Liver Involvement in Hereditary Hemorrhagic Telangiectasia: CT and Clinical Findings Do Not Correlate in Symptomatic Patients

Jim S. Wu[1,2]
Sanjay Saluja[1]
Guadalupe Garcia-Tsao[3]
Alice Chong[1]
Katherine J. Henderson[1]
Robert I. White, Jr.[1]

OBJECTIVE. The purpose of our study was to report the multiphasic CT findings in patients with symptomatic liver involvement by hereditary hemorrhagic telangiectasia (HHT) and to correlate the CT findings with the type of clinical presentation.

CONCLUSION. Patients with symptomatic HHT liver disease have diffuse hepatic telangiectases, a dilated common hepatic artery, and a high incidence of biliary abnormalities. Multiphasic CT is useful in diagnosing liver involvement due to HHT; however, no strong correlation was seen between CT findings and the clinical subtype of HHT liver disease.

Keywords: congenital malformations, CT, hepatobiliary imaging, hereditary hemorrhagic telangiectasia, liver disease

DOI:10.2214/AJR.05.1068

Received June 21, 2005; accepted after revision August 22, 2005.

[1]Department of Diagnostic Radiology, Yale University School of Medicine and Yale–New Haven Hospital, New Haven, CT.

[2]Present address: Department of Radiology, Beth Israel Deaconness Medical Center, 330 Brookline Ave., Landry Bldg., Rm. 357, Boston, MA 02215. Address correspondence to J. S. Wu.

[3]Department of Gastroenterology, Section of Digestive Diseases, Yale University School of Medicine and Yale–New Haven Hospital, New Haven, CT.

WEB
This is a Web exclusive article.

AJR 2006; 187:W399–W405

0361–803X/06/1874–W399

Hereditary hemorrhagic telangiectasia (HHT), or Osler-Weber-Rendu disease, is characterized by an autosomal dominant inheritance pattern, multiple mucocutaneous telangiectases, epistaxis, and visceral arteriovenous malformations [1–3]. HHT affects 1 in 5,000–8,000 individuals [1, 2, 4]. Pulmonary, cerebral, and spinal arteriovenous malformations have been well documented in patients with HHT. Liver involvement is now believed to be a common manifestation of HHT. Recent studies have shown that up to 60% of patients with HHT have liver involvement; however, most of these patients are asymptomatic [3–8].

Symptomatic liver involvement by HHT is uncommon and has been categorized into three distinct clinical patterns by Garcia-Tsao et al. [9]. Patients are divided into clinical subtypes characterized by high-output cardiac failure, portal hypertension, or biliary disease. The clinical symptoms are believed to be a consequence of the predominant hepatic shunt pattern in each patient: arteriovenous, arterioportal, or portovenous.

Imaging of patients with liver disease in HHT can be performed using a variety of techniques: angiography, sonography, MRI, and CT. However, multiphasic CT is probably the best noninvasive test for diagnosing HHT liver disease and for evaluation of the different shunts. With multiphasic CT, the type of shunt can be determined by evaluating early or differential enhancement of the hepatic or portal veins during the various phases of imaging. For example, visualizing contrast material in the main hepatic veins during the arterial phase of the study suggests that the predominant shunt pattern is arteriovenous (hepatic artery to hepatic vein), a consequence of the numerous macro- and microscopic shunts. Contrast-enhanced CT has been shown in small patient groups to have 100% sensitivity in diagnosing patients with HHT liver disease [5, 10, 11].

In our HHT center, routine screening for liver disease is not performed. CT is performed when suggestive symptoms arise or if a liver bruit is present. Thus, only symptomatic patients were evaluated in this study. The purpose of this article is to report the multiphasic CT findings in patients with symptomatic liver involvement by HHT and to correlate the CT findings with the type of clinical presentation.

