

# CHEST<sup>®</sup>

Official publication of the American College of Chest Physicians



## Massive Upper GI Bleeding in a Long-term Hemodialysis Patient

Alpana Chandra, Raymond Tso, Jacob Cynamon and Gregg Miller

*Chest* 2005;128:1868-1873  
DOI 10.1378/chest.128.3.1868

The online version of this article, along with updated information and services can be found online on the World Wide Web at:  
<http://chestjournal.org>

CHEST is the official journal of the American College of Chest Physicians. It has been published monthly since 1935. Copyright 2007 by the American College of Chest Physicians, 3300 Dundee Road, Northbrook IL 60062. All rights reserved. No part of this article or PDF may be reproduced or distributed without the prior written permission of the copyright holder (<http://www.chestjournal.org/misc/reprints.shtml>). ISSN: 0012-3692.

A M E R I C A N C O L L E G E O F



P H Y S I C I A N S<sup>®</sup>

# Massive Upper GI Bleeding in a Long-term Hemodialysis Patient\*

Alpana Chandra, MD; Raymond Tso, MD; Jacob Cynamon, MD; and Gregg Miller, MD

(CHEST 2005; 128:1868–1873)

A 55-year-old diabetic, hypertensive man receiving long-term hemodialysis presented to the emergency department with a complaint of two episodes of lightheadedness, the last being on the morning of hospital admission while straining at stool. He denied having lost consciousness at any time. The patient had been dialysis dependent for the last 5 years and had recurrent episodes of arteriovenous graft thrombosis requiring revisions, with recent placement of a right internal jugular hemodialysis catheter 2 weeks ago after removal of a left internal jugular vein hemodialysis catheter. His diabetes and hypertension were poorly controlled, and he had recently been switched from oral medications to insulin. A review of systems did not reveal any history of conduction abnormalities, heart failure symptoms, or previous ischemic events, but was significant for both resting and exertional dyspnea for the last 1 week.

## Physical Examination

The patient was afebrile and hemodynamically stable (BP 150/60 mm Hg) with no orthostasis: pulse rate, 76 beats/min; normal sinus rhythm; pulse oxygen saturation, 96% on room air; and no acute distress. The examination was significant for neck vein engorgement, a right subclavian hemodialysis

catheter, and a clotted left forearm arteriovenous graft. His right forearm arteriovenous fistula was functioning with a palpable bruit.

## Laboratory Data

The laboratory tests were significant for mild anemia (hemoglobin, 110 g/L), a normal WBC count, and elevated creatinine (14.6 mg/dL) and BUN (65 mg/dL) levels. Liver function test results were normal. Chest radiography revealed no infiltrates and a normal-sized cardiac silhouette.

## Hospital Course

Following hospital admission, assessment for cardiogenic syncope, including 24-h Holter monitoring, echocardiography and tilt-table testing, were negative with no acute pathology on noncontrast CT scans. Three days following hospital admission, the patient reported worsening facial edema with increasing swelling of bilateral upper extremities also noted on examination. Doppler ultrasound of the upper extremities revealed acute, occlusive, and obstructive deep vein thrombosis of the proximal right internal jugular, subclavian, and axillary veins. The left subclavian and internal jugular veins were patent. The findings were confirmed with a contrast chest CT scan (not shown here, but refer to the venogram shown in Fig 1), which also revealed the presence of thrombosis in the left innominate vein extending into the superior vena cava (SVC). The patient was started on anticoagulation with heparin.

After being clinically stable for 24 h, the patient began to have massive hematemesis. IV heparin was stopped, but the patient eventually required intubation for airway protection.

*What is the cause of this patient's condition?*

\*From the Department of Medicine (Drs. Chandra, Tso, and Miller), Division of Critical Care Medicine, and Department of Radiology (Dr. Cynamon), Montefiore Medical Center, Bronx NY.

Manuscript received September 18, 2004; revision accepted November 9, 2004.

Reproduction of this article is prohibited without written permission from the American College of Chest Physicians ([www.chestjournal.org/misc/reprints.shtml](http://www.chestjournal.org/misc/reprints.shtml)).

