

CIRA Case of the Day
April 2015

Case courtesy of Drs. Simon Blais, L. Bouchard and P. Perreault
CHUM

Clinical History

- PB is a 59 year old patient who was referred to our center for severe denutrition and hepatic encephalopathy
- Gastrectomy and pancreatic bypass two years prior, for morbid obesity (BMI 64, 488 pounds)
- During that surgery, NASH cirrhosis was discovered

Clinical History

- Upon arrival, patient weighed 228 pounds.
- He had a CHILD B score
 - INR 1.6
 - Bilirubin 19 mmol/L
 - Albumin 25 g/L
 - Moderate ascites
 - Severe encephalopathy
 - Ammonia 160 mmol/L (N: 3-5)

Legend

- RGV : right gonadic vein
- SMV : superior mesenteric vein
- PV : portal vein
- HA : hepatic artery
- IVC : inferior vena cava
- HE : hepatic encephalopathy
- PSS : portosystemic shunt
- SPSSs: spontaneous portosystemic shunts

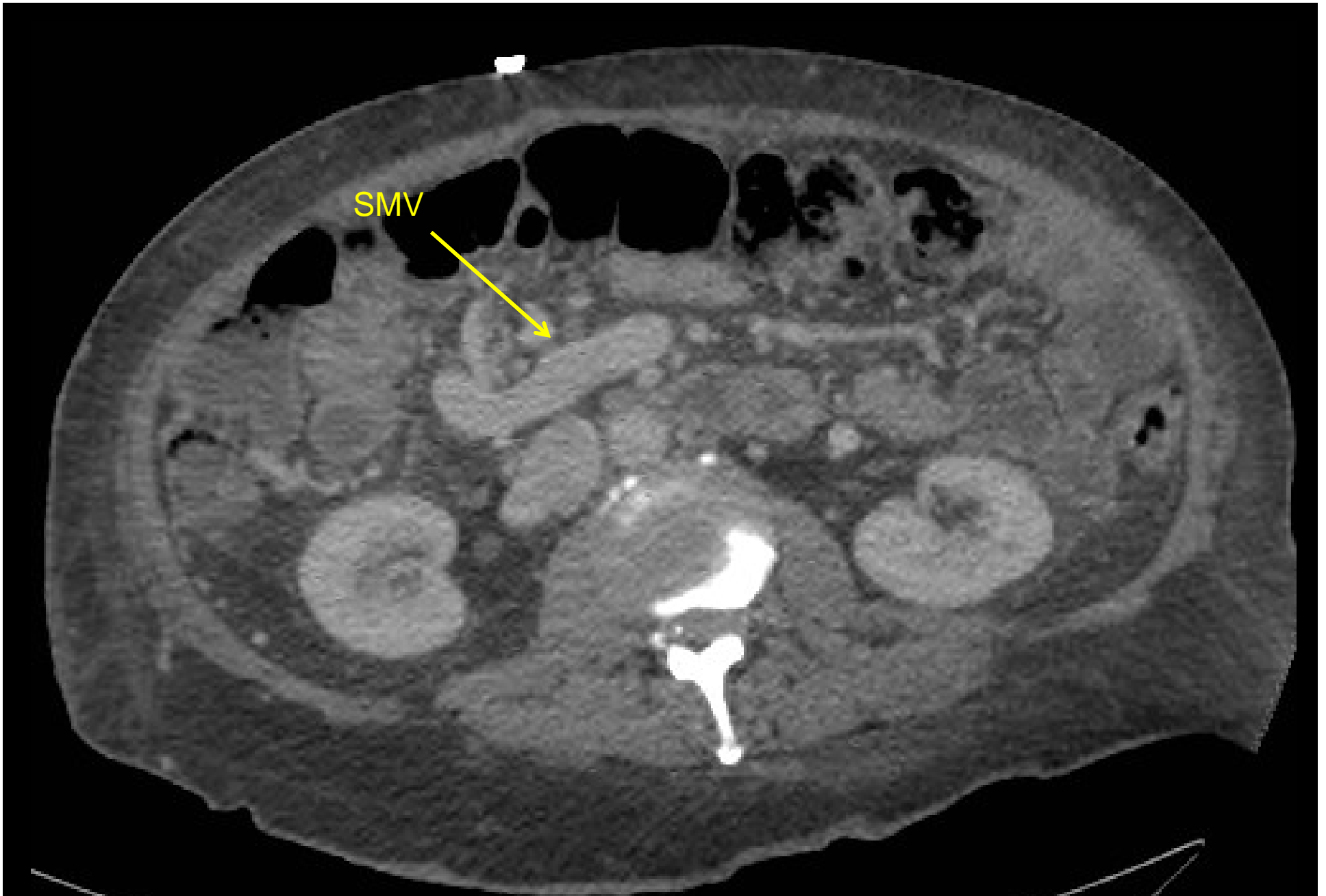


Figure 1a. Angio CT, portal phase shows enlarged superior mesenteric vein

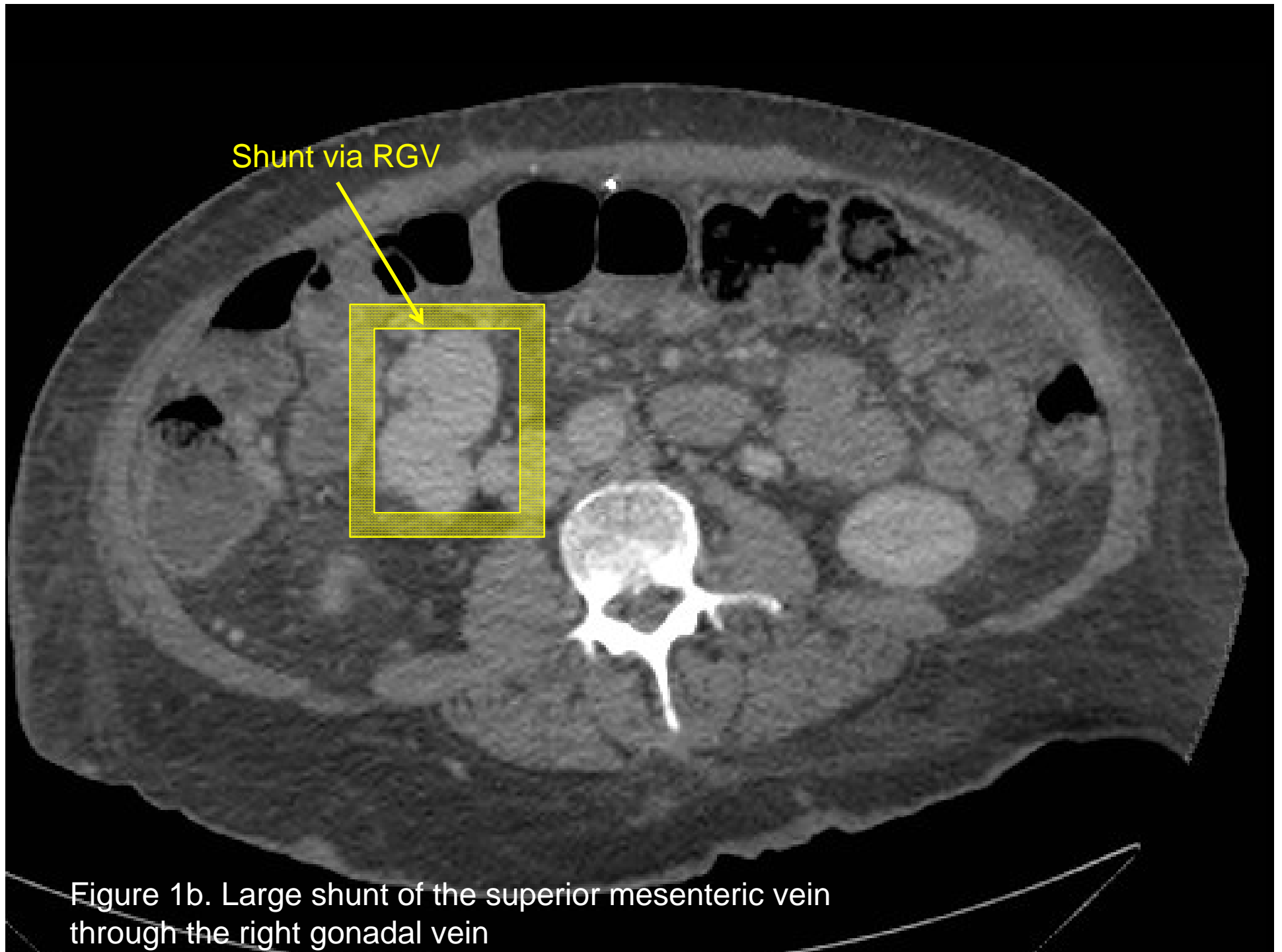




Figure 1c. Numerous varices

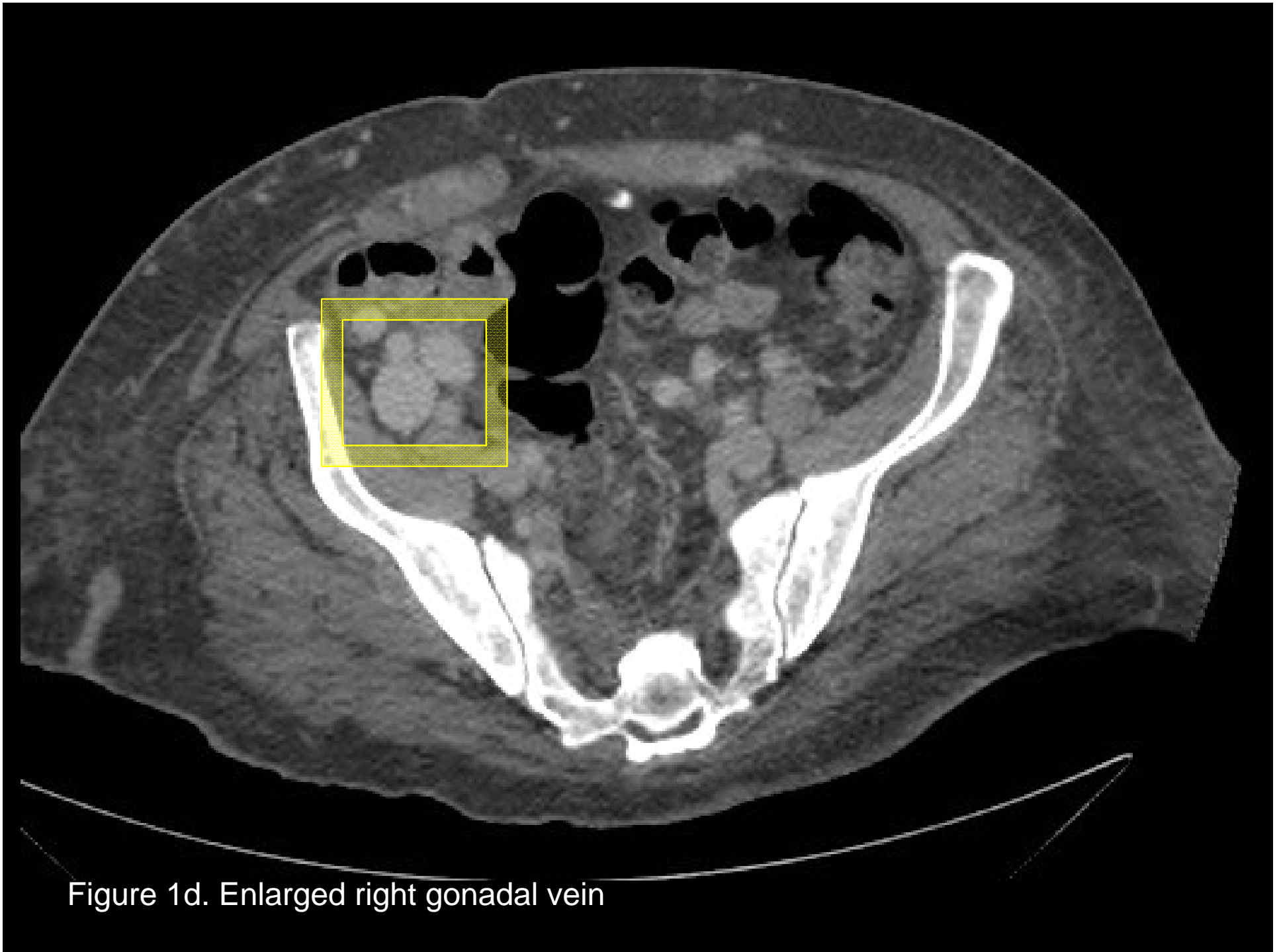
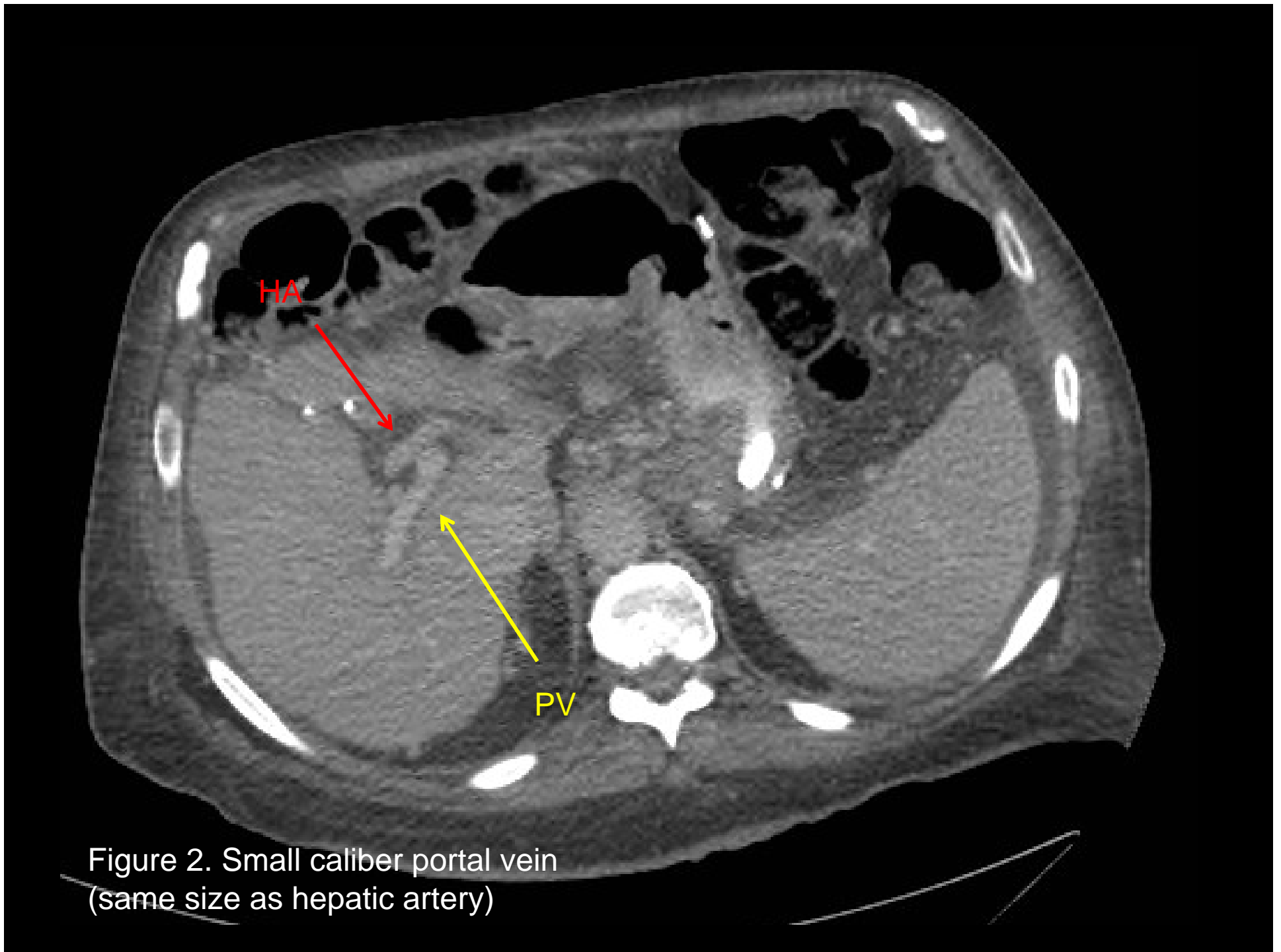


