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LargePleuralEffusionAfterTransjugularIntrahepatic Portosystemic Shunt,a Rare but Deadly Complication

Kushang Shah [1] , Michael X. Jin [2] , Rachel Bright [2]

1. Internal Medicine, Stony Brook University Hospital, Stony Brook, USA 2. Radiology, Stony Brook University Hospital, Stony Brook, USA

Corresponding author: Michael X. Jin, drmichaelxjin@gmail.com

Abstract

Cirrhosis affectsmore than630,000 adultsglobally and can lead to development of ascites. Transjugular intrahepatic portosystemic shunt (TIPS) is an alternative option forrefractory ascites in patients who are ineligible or are waiting for liver transplants. However, this procedure can have serious complications. We present a case that highlights the development ofacomplex pleural effusion complicated by hemorrhagic shockanddisseminated intravascular coagulationafter TIPS in a 54-year-old man. Our case is the first to report such a complication and aims to provide awareness.

Categories: Internal Medicine, Radiology, Gastroenterology
Keywords: transjugular intrahepatic portosystemic shunt, ascites, pleural effusion, hemorrhagic shock, disseminated intravascular coagulation (dic)

Introduction

Cirrhosis, also known as end-stage liver disease, affectsmore than630,000 adultsglobally [1].It results from scarring caused by chronic liver damage leading to permanent loss of liver function. As the liver loses function and scars, portal venous pressure is increased because blood cannot as easily transverse the liver parenchyma. This results in transudate accumulating within the peritoneal cavity [2]. As more fluid accumulates within the peritoneal cavity, patients may developspontaneous bacterial peritonitis, a condition witha17-32% mortality rate [2,3].

Definitive treatmentfor cirrhosis-induced ascitesis liver transplantation. However, only approximately half of all eligiblepatients onlivertransplantwaitlistslaterreceivea transplant due to the low supply of donor livers [4]. Transjugularintrahepatic portosystemic shunt (TIPS) is an alternative option forrefractory ascites in such patients.TIPS is an artificial tract created under X-ray imaging within the liver to bypass the scarred liver tissue, thereby reducing the amount of back pressure and resulting ascites. However, TIPS can be associated with a variety of complications including bleeding, infection, and hepatic encephalopathy [4]. Other complications can be rare but still occur.Our casedescribes one such complication in a patient who underwentTIPS and subsequently developed pleural effusions.

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Case Presentation

A54-year-oldman withahistory ofintravenous(IV)drugand alcohol abuse,hepatitis C, decompensated cirrhosiscomplicated byascitesandportosystemic encephalopathy,andfactor XI deficiency presentedtothe emergency departmentwith two days of difficulty breathingfromascites-induced abdominal distention.The patienthad received nine therapeutic paracenteses withinthepastthree monthsdue tosymptomatic ascites.On this admission, the patient's vital signs were stable.He denied fever, chills, chest pain, nausea, vomiting, diarrhea, and constipation. Physical examination revealed moderate ascites and anasarca; minimal encephalopathy was noted per West Haven criteria [5]. Chemistry and X-ray showed elevated liver enzymes and mild pulmonary vascular congestion (Table *1*). Therapeutic paracentesis drained 3.7 L of fluid negative for spontaneous bacterial paracentesis and malignancy.

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Chemistry	
Sodium	134 mmol/L
Potassium	3.7 mmol/L
Chloride	106 mmol/L
Bicarbonate	19 mmol/L
Glucose Level	97 mg/dL
BUN	40 mg/dL
Creatinine	1.03 mg/dL
Anion Gap	9 mmol/L
Calcium	8.3 mg/dL
Phosphorus	2.8 mg/dL
Magnesium	2.2 mg/dL
Bilirubin, Total	2.1 mg/dL
Bilirubin, Direct	1.0 mg/dL
ALT (SGPT)	64 IU/L
AST (SGOT)	108 IU/L
Alkaline Phosphatase	257 IU/L
Albumin	3.0 g/dL
Total Protein	6.5 g/dL
Lipase Level	92 IU/L
Ntpro B-Type Natriuretic Peptide	215 pg/mL
Ammonia	66 umol/L
Lactic Acid	0.6 mmol/L
Blood Count	
WBC Count	5.24 k/uL
RBC Count	2.37 M/uL
Hemoglobin	8.7 g/dL (9.1 g/dL previous hospitalization)
MCV	108.0 fL
RDW	16.7%
Platelets	53 k/uL (44 k/uL previous hospitalization)
INR	1.4
PTT	47.8

TABLE 1: Patient's blood count and chemistry on admission.