Correspondence to: Alpana Chandra, MD, Montefiore Medical Center, Division Critical Care Medicine, Goldzone Main Floor, 111 E 210th St, Bronx, NY 10467; e-mail: [achandra@montefiore.org](mailto:achandra@montefiore.org)

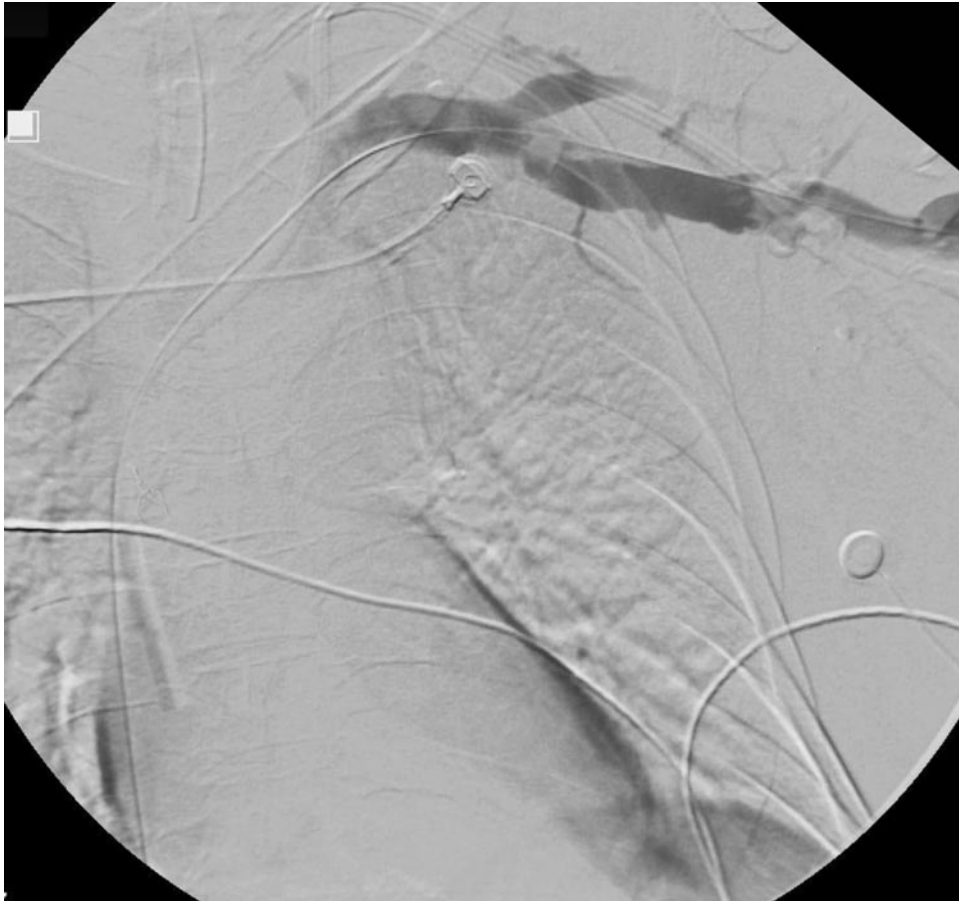


FIGURE 1. Left-upper-extremity venogram showing obstruction of innominate vein due to the presence of the hemodialysis catheter and thrombosis around the catheter.

*Diagnosis: SVC syndrome secondary to thrombosis from hemodialysis catheter.*

*What is the cause of GI bleeding in this patient?*

*How should we manage a case of SVC syndrome of benign etiology?*

SVC syndrome is most commonly secondary to malignancy; however, nonmalignant conditions account for 15 to 22% of SVC obstructions in modern retrospective series. The latter have become increasingly common with the increased use of central venous catheterization and instrumentation, particularly with long-term use of indwelling catheters and intracardiac leads.

Approximately half of these patients present with upper-extremity and/or facial swelling. Dyspnea is the most frequently reported symptom, occurring in up to 70 to 80% of these patients. In addition, 60 to

80% of patients also complain of symptoms associated with increased intracranial pressure manifested as dizziness, syncope, ataxia, convulsions, or coma. Obstruction of the SVC leads to formation of collateral venous channels. These include venous dilatation and varices on the chest wall as well as visceral varices including pericardial varices, breast varices, and “downhill” esophageal varices. Downhill esophageal varices have been reported to occur in 22 to 70% of all patients with SVC obstruction.