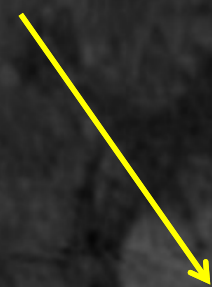
Figure 1d. Enlarged right gonadal vein



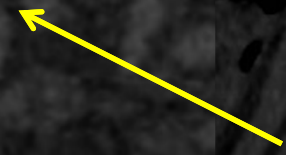
Age: 59, M
Se: 6
2011/02/14 1:40 PM
Kern: B
PHASE PORTALE
C: CONTRAST

1024x1024
CPR
MIP
Tranche: 1.00 mm
Filtre: Aucun

Shunt via RGV



SMV



HELIX
120 kV
236 mA
Tilt: 0.00
No: 139

Figure 3a. Coronal view of the shunt

TERARECON
W: 826 L: 311

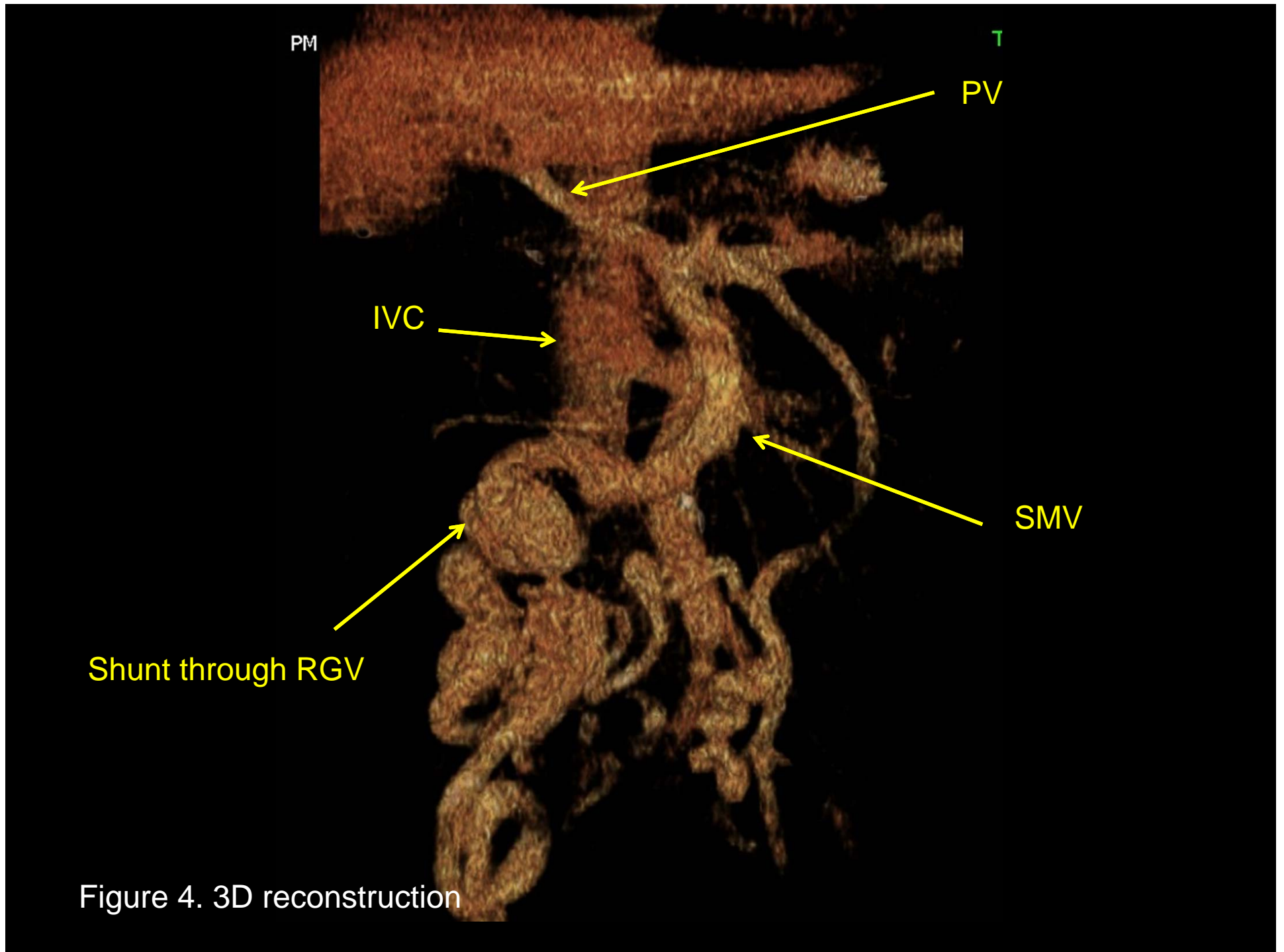
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1024x1024
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HELIX
120 kV
236 mA
Tilt: 0.00
No: 142