Materials and Methods

Over a 10-year period (1994–2003), 2,002 patients were evaluated at our HHT center. Of these, 40 had clinical symptoms suggestive of liver involvement by HHT. Patients were categorized into the different HHT subtypes depending on their symptoms at initial presentation. Patients with high-output heart failure all had shortness of breath in the absence of anemia or clinically significant pulmonary arteriovenous malformations. Most patients with high-output failure had liver bruits and some had peripheral edema. Patients in the portal hypertension group presented with ascites or gastrointestinal hemorrhage. Splenomegaly was also noted. The few patients with a biliary subtype presented with abdominal pain and abnormal liver enzymes (elevated levels of alkaline phosphates).

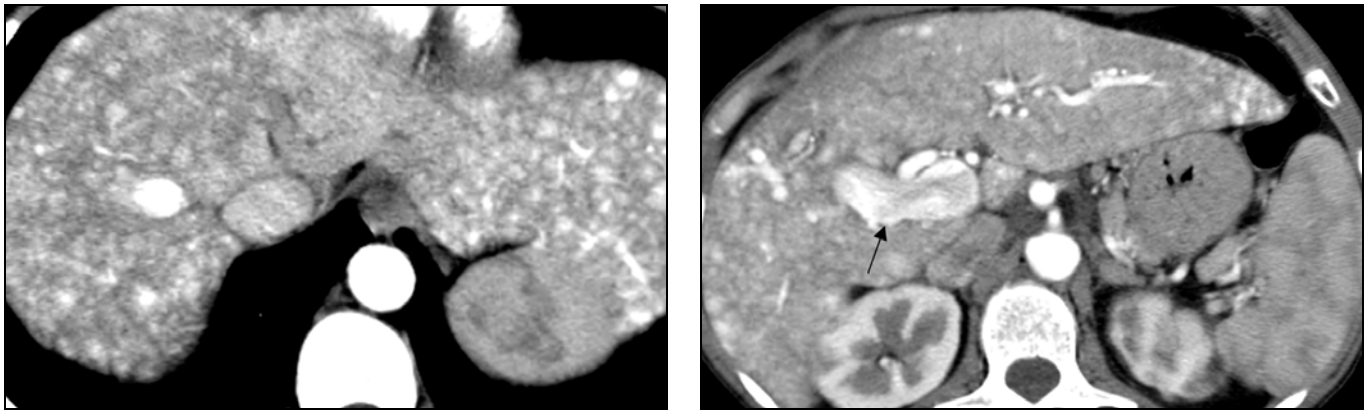


Fig. 1—Axial CT angiography arterial phase images in 46-year-old woman with hereditary hemorrhagic telangiectasia liver disease.
A, CT angiogram shows diffuse parenchymal heterogeneity and numerous telangiectases. All patients in this study showed this heterogenous parenchymal enhancement pattern.
B, Dilation and early filling of main portal vein (*arrow*) during arterial phase are consistent with arterioportal shunt.

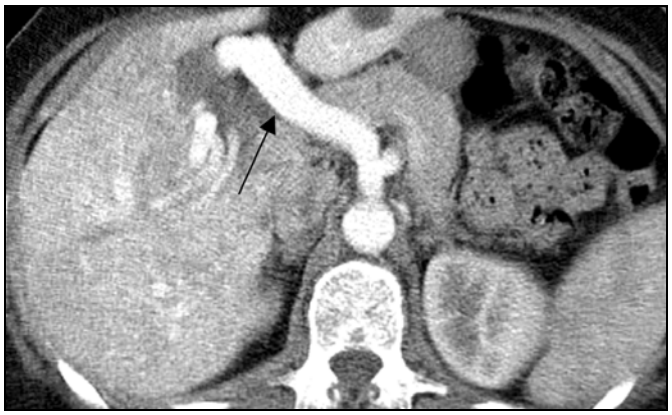


Fig. 2—Axial CT angiography arterial phase image in 57-year-old woman with symptomatic hereditary hemorrhagic telangiectasia liver disease shows markedly dilated common hepatic artery that measures 18 mm in diameter (*arrow*). Dilated common hepatic artery (> 4.5 mm) was seen in all of our patients.

