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“Pleurex Desalination” in Malignancy-Related Ascites: A Novel Mechanism of Hyponatremia

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Abstract

Objective: Pleurex catheters are a form of palliative therapy for patients, offering relief from symptomatic ascites while also affording greater independence and flexibility; however, aggressive drainage can lead to significant total body sodium losses. We describe the course of patients with “Pleurex desalination,” an under-appreciated cause of hypovolemic hyponatremia, highlighting its unique pathophysiology and providing recommendations on how to manage these complex patients.

Patients and Methods: We included representative patients with “Pleurex desalination” who were evaluated and treated by the renal consult service at Brigham and Women’s between 2017 and 2019.

Results: We identified 3 patients who were hospitalized with “Pleurex desalination” and had complete data on serum and urine studies, as well as treatment course. We demonstrate that patients with “Pleurex desalination” were removing up to 1 to 2 liters of ascitic fluid a day and were admitted with signs and symptoms of profound hypovolemia and hyponatremia. Patients worsened with administration of diuretics and salt restriction and improved with aggressive fluid resuscitation in the form of hypertonic saline, normal saline, and/or intravenous albumin.

Conclusion: “Pleurex desalination” is an under-recognized cause of hyponatremia; at-risk patients require close observation and periodic resuscitation with intravenous, volume-expanding fluids.

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Keywords

Pleurex catheter; hyponatremia; ascites; malignancy; nephrology

Introduction:

Malignancy-related ascites is a debilitating condition occurring in the setting of terminal malignancies. Ascites has traditionally been managed with diuretics and repeated paracentesis, and now more recently by intermittent percutaneous drainage via Pleurex catheter.^{1–3} Pleurex catheters are a form of palliative therapy for patients, offering relief from symptomatic ascites while also affording greater independence and. However, aggressive drainage can lead to significant total body sodium losses of up to 140 to 280 milliequivalents (mEq) in patients draining 1 to 2 liters (L) of ascites per day, leading to salt and volume depletion. Without repletion of these losses, patients are at risk for a form of hypovolemic hyponatremia, which we have termed “Pleurex desalination.”

“Pleurex desalination” differs from other well-known forms of hypovolemic hyponatremia secondary to vomiting, diarrhea, or diuretic therapy, because it is primarily driven by chronic salt depletion rather than transient volume loss with free water excess.^{4,5} Salt losses can be massive, because the ascitic fluid is often isotonic to serum. Furthermore, patients often cannot increase their salt intake commensurate to their cumulative or ongoing losses.

At Brigham and Women’s Hospital/Dana-Farber Cancer Center, we identified three patients who had Pleurex catheters placed for malignancy-related ascites and were evaluated by the renal consult service between 2017 and 2019 for new or worsening hyponatremia after catheter placement. We describe each patient’s baseline characteristics and treatment course (Table 1, Figure 1); the unique pathophysiology of “Pleurex desalination” compared with other causes of hypovolemic hyponatremia; and recommendations on how to manage these complex patients.

Patient 1:

A 39-year-old male with a history of peritoneal mesothelioma had a Pleurex catheter in place for malignancy-related ascites for approximately 2 years. His sodium pre-Pleurex was 135 mEq/L, and serum albumin was 3.2 grams/deciliter (g/dL). In the first few months after Pleurex placement, his sodium remained between 133 and 135 mEq/L. Approximately 6 months after Pleurex placement, he was prescribed furosemide 40 milligrams (mg) along with spironolactone 100 mg sodium for intractable ascites. He was continuing to drain approximately 2 L every day from his catheter. His sodium began to fall over the course of several weeks to 124 to 126 mEq/L. He was asymptomatic, specifically denying loss of appetite, uncontrolled pain, nausea, vomiting, or diarrhea. He also reported eating a jar of pickles each day. His outpatient oncologist obtained urine studies, which showed a urine osmolality 578 milliosmoles/kilogram (mOsm/kg) and urine sodium <20 meQ/L. He began receiving intravenous 25% albumin infusions bi-weekly, and was prescribed salt tablets 2 grams (g) three times a day for hyponatremia.

The patient developed nausea and vomiting, which he attributed to his salt tablets. He discontinued these, and was also no longer able to consume pickles every day. The patient subsequently was admitted to the hospital after falling on his left hip, and was found to have a serum sodium of 113 milliequivalents/liter (mEq/L). He was confused and had dry mucous membranes and bilateral lower extremity edema on physical examination. Jugular venous distension (JVD) was notably absent. In addition to the severe hyponatremia, his labs were notable for potassium 5.1 mEq/L, chloride 80 mEq/L, bicarbonate 22 mEq/L, blood urea nitrogen (BUN) 10 mg/dL, serum creatinine (SCr) 0.48 mg/dL, glucose 100 mg/dL, and anion gap of 11 mEq/L. Serum osmolality was 243 mOsm/kg. Urine sodium prior to treatment was <20 mEq/L, and urine osmolality was 413 mOsm/kg. Liver enzyme tests were normal. His serum albumin was 1.9 g/dL.