TERARECON
W: 749 L: 304

Figure 3b. Sagittal view of the shunt



Imaging Findings

- Massive portosystemic shunt through the right gonadal vein with anastomosis with the superior mesenteric vein
- Mild signs of cirrhosis
- Mild ascites
- NO gastric/esophageal varices

Imaging

- After discussing with the hepatology team, decision was taken to embolize this massive shunt to improve the patient status, especially to treat the medication-refractory encephalopathy.



Figure 5a. Venography of the RGV



Figure 5b. Venography of the RGV and opacification of the inferior vena cava

OAD 41°
CAUD 0°
FD 15 cm

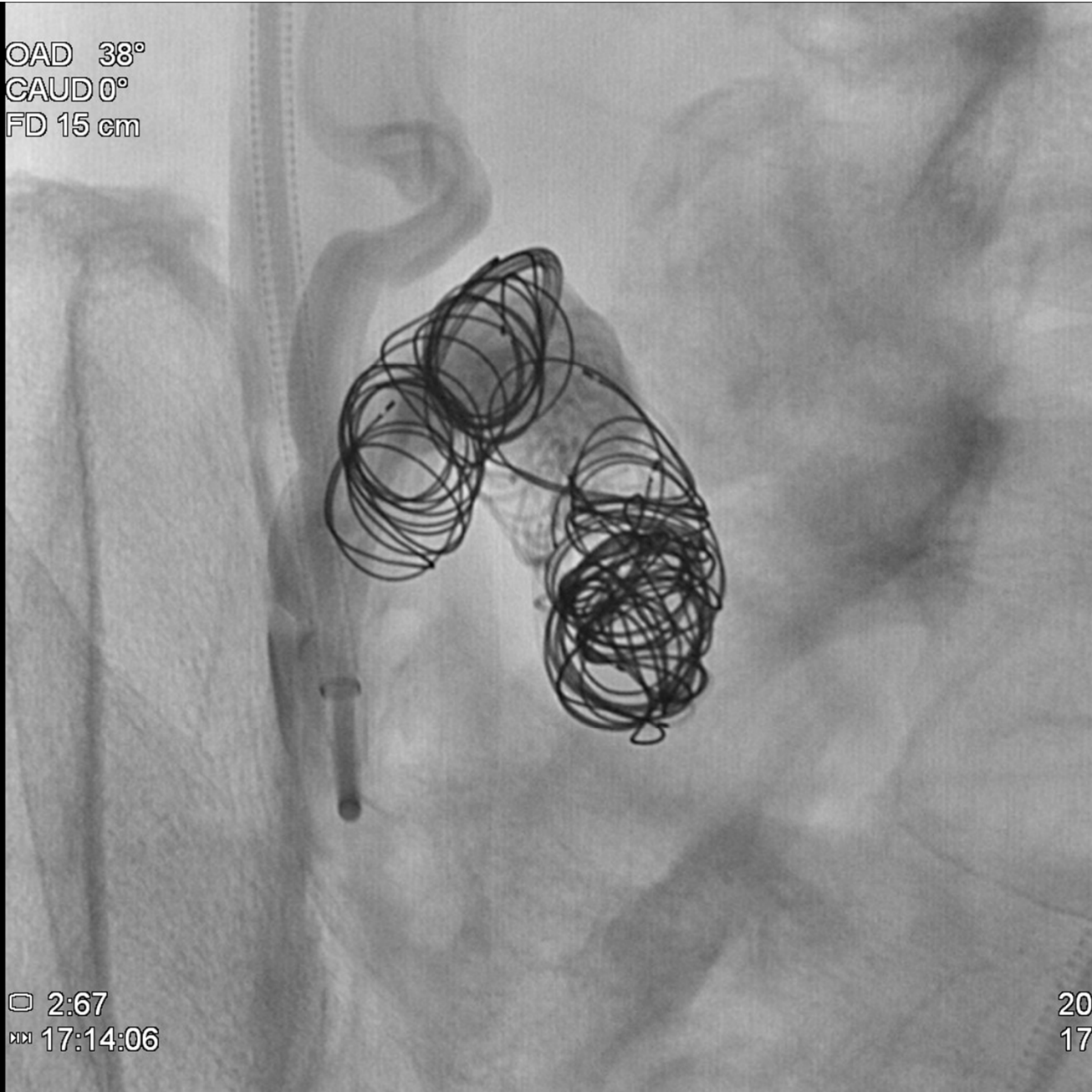


9:93
16:59:35

18
150

Figure 5c. IDC coils deployed in the varices

OAD 38°
CAUD 0°
FD 15 cm



□ 2:67
KX 17:14:06

20
17

Figure 5c. IDC coils deployed in the varices

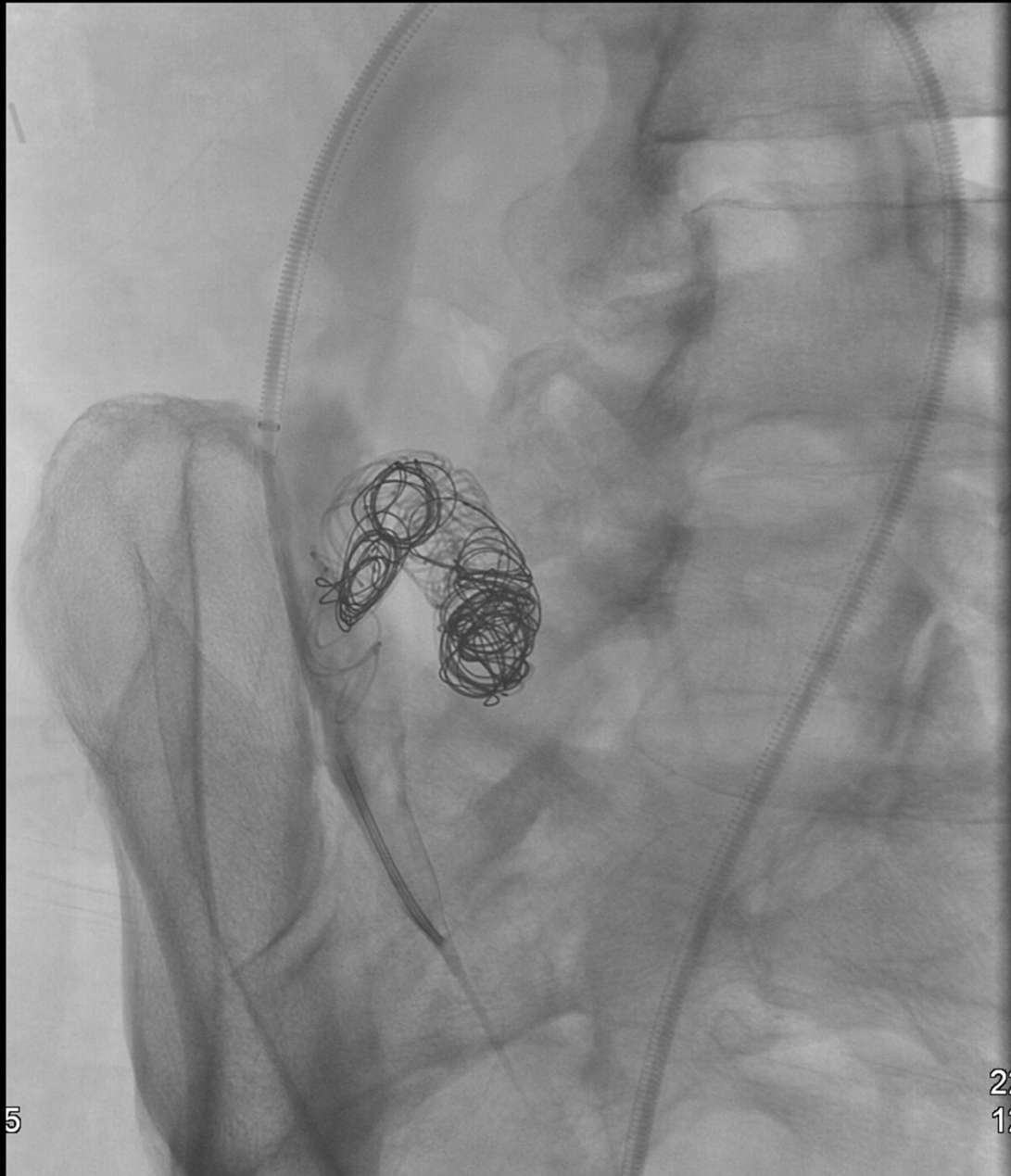


Figure 5c. IDC coils deployed in the varices



Figure 5d. 6F Chuang catheter further in the collateral branches of the RGV



Figure 5e. Multiple Amplatzer II plugs deployed in the shunt

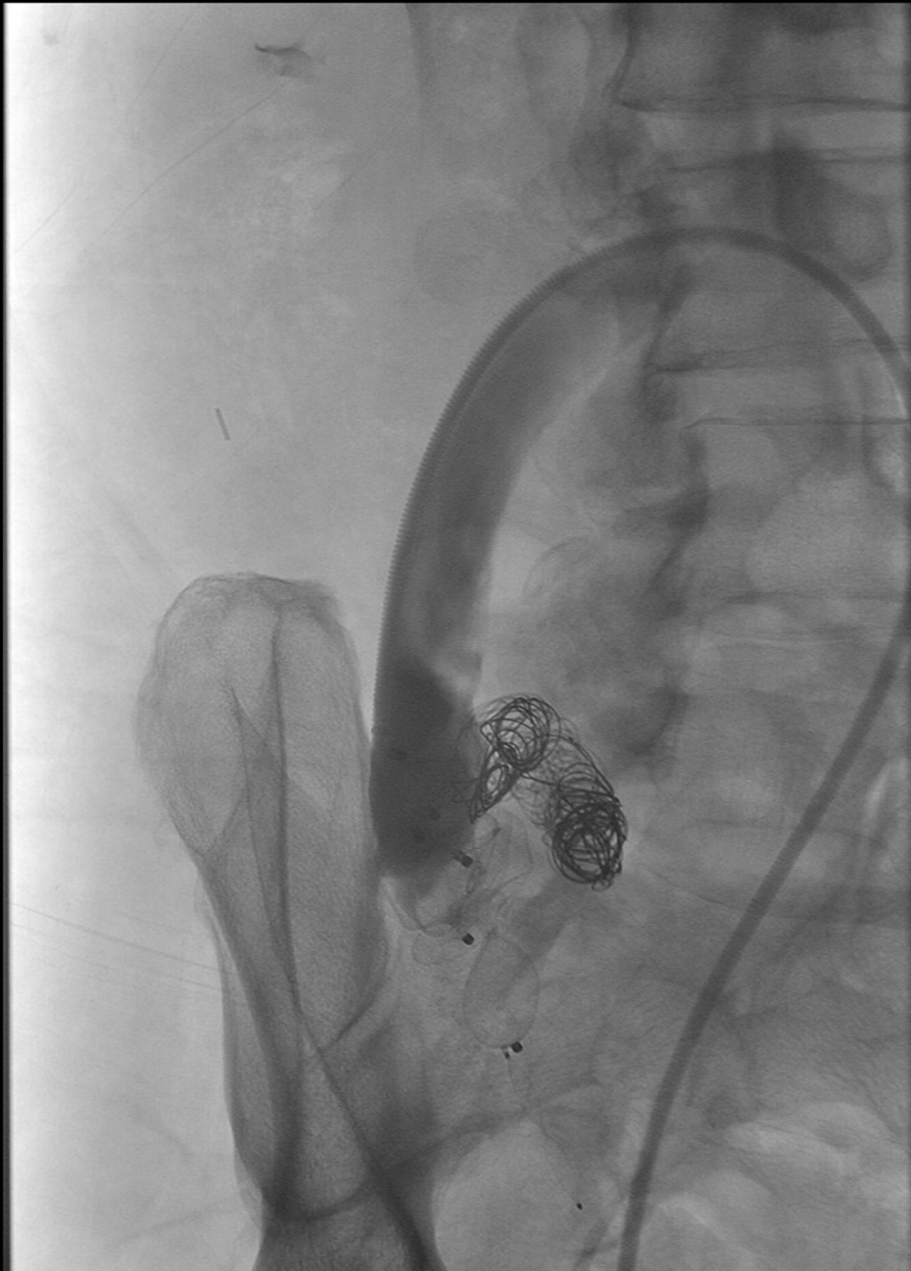
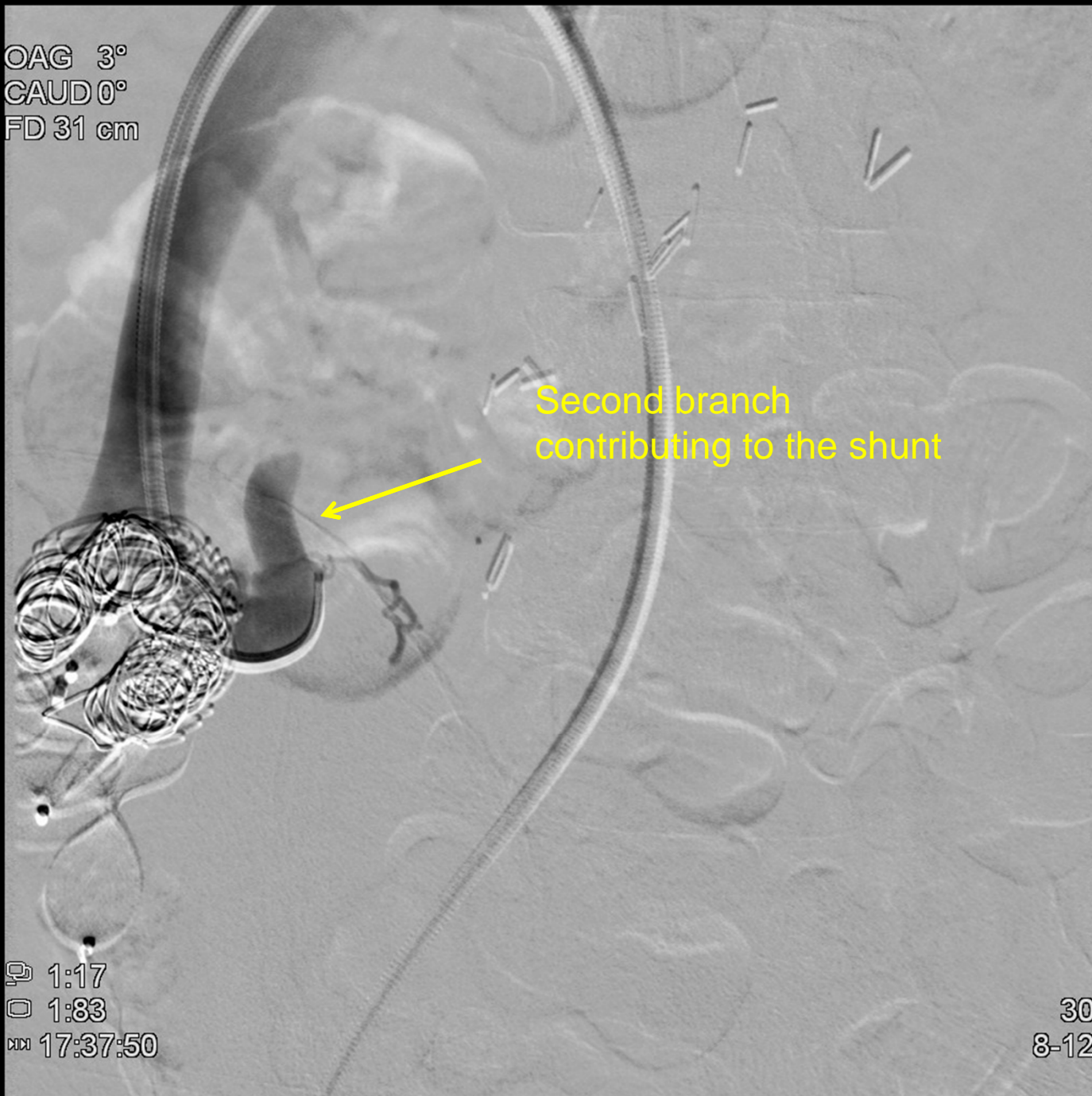