Downhill esophageal varices, classically defined as those that develop in the upper region of the esophagus, are less common than the “uphill” type, which are usually produced by portal hypertension. Causes of downhill varices include lung or thyroid carcinoma, mediastinal fibrosis, SVC ligation, metastatic carcinoma, mediastinal mass of unknown origin, venulitis, and muscular constriction of abnormal



FIGURE 2. Same day, after AngioJet and angioplasty procedure with the sheath *in situ* (solid arrow). Now there is prograde flow to innominate vein and SVC also showing presence of collaterals. The hemocath (dotted arrow) was left in place.

extensions of posterior hypopharyngeal veins. Though SVC stenosis or obstruction is a known complication of hemodialysis catheters, Pop and Cutler were the first to describe downhill varices as a specific end result of hemodialysis access. As opposed to uphill varices due to portal hypertension, the downhill varices are caused due to systemic venous obstruction above the diaphragm where the shunted blood flows in parallel with the aorta or downhill. The location and extent of such varices depends on the anatomic site and the duration of venous obstruction.

When the SVC is obstructed at a point further from the heart than the insertion of the azygous system, the drainage of the venous blood is via mediastinal collaterals to the patent azygous-hemiazygous system. The shunted blood then flows to the unobstructed portion of the SVC draining into the heart. Therefore, any such

varices are confined to the cervical esophagus. Thyroid carcinoma, mediastinal fibrosis, occlusion of inferior thyroid veins (resulting from prior thyroidectomy), and occlusion of bilateral brachiocephalic veins (resulting from substernal goiter) have all caused such a pattern of isolated cervical downhill esophageal varices. SVC obstruction at a point closer to the heart than the insertion of the azygous precludes venous return to the heart via both the SVC and the azygous system. Thus, venous blood is diverted via collaterals along the entire length of the periesophageal plexus to the portal vein. The shunted blood returns to the heart via the inferior vena cava. Varices in this instance can be observed along the entire length of the esophagus. There is evidence to suggest that downhill varices initially limited to the cervical esophagus can eventually progress to involve the entire esophagus if SVC obstruction persists long term.