Concerned about both hypovolemic hyponatremia and possible syndrome of antidiuretic hormone (SIADH), the inpatient renal consult team recommended treatment with one dose of intravenous desmopressin (ddAVP) 2 micrograms (mcg) and initiation of 3% hypertonic saline at 30 milliliters/hour (mL/hr). Further Pleurex drainage was held. Repeat urine studies showed urine Na <20 mEq/L and urine osmolality 438 mEq/L. His sodium improved from 113 to 118 mEq/L over the course of 48 hours. Hypertonic saline was discontinued due to improvements in his mental status. His treatment was transitioned to a combination of fluid restriction to <2L/day, 2 g salt tablets three times a day, intravenous albumin (50 g of 25%), and liberal oral salt (pickle) intake. His sodium subsequently improved to 135 mEq/L on day 7, chloride normalized to 98 mEq/L, and potassium to 4.0 mEq/L.

Unfortunately, his hospital course was later complicated by septic shock and acute kidney injury requiring continuous veno-venous hemofiltration ultimately leading to his death on day 25 of hospitalization.

Patient 2:

A 38-year-old female with breast adenocarcinoma, with known metastases to the liver and subsequent cirrhosis, had a Pleurex placed for recurrent malignant ascites. Prior to Pleurex catheter placement, her sodium was normal at 137 mEq/L, and her serum albumin was 2.3 g/dL. She was draining 1 to 2 L from her Pleurex daily. Three weeks after placement, she was noted to have a sodium of 119 mEq/L, with normal renal function. She was admitted for symptomatic hyponatremia, with fatigue and lethargy. She was treated with salt tablets and a 1 L fluid restriction, and she was discharged after a 3-day hospitalization with a sodium of 127 mEq/L. During a follow-up appointment one week later, her salt tablets were discontinued due to concerns that treatment was worsening her ascites. At that appointment, her sodium was 129 mEq/L. She was started on diuretics and advised to restrict her salt and water intake.

One week later, she was admitted to the hospital with lethargy, nausea, and lightheadedness and was found to have a serum sodium of 114 mEq/L. Other labwork was notable for potassium 5.9 mEq/L, chloride 83 mEq/L, bicarbonate 20 mEq/L, BUN 22 mg/dL, SCr 0.63 mg/dL (baseline), glucose 120 mg/dL, and anion gap 7 mEq/L. Serum osmolality was 251 mOsm/kg. Her urine sodium was <20 mEq/L and her urine osmolality was 979 mOsm/kg.

Thyroid function and a cortisol stimulation test were normal. Her serum albumin had decreased to 1.5 g/dL. In the emergency room, she was tachycardic with dry mucous membranes, and without JVD. She was seen by nephrology and started on hypertonic saline at 30–45 mL/hr for 10 hours. Her sodium increased to 120 mEq/L. On day 2 of her hospital stay, she was transitioned to isotonic 0.9% saline at 150 mL/h and 2 g salt tablets three times a day due to concerns of ongoing intravascular depletion. Ascitic fluid electrolytes checked that day showed that the ascites was isotonic to serum (sodium 122 mEq/L, potassium 5.2 mEq/L, and chloride 97 mEq/L).

On days 3 and 4 of her hospitalization, her sodium improved to 122–123 mEq/L. Her intravenous fluids were discontinued, but she had significant abdominal distension, requiring 4.5 L of drainage from her Pleurex. Her salt tablets were increased to 4 grams three times a day, and she was discharged on day 5 with a sodium of 122 mEq/L.

The patient was then admitted 12 days later with hematemesis from an upper gastrointestinal bleed, along with confusion and lethargy. In the days preceding this admission, she was adherent to the salt tablets as prescribed, but had increased her daily Pleurex drainage from 2 to 5 L. Her labs were notable for sodium of 110 mEq/L, potassium of 7.7 mEq/L, and SCr of 1.2 mg/dL (up from baseline of 0.6 mg/dL). Her urine sodium was <20 mEq/L and her urine osmolality was 806 mOsm/kg. She received 4 L of 0.9% normal saline, followed by 1 unit of packed red blood cells, 1 unit of fresh frozen plasma, and hypertonic saline at 30 mL/hr.