Figure 5e. Multiple Amplatzer II plugs deployed in the shunt

OAG 3°
CAUD 0°
FD 31 cm



1:17
1:83
17:37:50

30
8-12



Figure 5f. Catheter in the second main branch of the shunt

OAG 3°
CAUD 0°
FD 31 cm



Traumatic leak

0:67
2:33
17:43:02

32
5-15

Figure 5g. Catheter in the second main branch of the shunt. Small traumatic leak.



Figure 5h. Two Amplatzer IV plugs in the second main branch of the shunt



Figure 5i. Final view of the RGV and venography of the the IVC

Procedural Details

- RFV approach
- 7F braided sheath
- 6F Chuang catheter
- IDC coils
- Multiple Amplatzer II plugs
- Multiple Amplatzer IV plugs

Portal hypertension

- Defined as portal venous pressure > 10 mmHg
- Two separate mechanisms
 - Increased sinusoidal pressure from deposition of collagen and swelling of hepatocytes causes increase resistance and backward flow
 - Hemodynamic factors : intrahepatic endogenous vasoconstrictors and mesenteric vasodilators both increase blood flow and pressure in the PV system
- Leads to portosystemic collateral vessels

Hepatic encephalopathy

- Major cause of morbidity in patients with cirrhosis
- Due to accumulation of toxins secondary to hepatocellular dysfunction or portosystemic shunting
- 3 types:
 - A : associated with acute liver disease
 - B: associated with portal-systemic bypass and no intrinsic liver disease
 - C: associated with cirrhosis & portal-systemic shunts

Hepatic encephalopathy

- One of the most accepted causes is ammonia influx to the brain which cause edema
- Patient with persistent HE without precipitating event and poorly controlled HE with medication **should be investigated for portosystemic shunts**
- Reports have suggested that 46-70 % of patients with refractory HE have large PSS

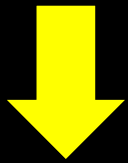
Haussinger et al. 2006

Laleman & al, 2013

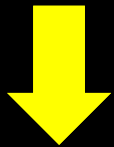
Treatment of refractory HE

- Few series have described occlusion of SPSSs (vascular & surgical)
- In a 37-patients series treated with either coils, amplatzer plugs or matrix, Laleman & al. concluded that
 - 59.4 % of the patients were free of HE in a 100 day period
 - 48.6 % patients remained HE free over a year of follow-up

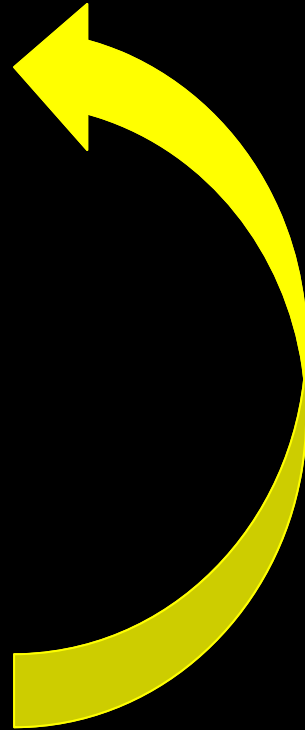
Liver disease



Portal Hypertension



Portosystemic shunts
(+/- varices)