BUN, blood urea nitrogen; ALT, alanine aminotransferase; SGPT, serum glutamic-pyruvic transaminase; AST, aspartate aminotransferase; SGOT, glutamic-oxalacetic transaminase; WBC, white blood cells; RBC, red blood cells; MCV, mean corpuscular volume; RDW, red cell distribution width; INR, international normalized ratio; PTT, partial thromboplastin time.

Ourpatient was nota candidate forliver transplantduetoactiveIV druguse, so he elected to proceed with TIPS(Figures *1*,*2*).During the procedure, afirm, cirrhotic liver was noted, causing difficulties for anastomosiscreationbetween right hepatic and portal veins. Anastomosis was eventually createdwitha 2

cm/6 cmViatorr10 mm TIPS stent and a 10 mm x 59 mm covered Viabahn extension, resulting in aportal venous gradientdecreasefrom 11 to 1 mmHgwithminimalblood loss.

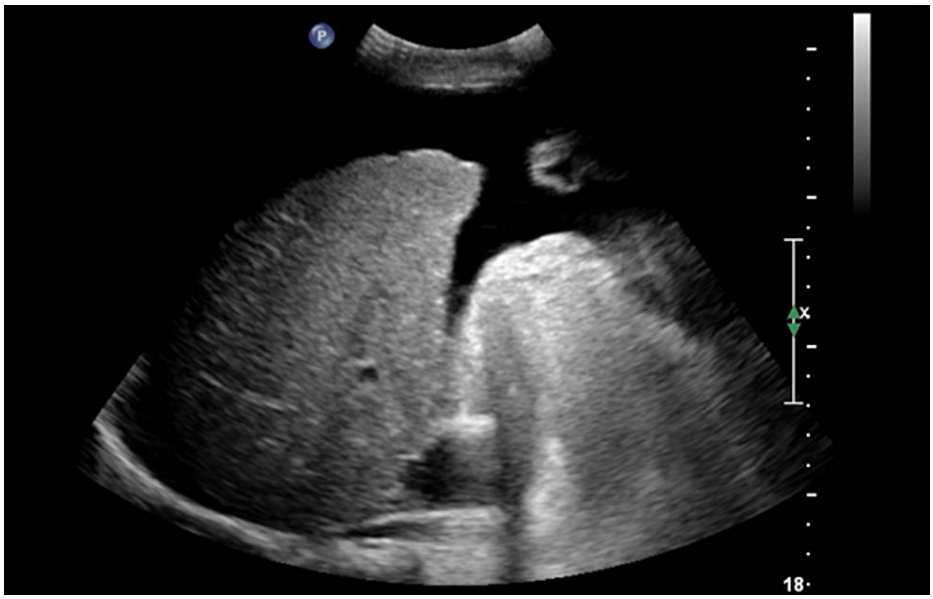

FIGURE 1: Ultrasound of right upper abdominal quadrant beforetransjugularintrahepatic portosystemic shuntdemonstrating mild ascites.