With these treatments, her sodium improved to 116 mEq/L by day 2. She was continued on hypertonic saline at 30 mL/hr and also received 1 bottle of 25% albumin. Her ascitic fluid drainage was reduced to 2 L each day. By day 3, her sodium had improved to 122 mEq/L leading to discontinuation of her hypertonic saline. A peripherally inserted central catheter (PICC) was placed for infusion of 1 L of 0.9% normal saline daily. She was discharged home on hospice with a serum sodium ranging from 122–123 mEq/L and died 10 days later.

Patient 3

A 58-year-old female with metastatic renal cell cancer had a Pleurex in place for 1 month for progressively worsening ascites. Her catheter was draining 500 mL to 1 L every other day. Her serum sodium was 125–131 mEq/L prior to Pleurex placement. Her baseline SCr was 0.9 mg/dL after a radical nephrectomy early in the course of her disease.

Ten days after Pleurex placement, she was admitted to the hospital with dyspnea and hyponatremia, with a sodium of 126 mEq/L. Serum osmolality was 261 mOsm/kg. Urine studies were notable for a urine sodium <20 mEq/L and urine osmolality of 581 mOsm/kg. The nephrology consult service felt that her presentation was most consistent with hypovolemic hyponatremia along with SIADH. She was managed with intravenous furosemide and albumin, as well as drainage of her Pleurex of 1 L every other day. She notably also had acute kidney injury, with an elevated SCr up to 1.31 mg/dL up from a baseline of 0.9 mg/dL. On discharge, her sodium had improved to 131 mEq/L and her SCr to 0.97 mg/dL; she was instructed to take furosemide 40 mg daily and to continue drainage of 1 to 2 L every other day.

The patient was then readmitted 8 days later with fatigue and weakness, and a sodium of 123 mEq/L. On exam, she was hypotensive to 86/57, cachectic with dry mucous membranes, reduced breath sounds at bilateral bases, abdominal distension, pitting edema. JVD was not documented. Other labs were notable for a hemolyzed potassium level, serum chloride 99 mEq/L, HCO₃ 22 mEq/L, and BUN 29 mg/dL. Her urine sodium was <20 mEq/L and urine osmolality was 711 mOsm/kg. Her liver function tests and morning cortisol were normal. Thyroid function tests were notable for a thyroid stimulating hormone of 15 IU/mL, and a free T4 level of 0.8 ng/dL. Her albumin was notably decreased to 1.8 g/dL from 2.8 g/dL on prior admission.

The patient was felt to have hypovolemic hyponatremia with salt wasting from her Pleurex. She was administered 500 mL of 0.9% normal saline, with repeat labs 5 hours later demonstrating a sodium of 125 mEq/L. However, she had also concurrently drained 2 L from her Pleurex and her sodium decreased to 123 mEq/L. She was started on normal saline 100 mL/hr to match output as well as salt tablets 1 grams three times a day, with improvement in her sodium back to 125 mEq/L. She was subsequently transitioned to hospice, with a plan for her to have regular normal saline repletion (1.5 L 0.9% normal saline bi-weekly) as an outpatient. Her salt tablets were discontinued.

Discussion

In this case series, we describe a novel form of hypovolemic hyponatremia, “Pleurex desalination,” occurring secondary to aggressive removal of ascitic fluid without replacement of resultant salt losses. Because the sodium content in ascitic fluid is similar to that of plasma,⁶ removal of 1 L of ascites can lead to significant salt depletion, not accounting for additional losses that may occur in sweat, stool, and urine. The pathophysiology of “Pleurex desalination” is unique, and can be distinguished from traditional causes of hypovolemic hyponatremia based on the profound salt wasting that occurs through Pleurex drainage. We demonstrate that aggressive drainage of ascitic fluid can cause significant hypovolemic hyponatremia if losses are not replaced with regular infusions of isotonic fluid or albumin. Furthermore, in our series, “Pleurex desalination” preceded death by only a few days to weeks, adding an additional burden to end-of-life care in these patients.

