Budd–Chiari syndrome: a rare complication of central venous line placement

Introduction

Budd–Chiari syndrome is an unusual event caused by obstruction of the hepatic veins which has rarely been described following central line placement. This article describes a case, lists the complications associated with central venous catheters and highlights the importance of checking serial radiographs for adequate line placement.

Discussion

Central venous lines are optimally positioned with a line tip distal to the venous valves at the origin of the superior vena cava, which lies to the right of the midline at the first intercostal space. On a chest radiograph this is just above the level of the right atrium overlying the anatomical position of the superior vena cava. Budd–Chiari syndrome is one of several potential complications associated with placement of central lines (*Table 1*).

Migrations are rarer than malpositioning (McGee et al, 1993) and both should be detected by chest radiography. Migrations of central lines to various sites have been described, including into the azygous vein (Haygood et al, 2012) and superior vena cava (Prabaharan and Thomas, 2014). To the authors' knowledge only one case of Budd–Chiari syndrome secondary to placement of a central venous catheter has been reported (Barreau et al, 2008).

Budd–Chiari syndrome is uncommon, occurring in approximately 1 per 100 000 of the general population (Valla, 2009), and results from obstruction of the hepatic veins, the inferior vena cava or both. Aetiological factors for primary Budd–Chiari syndrome are typically inherited or

Table 1. Complications of central venous catheters

Early (procedural)	Haematoma, arterial puncture, venous rupture
	Pneumothorax, haemothorax
	Pneumomediastinum
	Air embolism
	Malpositioning
	Catheter transection
Late	Catheter-related infection
	Catheter occlusion or thrombosis
	Catheter fracture
	Catheter migration
	Embolic events
	Budd–Chiari syndrome

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Figure 1. Repeat chest radiograph demonstrating migration of the central line tip to below the level of the right hemidiaphragm (arrow).

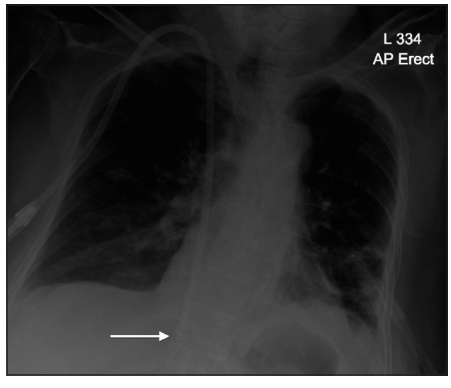
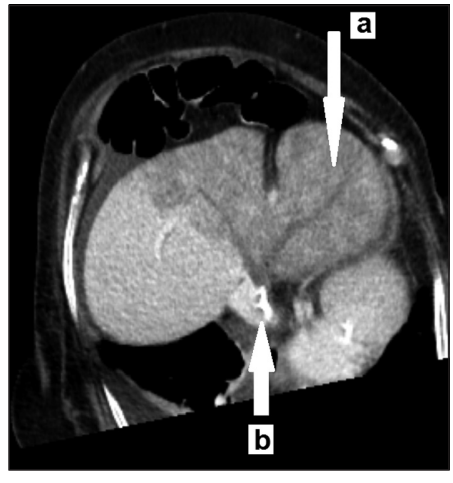


Figure 2. Axial contrast-enhanced computed tomography demonstrates thrombus in the hepatic veins extending out from the tip of the central line (arrow a) and low attenuation 'nutmeg' appearance of the liver (arrow b).



Case Report

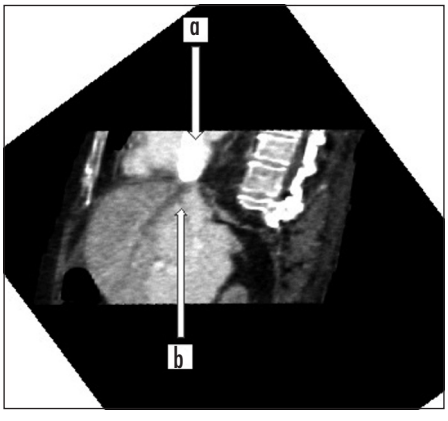
A 69-year-old woman with multiple comorbidities, including lobectomy for lung adenocarcinoma and end-stage renal failure secondary to amyloidosis, was admitted with severe hyperkalaemia and urinary sepsis. A right internal jugular line was inserted for resuscitation and possible dialysis. The initial chest radiograph showed the tip of the catheter at the level of the right atrium. The patient became increasingly unwell with vomiting, abdominal pain, decreased appetite, weight loss and jaundice. Serology indicated deranged liver function tests (albumin 14 g/litre (normal range 35–40 g/litre), alkaline phosphatase 786 IU/litre (30–130 IU/litre), alanine aminotransferase 78 IU/litre (10–40 IU/litre) and total protein 45 g/litre (61–79 g/litre)). The coagulation profile was normal.