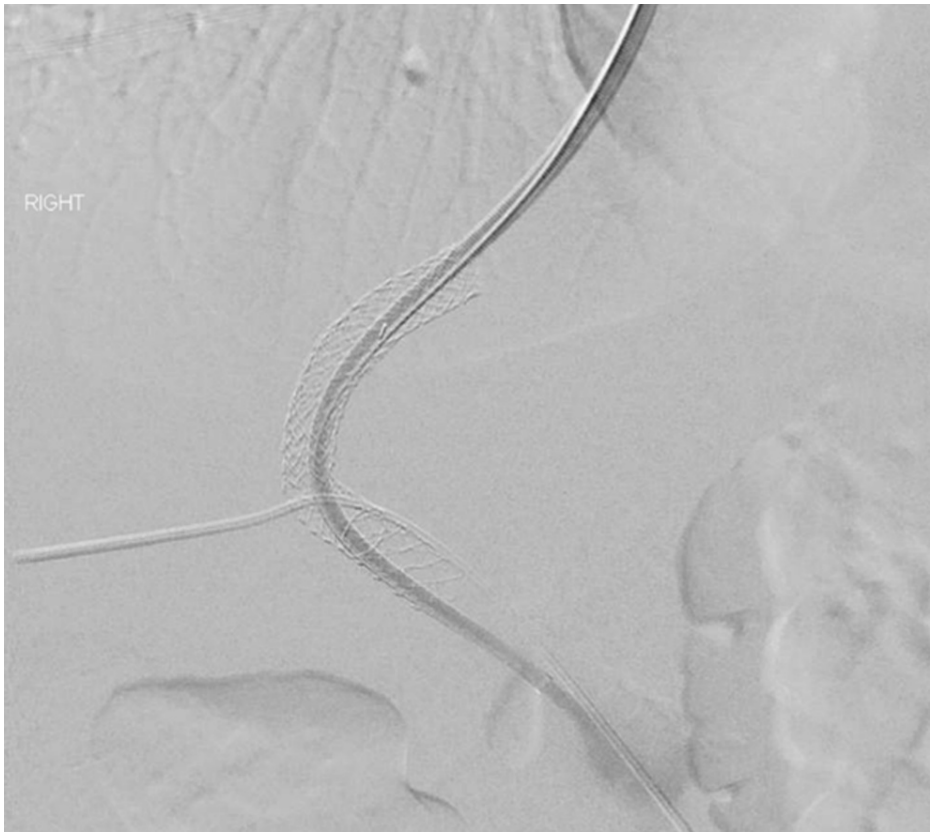

FIGURE 2: Digital subtraction angiography of the portal vein duringtransjugularintrahepatic portosystemic shunt placement showing successful placement of stent for procedure.

Tenhoursafter procedure,thepatient developed confusion. Lab work revealed a glucose level of 50 mg/dL,hemoglobinof6.7g/dL (8.7 g/dL on admission), hyperkalemia, anion gap metabolic acidosis with respiratoryalkalosis, and disseminated intravascular coagulation (DIC) (Table2).The patientwas given sodium polystyrene sulfonate, calcium gluconate, albuterol, dextrose with insulin, andone unitofpacked red blood cell (pRBC).His hemoglobin dropped to 6.1 g/dL 8 hours after transfusion.

Chemistry	
Sodium	135 mmol/L
Potassium	5.5 mmol/L
Chloride	109 mmol/L
Bicarbonate	17 mmol/L
Glucose Level	50 mg/dL
BUN	43 mg/dL
Creatinine	1.55 mg/dL
Anion Gap	9 mmol/L
Calcium	8.2 mg/dL
Phosphorus	4.5 mg/dL
Magnesium	2.0 mg/dL
Total Bilirubin	4.1 mg/dL
Direct Bilirubin	2.3 mg/dL
ALT	349 IU/dL
AST	953 IU/dL
LDH	742 IU/dL
Haptoglobin	10.5 mg/dL
Lipase	26 IU/L
Lactic Acid	8.0 mmol/L
Iron	102 ug/dL
TIBC	100 ug/dL
Ferritin	7877 ng/mL
B12	1773
Folate	>20
Ammonia	50 umol/mL
Troponin	0.8
Blood Count	
WBC Count	8.61 k/uL
RBC Count	1.89 M/uL
Hemoglobin	6.7 g/dL (6.1 g/dL after transfusion)
MCV	106.9 fL
RDW	19.1%
Platelet Count	74 k/uL
INR	2.1

aPTT	50.3 seconds
Arterial Blood Gas	
pH	7.29
pCO_2	27
pO_2	91
HCO_3	12

TABLE 2: Labs upon arrival to the intensive care unit.

BUN, blood urea nitrogen; ALT, alanine aminotransferase; AST, aspartate aminotransferase; LDH, lactate dehydrogenase; TIBC, total iron-binding capacity; WBC, white blood cells; RBC, red blood cells; MCV, mean corpuscular volume; RDW, red cell distribution width; INR, international normalized ratio; aPTT, activated partial thromboplastin time.