The human body is able to maintain serum sodium concentration within a narrow range. For instance, in a 70 kg individual, total body sodium (TBNa) levels approximate 4200 mEq, or 60 mEq/kg.^{7,8} Roughly 1800 mEq is contained in bone, and of the remaining 2400 mEq, only about 300 mEq is present in the intracellular fluid. The majority (2100 mEq) is contained in the extracellular fluid (ECF). Sodium is the primary determinant of ECF osmolality, and in most instances, hyponatremia indicates hypotonicity. Etiologies of hypotonic hyponatremia are then further differentiated based on volume status (hypovolemic, euvolemic, or hypervolemic), as well as urine osmolality and urine sodium level.^{9–11} Hypovolemic hyponatremia is characterized by high levels of circulating arginine vasopressin, low urine sodium, and high urine osmolality, and is primarily driven by *water retention* in association with mild sodium depletion (as in the case of diarrhea).⁹ As a result,

hyponatremia under these circumstances usually develops when intake of water exceeds the pace of excretion, rather than secondary to sodium losses (Figure 2).

In the case of “Pleurex desalination,” patients can lose up to 240–260 mEq of sodium per day via their catheter, which amounts to 5% of TBNa and ~10% of ECF sodium. All three of our patients were draining almost 2 L of ascitic fluid each day, equating to a deficit of up to 280 mEq of sodium per day. In cancer patients with nausea, vomiting, and decreased oral intake, it can be increasingly difficult to keep up with these losses, and a negative sodium balance ensues over time. Several patients in our series were prescribed salt tablets; however, these alone will not adequately replete losses, as a 1 g tablet of sodium chloride contains only 17 mEq of sodium. Furthermore, salt tablets often result in increased thirst and water intake, which can then lead to worsening hyponatremia.¹² Patient 1 had been consuming pickles and salt tablets; however, his hyponatremia worsened when he developed nausea and discontinued both, while continuing daily Pleurex drainage. All three patients had a low serum osmolality, low urine sodium, high urine osmolality, along with physical exam findings suggestive of hypovolemia.

Patients with Pleurex catheters often have concomitant liver failure, either due to metastases resulting in portal hypertension, or from direct parenchymal invasion by the primary tumor.¹³ Patient 2 had cirrhosis secondary to metastases to the liver. Treatment with furosemide, along with salt and fluid restriction, resulted in lower sodium levels. These findings suggest that the patient’s hyponatremia was not secondary to hypervolemic hyponatremia as is usually the case in patients with advanced liver disease, who develop hyponatremia due to disproportionate retention of water (Figure 2).^{14,15} Thus, while the use of loop diuretics and potassium-sparing diuretics can be effective in relieving fluid accumulation in patients with hypervolemic hyponatremia from liver failure,¹⁶ it can worsen hyponatremia in patients with malignancy-related ascites.¹⁷ It can often be difficult to distinguish hypervolemic hyponatremia secondary to cirrhosis from “Pleurex desalination” because both states are characterized by a lower serum osmolality, high urine osmolality, and low urine sodium.⁴ Furthermore, patients with malignancy-related ascites will often have lymphedema,^{18,19} thereby complicating assessment of volume status.

Aside from liver failure, hyponatremia can develop in cancer patients for a host of other reasons, and can be exceedingly difficult to manage.²⁰ Patients may have SIADH, extracellular fluid depletion, and increased hypotonic fluid intake. Patients 1 and 3 had pre-existing mild hyponatremia, which may have been secondary to SIADH from their underlying malignancies. Several patients were also concurrently prescribed opioids, selective serotonin reuptake inhibitors, and/or non-steroidal anti-inflammatory drugs, all of which have been associated with SIADH.^{20–22} However, patients 2 and 3 had a decline in their sodium within days to weeks of Pleurex placement, which is unlikely to occur due to SIADH alone.

Few studies have examined the effects of Pleurex catheters on serum sodium level. Reinglas et al. retrospectively examined the safety and effectiveness of Pleurex catheter placement for palliative management of refractory ascites, secondary to cirrhosis of the liver.²³ Among the 33 patients who underwent Pleurex placement, 20 were hyponatremic (<135 mmol/L) at

baseline. From time of insertion to first follow-up, patients had lower serum sodium values, by 2 mmol/L (SD, 4 mmol/L) on average. In contrast, in a multicenter, prospective study of Pleurex catheters implanted for malignancy-related ascites (n=34), there was no significant difference in blood chemistry results between baseline and 12 weeks, though 26 of the 34 patients died (76.4%) before the 12 week end-point, limiting the interpretation of these data.²⁴