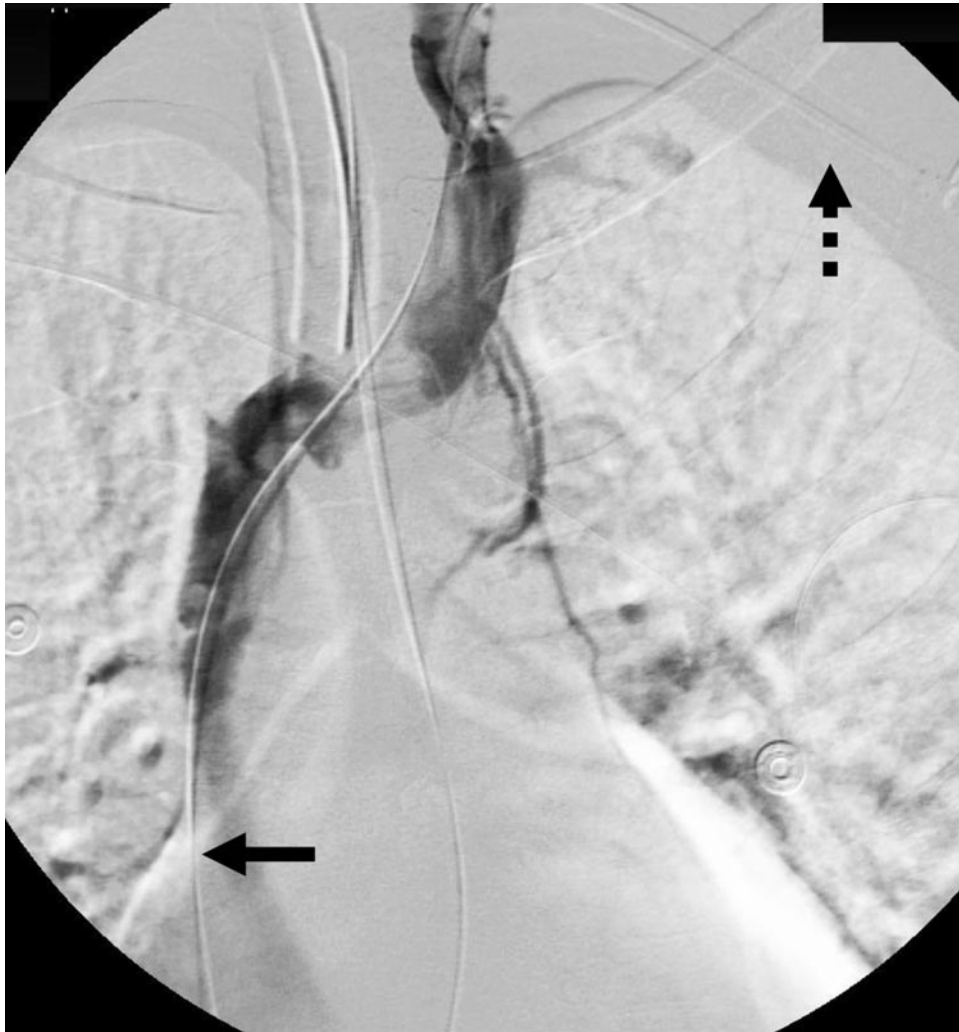


FIGURE 3. A repeat angiogram 5 days later showing partial resolution of thrombosis on venography, showing prograde flow and decrease in collaterals. The hemocath was removed (dotted arrow) and sheath was placed over guidewire (solid arrow).

The relative risk of bleeding from these downhill varices compared to varices caused by portal hypertension is not known. The best initial approach to a patient with bleeding downhill varices and a functioning arteriovenous fistula, in our experience at an outpatient hemodialysis center, is to pinch the arteriovenous fistula to temporarily decrease the venous return through the collaterals. Endoscopic intervention could be attempted; however, reported complications of endoscopic therapy for downhill varices include a greater risk of bleeding and perforation in the proximal esophagus and retrograde flow of sclerosant to the spinal veins. Most definitively, however, treatment of hemorrhage related to downhill varices should focus on attempting to relieve the underlying anatomic problem that is causing the downhill esophageal venous flow. SVC syndrome should be included in the differential diagnosis in all patients with esophageal varices, particularly those isolated to the upper two thirds of the esophagus.

Diagnosis of clinically or endoscopically suspected SVC syndrome is confirmed with contrast-enhanced

CT scans of the neck and chest. Helical CT scans with bilateral upper-extremity contrast injection are likely to combine the diagnostic benefit of CT scanning with the same degree of enhanced vascular detail as digital venography. However, in hemodialysis patients the best study might be a fistulogram. MRI and Doppler ultrasound may be reasonable alternatives for patients with contrast dye allergy.

As SVC obstruction occurring in the setting of an indwelling central venous device (including pacemakers) is generally thrombotic, use of IV heparin may reduce risk of clot progression. Subsequent oral anticoagulant therapy may reduce risk of recurrent thrombotic or distal embolic phenomenon. However, anticoagulation in the presence of bleeding varices, as in our patient, might become problematic.

Acute SVC thrombosis (< 5 days from time of onset of symptoms) is amenable to therapy that includes removal of the offending catheter, and catheter-directed lysis of the superimposed thrombus, with or without percutaneous transluminal angioplasty. The combination of local thrombolytic and



FIGURE 4. Self-expandable stent in the SVC (solid arrow).

endovascular stent placement allows a more aggressive approach to treatment of SVC syndrome and long-term palliation. In institutions where the expertise is available, stent placement might be a reasonable first choice treatment method achieving good mid-term patency. However, because the long-term patency rates of SVC stents are not known, most interventional radiologists tend to treat SVC stenosis from benign causes with balloon angioplasty before placing metallic stents. Symptoms are completely relieved as early as within 1 week of stent placement.

SVC bypass surgery with a spiral saphenous vein graft has also been used successfully for symptomatic relief in patients with benign SVC syndrome. However, long-term surgical success is variable due to the extensive local fibrosis and persistent collateral vessels. It is uncertain whether stent therapy is preferable to surgical bypass in these patients as long-term outcomes are not currently available.

#### Further Clinical Course

Esophagogastroduodenoscopy in our patient revealed three columns of bleeding grade 3 varices in the middle and upper esophagus, most prominently in the upper third. His esophageal varices were attributed to SVC obstruction, although we did not attempt to confirm the downhill flow by venography or magnetic resonance venography.