Due to deteriorating clinical status, he was transferred to the medical intensive care unit (MICU). Uponarrival,thepatient was inrespiratory distress.Vitals were24 breaths per minute,temperature 34.8^{o}C, heart rate 94beatsperminute,andbloodpressurewas105/52 mmHg.Hehadgrade III encephalopathy perWest Haven criteria, abdominal distention, and asterixis. Right upper quadrant ultrasound revealedalarge, complex right-sided pleural effusion with increased sedimentation without fluid at porta hepatis or ascites(Figure *3*).Portalsystemicanastomosis was patent.Cardiac ultrasound showedahyperdynamic left ventricle withoutright ventricular strain.The patient was subsequently intubated.CT angiography did not show definite areas of extravasation.

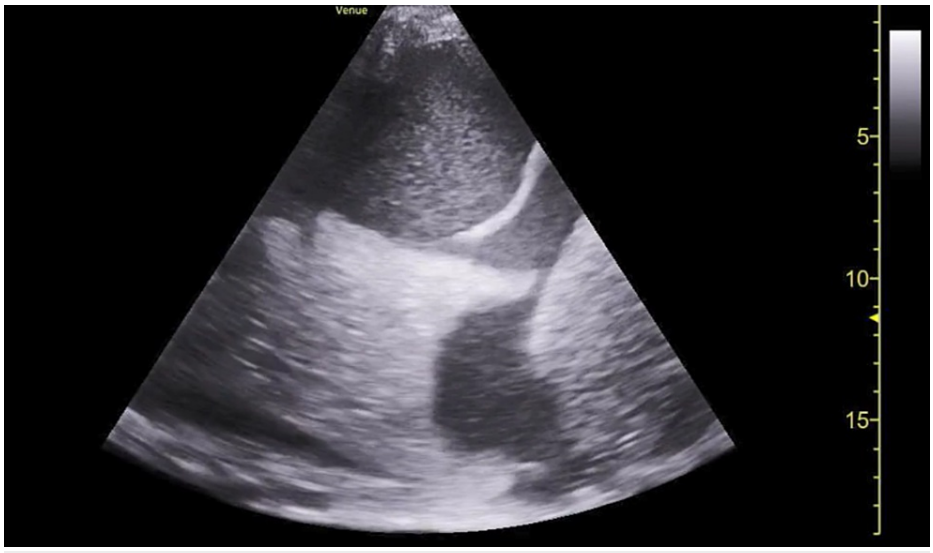

FIGURE 3: Complex right-sided pleural effusion.

Within 10 hoursthepatient received sixadditionalunitsofpRBC,four units fresh frozen plasma,two units of platelets, and minimal norepinephrine.Diagnostic thoracentesis produced red, turbid fluid with 1,960,000 red blood cells/μL (hematocrit 21.5%), meeting criteria for hemothorax. A chest tube was placed andimmediatelydrained 4 Lof fluid.Afterseven days,an additional1.47 Lwas drained.Repeat ultrasoundshowed noneweffusion,andthechest tube was removed.Onceourpatient was hemodynamically stable, he wasdowngradedtomedicine floors and discharged for outpatient follow-up.

Discussion

Case reportsregarding complications of TIPS exist;however,a variety ofserious complications can occur [6,7].Ourcasehighlightspost-TIPSdevelopment ofacomplex pleural effusion complicated by hemorrhagic shockandDIC.It is unclear how this effusion developed sinceno report exists of this complicationoccurringafter TIPS. Itis impossible that our patient'sacutechest outputwas4 L of frank bloodbecausetheaverage human body hasonly4.5-5.5 L ofblood.During the first 24 hoursin the MICU, the patient'snetfluid intake totaled2.7 L.Total output was 6.3 L, with 4.6 L from chest tube output. Had this patient exsanguinated 4 L offrankblood, even with vasopressors, he would have developedsevere

hemodynamic instability.However,our patient maintainedmean arterial pressure consistently>65mmHgwith minimal vasopressors.Therefore, the“hemothorax” was more likely ascitic fluid from aperitoneal-pleural communication (i.e.,diaphragmatic defect)created or enlarged intraoperatively, whichmixedwith blood from hepatic parenchymainjuryduringshunt creation [7,8]. Thismechanismis supported bytheX-rays showing persistentpleural effusionthatdrained1.47 Lover oneweek (Figure4).