Leads to :
-encephalopathy
-hepatic atrophy
-hepatic failure

Treatment of HE

- Risks:
 - Potential aggravation of portal hypertension
 - Procedure related thrombosis

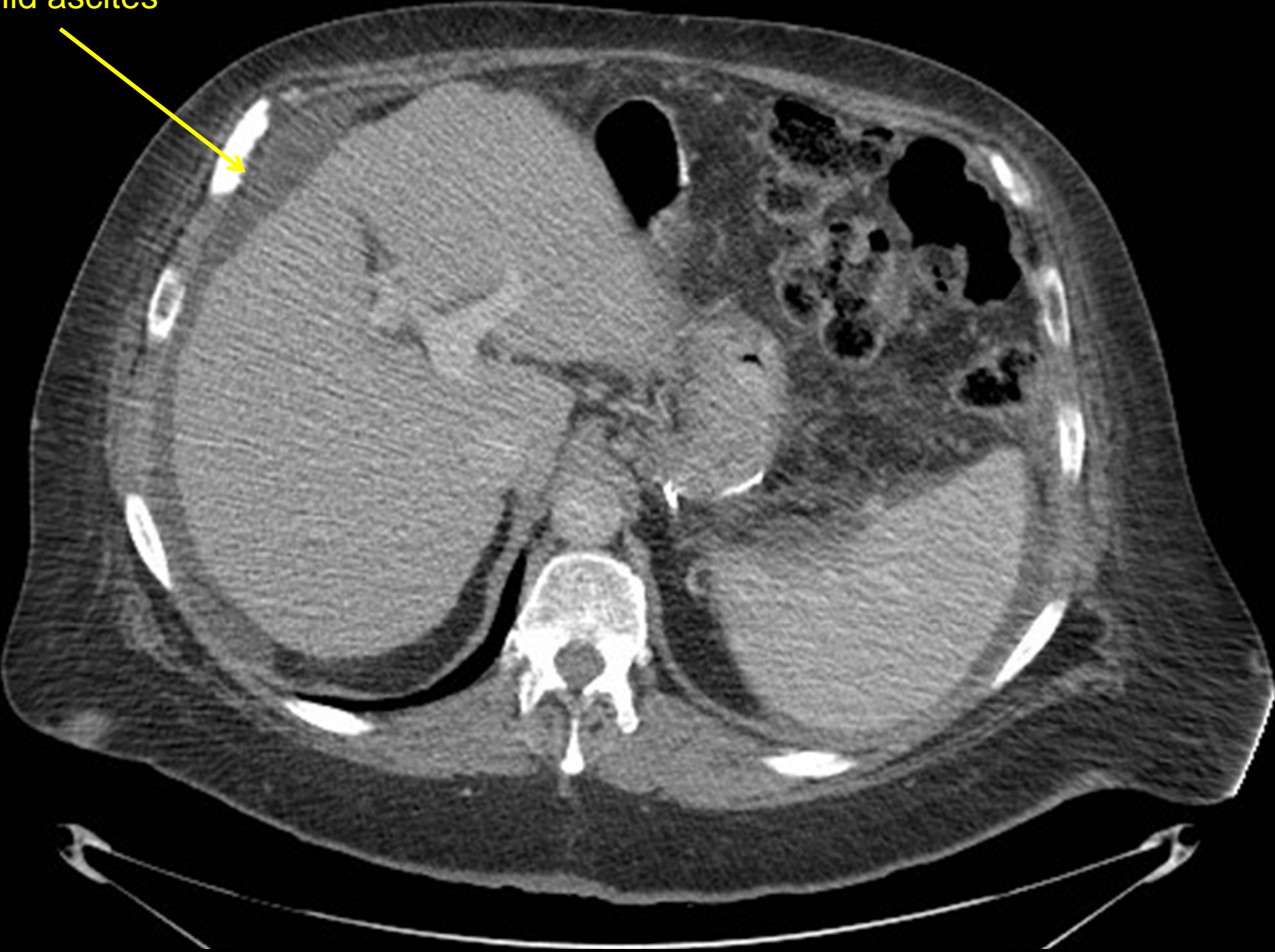
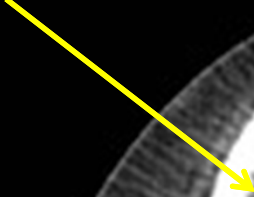
Back to our case...

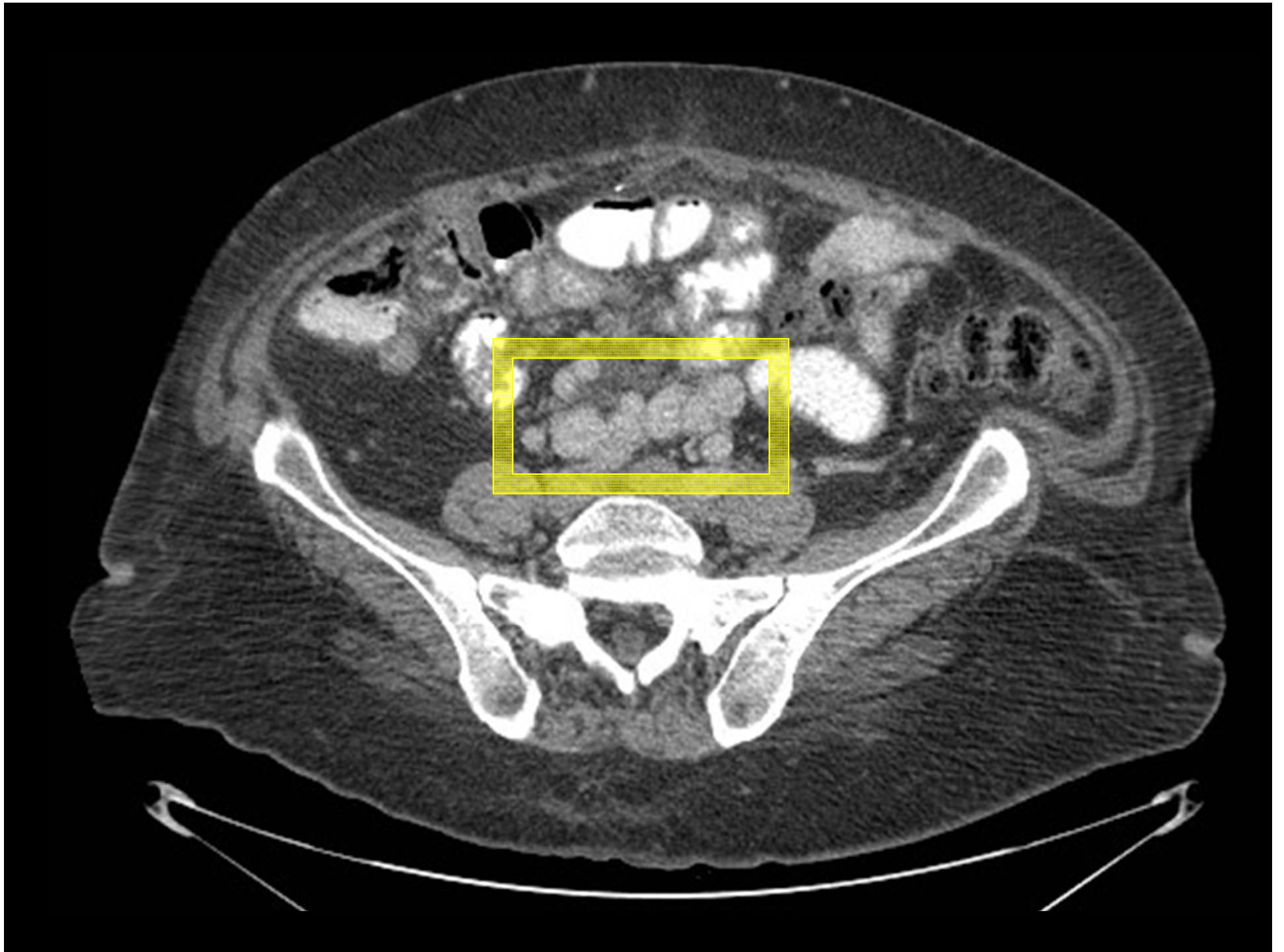
- The patient was discharged a week after the procedure with no encephalopathy and greatly improved clinical status.
- He was followed for a year at our center before being transferred to his local health center.
- The charts indicates a CHILD A cirrhosis, compensated with normal lab values, **no encephalopathy** and no ascites
- The patient is actively working