The variceal bleed in our patient was controlled with banding and then eventually by relief of the SVC obstruction. A left-upper-extremity venogram revealed innominate vein thrombosis (Fig 1). Local thrombus disruption (AngioJet; Possis Medical; Minneapolis, MN) was performed for the left subclavian, brachiocephalic vein and the superior vena cava. Balloon angioplasty of left brachiocephalic vein was done, but no SVC filter was placed at the time (Fig 2, 3).

Three months later, the patient was rehospitalized with a clotted right forearm hemodialysis graft. His facial and upper-extremity swelling had resolved, and he denied any dyspnea. A venogram was done, the graft declotted, and the venous anastomosis dilated. The venogram also incidentally revealed residual SVC stenosis from the last procedure. This time, a decision was taken to place a 14-mm self-expandable stent in the SVC, with good results (Fig 4).

1. Although the most common etiology of the SVC syndrome is still malignancy, nonmalignant conditions account for 15 to 22% of SVC obstructions.

2. There should be a high index of suspicion for SVC syndrome in all cases of unexplained esophageal varices, particularly those in the cervical esophagus.

3. The best initial approach to a patient with bleeding downhill varices and a functioning arteriovenous fistula is to pinch the fistula to temporarily decrease the venous return through the collateral vessels. Endoscopic intervention could be attempted; however, reported complications of endoscopic therapy for downhill varices include a greater risk of bleeding and perforation.

4. Contrast CT is the best diagnostic modality, which also helps to evaluate the site and extent of obstruction, collateral venous drainage, and cause of SVC obstruction.

5. The combination of local thrombolytic and endovascular stent placement allows a more aggressive approach to treatment of SVC syndrome.

6. SVC bypass surgery with a spiral saphenous vein graft has also been used successfully for symptomatic relief in patients with benign SVC syndrome.

#### SUGGESTED READINGS

- Blam ME, Kobrin S, Siegelman ES, et al. "Downhill" esophageal varices as an iatrogenic complication of upper extremity hemodialysis access. *Am J Gastroenterol* 2002; 97:216–218
- Bornak A, Wicky S, Ris HB, et al. Endovascular treatment of stenoses in the superior vena cava syndrome caused by nontumoral lesions. *Eur Radiol* 2003; 13:950–956
- Madan AK, Allmon JC, Harding M, et al. Dialysis access-induced superior vena cava syndrome. *Am Surg* 2002; 68:904–906
- Malra M, Glociczki P, Andrews JC, et al. Open surgical and endovascular treatment of superior vena cava syndrome caused by nonmalignant disease. *J Vasc Surg* 2003; 38:215–223
- Pop A, Cutler AF. Bleeding downhill esophageal varices: a complication of upper extremity hemodialysis access. *Gastrointest Endosc* 1998; 47:299–303
- Serin E, Ozer B, Gumurdulu Y, et al. A case of Castleman's disease with "downhill" varices in the absence of superior vena cava obstruction. *Endoscopy* 2002; 34:160–162

## Massive Upper GI Bleeding in a Long-term Hemodialysis Patient

Alpana Chandra, Raymond Tso, Jacob Cynamon and Gregg Miller

*Chest* 2005;128;1868-1873

DOI 10.1378/chest.128.3.1868

**This information is current as of February 26, 2008**

<b>Updated Information &amp; Services</b>	Updated information and services, including high-resolution figures, can be found at: <a href="http://chestjournal.org/cgi/content/full/128/3/1868">http://chestjournal.org/cgi/content/full/128/3/1868</a>
<b>Permissions &amp; Licensing</b>	Information about reproducing this article in parts (figures, tables) or in its entirety can be found online at: <a href="http://chestjournal.org/misc/reprints.shtml">http://chestjournal.org/misc/reprints.shtml</a>
<b>Reprints</b>	Information about ordering reprints can be found online: <a href="http://chestjournal.org/misc/reprints.shtml">http://chestjournal.org/misc/reprints.shtml</a>
<b>Email alerting service</b>	Receive free email alerts when new articles cite this article sign up in the box at the top right corner of the online article.
<b>Images in PowerPoint format</b>	Figures that appear in CHEST articles can be downloaded for teaching purposes in PowerPoint slide format. See any online article figure for directions.

A M E R I C A N C O L L E G E O F



P H Y S I C I A N S<sup>®</sup>