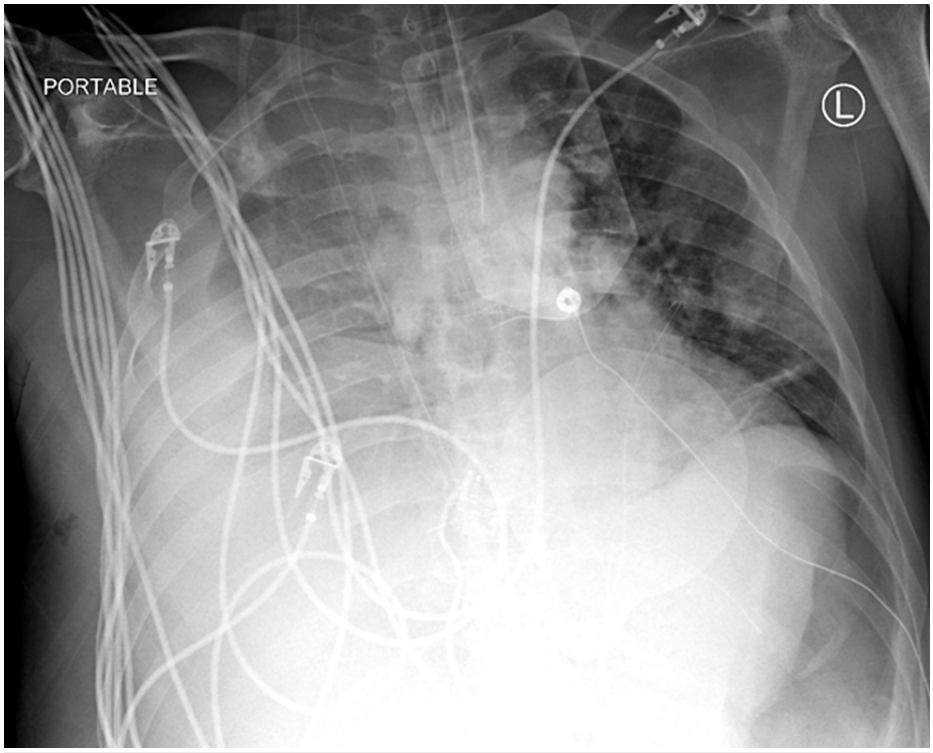

FIGURE 4: Development ofovernight effusion.

Alternatively,traumatotheinternal jugular veinorcarotid arteryduringtheprocedurecould havecontributed tothe hemothorax.Aslowlybleeding vesselmay notbeimmediatelyapparenton imagingbut may collect in the pleural space over time. A right internal jugular vein approach was used; while traversing from the internal jugularveintotheliver,any vessels in the vicinitycould be injuredand bleed into pleural spaceifacommunication exists [9]. Contrary to this theory,no hematoma or acute venous bleeding was noted during the procedure.

The risk of damage to intraperitoneal vasculature or traversal of the liver capsuleoccurring increases when multiple passes are made throughtheliver for TIPS and can cause hemothorax. Excessive blood loss can also result from extrahepatic puncture of the main portal vein, causing massive hemoperitoneum after balloon dilation. These etiologies areless likelybecause only 100 mL of blood loss was recorded intraoperatively and correct portosystemic shunt placement was documented. Right upper quadrant ultrasound 24 hours post-procedure also showed stable ascites with no hematoma or collection at the porta hepatis. Finally, flash malignant pleural effusion is unlikely ascomputed tomography (CT) chest, CT abdomen and pelvis, and bronchoscopy within the month prior to admission did not showany potentialsources of malignancy.

Conclusions

This case seeks tohighlighta rare, yet serious, complication of TIPSso thatpreventative and supportive measures can be taken.While treating these patients, vasopressors, IV fluids, and blood infusions are essential for maintaining patient's hemodynamic stability. Close-interval chest X-ray follow-up is also important to monitor patient’s pleural effusions’ sizes and need for a thoracentesis and chest tubes. In our case, angiography was not conducted to definitely determine the cause of bleeding due to patient instability. However,giventhe knowledge of the procedure and anatomy,wecan hypothesize that the source of bleeding was likely from bloody peritoneal fluid from the procedure, damage to the jugular vein or other blood vessels, or damage to the liver capsule. Future studies would benefit from emergent angiogram if the patient is stable enough to identify a definite source of bleeding.

Additional Information

Disclosures

Human subjects: Consent was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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