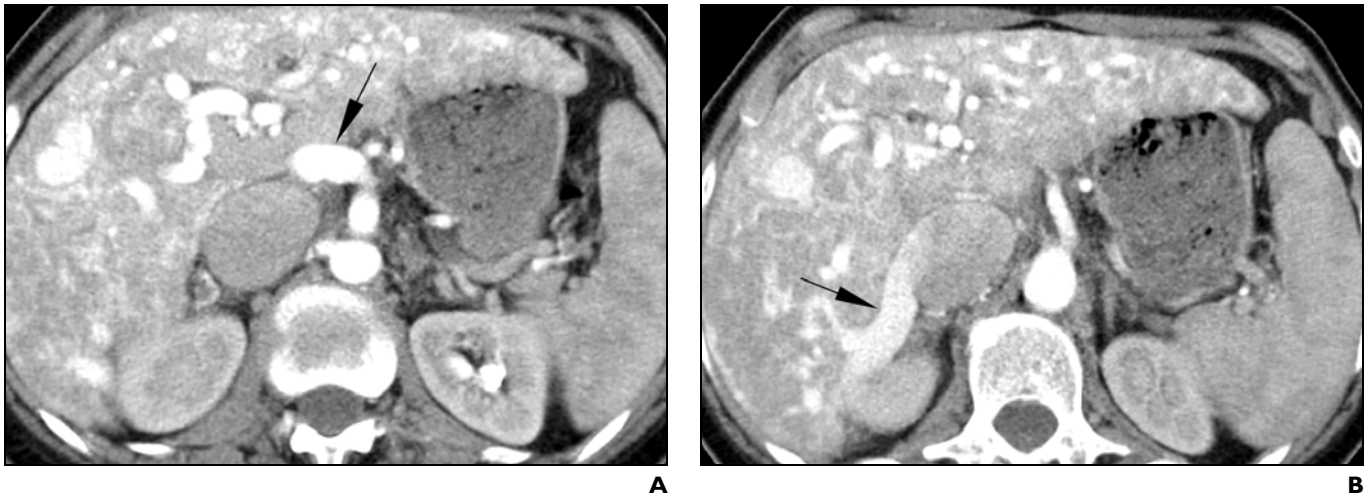


Fig. 3—Axial CT angiography arterial phase images in 64-year-old woman with high-output cardiac failure subtype of hereditary hemorrhagic telangiectasia liver disease.
A, Marked dilation of common hepatic artery (*arrow*) and extensive parenchymal heterogeneity and vascularity are seen.
B, Early filling of right hepatic vein (*arrow*) is consistent with arteriovenous shunt.

Other causes of hepatic dysfunction, such as hepatitis B and C, alcoholic cirrhosis, Wilson's disease, and transfusion-related hemosiderosis, were excluded. Of these 40 patients, 24 underwent multiphasic CT. The 16 patients not included in the study predated the use of multiphasic helical CT at our institution and were imaged via other techniques such as angiography, MRI, sonography, or nonmultiphasic CT. At the time of imaging, 16 patients had symptoms predominantly related to high-output heart failure, 6 had symptoms related to portal hypertension, and 2 had biliary symptoms.

All 24 CT examinations were performed using a multiphasic technique: 13 were performed on a single-detector helical scanner and 11 were performed on a 4-MDCT scanner. Studies consisted of unenhanced, arterial phase, portovenous phase, and delayed phase images. The IV bolus was 140 mL of iohexol (Omnipaque, Nycomed) using an automated power injector at a rate of 3 mL/s. A pitch of 1.0 was used for single-detector CT and a pitch of 1.25 for MDCT. No oral contrast medium was given. The arterial phase images were obtained after a 20-second delay from the onset of contrast administration, the portovenous phase images at 70 seconds, and the delayed phase images at 120 seconds after contrast administration.