In our series, “Pleurex desalination” responded well to resuscitation with intravenous fluid. Patients 1 and 2 were treated with hypertonic saline, as they presented with severe hyponatremia (sodium levels <120 mEq/L). Hypertonic saline, which contains 513 mEq/L of sodium, is commonly accepted as the treatment of choice in patients with acute and symptomatic hyponatremia.^{4,25} Patients 2 and 3 responded well to normal saline, which can help restore extracellular volume by replacing both salt and water losses.^{11,25} Intravenous 25% albumin, a sterile aqueous solution that is hyperoncotic with respect to plasma, was also successfully used in patients. All commercial albumin products contain sodium ranging from 130 to 160 mEq/L.²⁶ The 25% solution contains 154 mEq of sodium per L and may be useful for both volume expansion and diminishing ascites and tissue edema.^{26,27} While hypoalbuminemia is common among cancer patients for a variety of reasons, including inflammation, malnutrition, and reduced hepatic synthesis,^{29,30} all 4 patients had a progressive decline in their serum albumin levels down to <2 g/dL after Pleurex placement. Albumin infusion prior to large volume paracenteses in patients with liver failure has been shown to decrease the risk of circulatory dysfunction, hyponatremia, and even mortality;^{31,32} larger studies are needed in order to determine whether patients with malignant ascites derive the same benefit.

Based on our study and clinical experience, patients should be counseled prior to Pleurex placement about the symptoms of hyponatremia, as well as the need for close monitoring of labs and regular infusions of fluid. We recommend that if lab draws are within a patient’s goals of care, a basic metabolic panel should be checked bi-weekly after Pleurex placement. Patients should be carefully instructed about the maximum amount of fluid that should be removed at a time. If a patient develops hyponatremia with a sodium level <135 mEq/L, then serum osmolality, urine osmolality, and urine sodium levels should be obtained, and monitored regularly with any change in therapy. Patients should be assessed for thyroid dysfunction and adrenal insufficiency. Nephrology consultation should be considered, given the severity and complicated pathophysiology of hyponatremia in these patients.

With regards to treatment, patients with suspected hypovolemic hyponatremia due to “Pleurex desalination” should be treated with regular infusions of normal saline, to match output of the Pleurex catheter. Free water restriction may be necessary, but diuretics should be avoided. Patients should also be evaluated by dietitians to maximize oral sodium intake. Alternatively, total parenteral nutrition may be considered (sodium content 140 to 154 mEq/L).³³ Notably, many patients with Pleurex catheters may also have a tunneled central line, PICC, or Port-a-cath, which should be utilized for intravenous fluid replacement at home.

In conclusion, “Pleurex desalination” is a previously undescribed and under-appreciated form of iatrogenic hyponatremia that can occur in patients with Pleurex catheters for malignant ascites. Though often a palliative procedure, patients with Pleurex catheters may require close monitoring of their serum electrolytes, as well as regular infusions of intravenous isotonic or hypertonic fluid to counteract excessive sodium losses. Larger epidemiologic studies are needed to characterize the true incidence of hyponatremia from “Pleurex desalination,” and its association with outcomes in these patients.

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Patient 1: Sodium Trend During Hospitalization

Patient 2: Sodium Trend Post-Pleurex

Figure 1.

↓ Hypertonic Saline ↓ albumin + salt tablets ↓ normal saline

Abbreviations: mg/dL- milligrams/deciliter

Figure 2.

Hypovolemic hyponatremia from diarrhea and vomiting is usually characterized by small amounts of salt losses coupled with increased free water intake. There is overlap with "Pleurex desalination" in that patients may present with signs of volume depletion, including tachycardia, hypotension, and dry mucous membranes on exam. However, the pathophysiology of "Pleurex desalination" is unique because patients have profound salt depletion from massive losses of sodium through their catheter. They often have nausea and vomiting, which limits their ability to increase their salt intake commensurate to their losses. Patients require regular infusions of isotonic, volume-expanding fluid. In the case of hypervolemic hyponatremia from cirrhosis, patients have portal hypertension and a hyperdynamic circulation, promoting water retention. Unlike "Pleurex desalination," the cornerstone of management is removal of ascites at regular intervals, diuretics, and salt and water restriction. Patients with cirrhosis and those with "Pleurex desalination" may both have peripheral edema on exam and low serum albumin levels, which can make it difficult to distinguish between them. All three causes of hypotonic hyponatremia pictured here are associated with an increase in serum AVP levels, low serum osmolality, high urine osmolality, and low urine sodium levels.

Abbreviations: HTN-hypertension; Tx-treatment; AVP-arginine vasopressin; osm-osmolality; Na-sodium; NS-normal saline

Table 1:

Baseline Characteristics

Patient	Age at Pleurex Placement	Sex	Race	Malignancy	Days with Pleurex	Liver Disease
1	35	M	White	Mesothelioma	921	No
2	38	F	White	Breast	72	Yes
3	58	F	White	Renal Cell	57	No

Abbreviations: M-male; F-female