Follow-up imaging

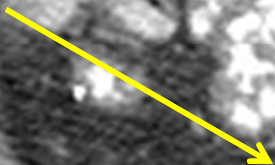


Mild ascites

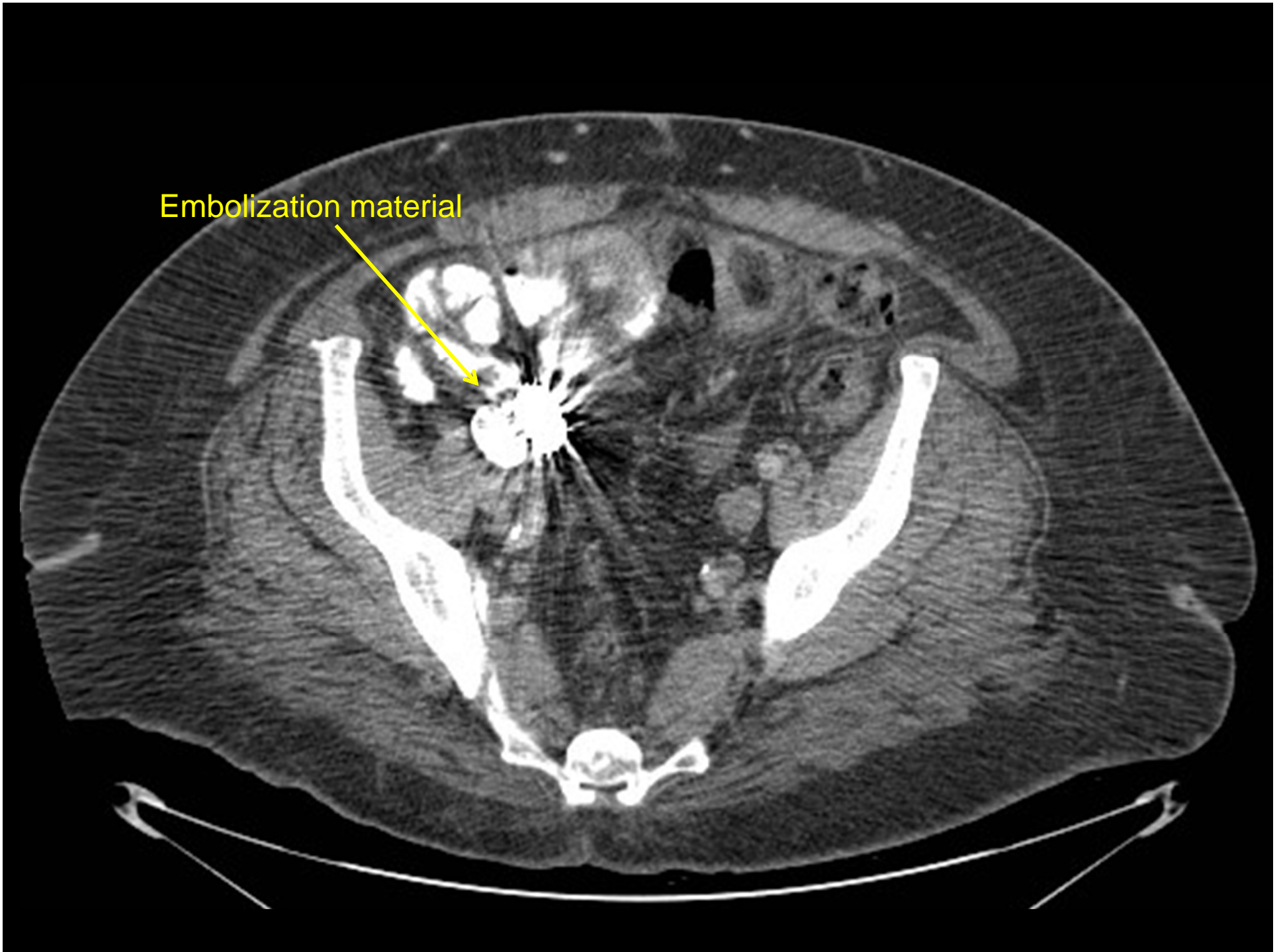
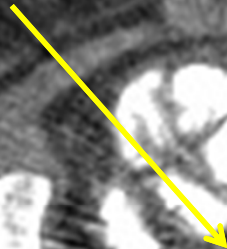




Persistent collateral veins



Embolization material



Follow-Up Imaging

- Some varices left, greatly diminished
- Mild ascites

Take-Home Message

- Severe HE should be investigated for portosystemic shunts
- Closure of those shunts should be considered and discussed with the hepatology team

References

- Laleman et al., *Embolization of Large Spontaneous Portosystemic Shunts for Refractory Hepatic Encephalopathy: A Multicenter Survey on Safety and Efficacy*, Hepatology, vol 57, no 6, 2013
- Wael E. Saad. *Vascular Anatomy and the Morphologic and Hemodynamic Classifications of Gastric Varices and Spontaneous Portosystemic Shunts Relevant to the BRTO Procedure*, TVIR, 2013
- Häussinger et al. *Low grade cerebral edema and the pathogenesis of encephalopathy in cirrhosis*, Hepatology, 2006