt-free soups and low-sodium bouillon cubes, unsalted broth, homemade soup without added salt	Regular canned or prepared soups, stews, broths, or bouillon; packaged and frozen soups
Desserts:	
Gelatin, sherbet, pudding, ice cream, salt-free baked goods, sugar, honey, jam, jelly, marmalade, syrup	Packaged baked goods
Beverages:	
Coffee, tea, soft drinks, fruit-flavored drinks, low-salt tomato juice, any fruit juice	Softened water; carbonated beverages with sodium or salt added; regular tomato juice (V-8); ask about alcoholic beverages
Condiments:	
Fresh and dried herbs; lemon juice; low-salt mustard, vinegar, Tabasco sauce; low- or no-salt ketchup; seasoning blends that do not contain salt	Table salt, lite salt, bouillon cubes, meat extract, taco seasoning, Worcestershire sauce, tartar sauce, ketchup, chili sauce, cooking sherry and wine, onion salt, mustard, garlic salt, soy sauce, tamari, meat flavoring or tenderizer, steak and barbecue sauce, seasoned salt, monosodium glutamate (MSG), Dutch processed cocoa

Graphic 63387 Version 1.0

Contributor Disclosures

Bruce A Runyon, MD, FAASLD No relevant financial relationship(s) with ineligible companies to disclose. **Keith D Lindor, MD** Consultant/Advisory Boards: Pliant [DSMB member]. All of the relevant financial relationships listed have been mitigated. **Kristen M Robson, MD, MBA, FACG** No relevant financial relationship(s) with ineligible companies to disclose.

Contributor disclosures are reviewed for conflicts of interest by the editorial group. When found, these are addressed by vetting through a multi-level review process, and through requirements for references to be provided to support the content. Appropriately referenced content is required of all authors and must conform to UpToDate standards of evidence.

Conflict of interest policy

→