Repeat chest radiography showed inferior migration of the catheter tip to a sub-diaphragmatic position (*Figure 1*). Computed tomography of the chest and abdomen excluded malignant recurrence, but demonstrated moderate ascites and a right pleural effusion. The catheter was confirmed to be in the inferior vena cava with thrombus extending from the tip to within the suprahepatic inferior vena cava and left and middle hepatic veins. Heterogeneous hypo-attenuation of much of the liver was also evident (*Figures 2* and *3*). Clinical and radiological findings were consistent with Budd–Chiari syndrome. Owing to the aforementioned comorbidities and deteriorating hepatic and renal function, the patient died.

acquired hypercoaguable states including factor V Leiden mutation, myeloproliferative diseases, pregnancy and oral contraceptive use. In western societies most cases result from hepatic vein thrombosis, and up to one third are idiopathic. Budd–Chiari syndrome is most common in females in their third and fourth decades (Mahmoud et al, 1996). Secondary Budd–Chiari syndrome may be caused by invasion or compression of the hepatic veins by tumour.

The clinical presentation is subacute or chronic in 60% and acute in 20% (Senzolo et al, 2005). Patients with the acute form rapidly develop abdominal pain, ascites, hepatomegaly and jaundice. Uncommonly (<5%), there is fulminant or subfulminant hepatic failure with ascites, tender hepatomegaly, jaundice and renal failure.

Figure 3. Sagittal reformat demonstrates the catheter tip within the suprahepatic inferior vena cava (arrow a) with low attenuation thrombus (arrow b) extending to the liver.



Imaging findings depend on the stage of the disease (Brancatelli et al, 2007). Typical computed tomographic appearances are of reduced attenuation in the liver as a result of reduced portal flow, venous congestion and rarely infarction. In chronic cases associated findings include collateralisation and portal hypertension. A histological 'nutmeg' appearance of the liver is caused by regenerative nodules and patchy enhancement.

Treatment is generally that of any underlying disease where possible. Early anticoagulation is also recommended but most patients require surgical or endovascular intervention as medical management alone has limited success (Klein, 2006). Percutaneous angioplasty and portosystemic shunting are typically used, but up to 20% of patients will require liver transplantation.

Conclusions

This case highlights the importance of awareness of complications of central line placement. Correct positioning of central venous catheters and the role of radiography in assessing for complications, including line migration, are integral to this. BJHM

Barreau M, Minville V, Muscari F et al (2008) Acute iatrogenic Budd-Chiari syndrome occurring on a jugular dialysis catheter. *Am J Case Rep* **9**: 419–21
Brancatelli G, Vilgrain V, Fedele M et al (2007) Budd-Chiari syndrome: spectrum of imaging findings. *AJR* **188**: W168–W176
Haygood TM, Malhotra K, Ng C et al (2012) Migration of central lines from the superior vena cava to the azygous vein. *Clin Radiol* **67**(1): 49–54 (doi: 10.1016/j.crad.2011.05.011)
Klein AS (2006) Management of Budd-Chiari syndrome. *Liver Transpl* **12**(suppl 2): S23–S28 (doi: 10.1002/lt.20941)
Mahmoud AE, Mendoza A, Meshikhes AN et al (1996) Clinical spectrum, investigations and treatment of Budd-Chiari syndrome. *QJM* **89**(1): 37–43
McGee WT, Ackerman BL, Rouben LR et al (1993) Accurate placement of central venous catheters: A prospective, randomized, multicenter trial. *Crit Care Med* **21**: 1118–23
Prabaharan B, Thomas S (2014) Spontaneous migration of central venous catheter tip following extubation. *Saudi J Anaesth* **8**(1): 131–3 (doi: 10.4103/1658-354X.125975)
Senzolo M, Cholongitas EC, Patch D et al (2005) Update on the classification, assessment of prognosis and therapy of Budd-Chiari syndrome. *Nat Clin Pract Gastroenterol Hepatol* **2**: 182–90 (doi: 10.1038/ncpgasthep0143)
Valla DC (2009) Primary Budd-Chiari syndrome. *Hepatology* **50**: 195–203 (doi: 10.1016/j.jhep.2008.10.007)

LEARNING POINTS

- Budd–Chiari syndrome is a rare complication of placing central venous lines.
- Chest radiographs can ensure continued correct line positioning.