The hepatic enhancement pattern and vascular malformations such as diffuse telangiectases were documented (Fig. 1A). The size of the common hepatic artery was noted and was considered enlarged if the diameter was greater than 4.5 mm [4, 6, 12], measured before the origin of the gastroduodenal artery (Fig. 2). Cirrhosislike changes were noted if there was surface contour nodularity, hypertrophy of the caudate and left hepatic lobe, or atrophy of the right hepatic lobe. The type of vascular shunting pattern (arteriovenous, arterioportal, or portovenous) was documented. An arteriovenous shunt was diagnosed if there was early filling of the hepatic veins on the arterial phase portion of the study (Figs. 3 and 4). An arterioportal shunt was detected if there was early filling of the portal veins on the arterial phase images (Fig. 1B). Portovenous shunts were documented if a vessel connecting the portal to the hepatic veins or the inferior vena cava was visualized.

The images were also evaluated for signs of high-output cardiac failure, portal hypertension, and biliary disease to predict the clinical subtype of HHT liver disease. High-output cardiac disease was predicted if cardiomegaly was present. Cardiomegaly was documented if the ratio of the heart width to the chest cavity on the anteroposterior scout radiograph was greater than 0.7. Portal hypertension was predicted if any two of the following three findings were present: splenomegaly, arterioportal shunt, or dilation of the main portal vein (Fig. 5). Splenomegaly was noted if the craniocaudal dimension was greater than 13 cm. Portal vein enlargement was considered to be present if the vein diameter exceeded 13 mm, measured just before the formation of the right and left portal veins [13]. The presence of biliary cysts or focal biliary dilation were indications of biliary disease (Fig. 6). The "predicted" subtype of HHT liver disease for each patient, based on the CT findings, was determined in consensus by two board-certified radiologists who were blinded to the clinical data.

Results

Results are summarized in Table 1. Of the 24 patients in the study, 19 were women (79%) and five were men (21%), and the median age of all patients was 62 years (range, 35–81 years). All patients met the definite criteria for HHT and had symptomatic liver disease not attributable to other causes (hepatitis B and C, alcoholic cirrhosis, Wilson's disease, or transfusion-related hemosiderosis).

On CT, all patients had heterogeneous enhancement of the entire liver with small vascular malformations (< 10 mm) consistent with diffuse telangiectases. The common hepatic artery was dilated (> 4.5 mm) in all patients, with a median diameter of 11 cm (range, 6–18 cm). Marked dilation of the common hepatic artery (≥ 10 mm) was seen in 14 (58%) of 24 patients. Cardiomegaly was seen in 9 patients (38%), splenomegaly in 4 patients (17%), a nodular cirrhosislike liver in 9 patients (38%), and ascites in 2 patients (8%).

The most common shunt was the arteriovenous (hepatic artery to hepatic vein) shunt, which was observed in 13 (54%) of 24 cases overall, in 9 (56%) of 16 patients with clinical heart failure, in 2 (33%) of 6 patients in the portal hypertension group, and in both patients with biliary symptoms. There were 8 (33%) of 24 cases of arterioportal shunt overall—4 (25%) of 16 of the heart failure patients, 4 (67%) of 6 of the patients with portal hypertension—and in neither of the 2 patients with biliary disease. Only one portovenous shunt was visualized, and this patient was in the portal hypertension group but had no evidence of encephalopathy. A specific shunt was not visualized in 7 (29%) of 24 patients. In one patient, all three shunt types were seen; and in three patients, both an arteriovenous shunt and an arterioportal shunt were present.

Regarding the correlation between clinical presentation and CT findings, 4 (17%) of 24 patients could not be classified on CT because of a paucity of findings. The most common abnormality identified on CT was biliary disease, with 11 (46%) of 24 patients having biliary dilation or cysts. Biliary abnormalities identified on CT were not specific for clinical type, and the two patients with biliary clinical presentations had both heart failure and biliary findings on CT (although one patient developed clinical heart failure later in the course of her disease). Of the 16 patients with clinical heart failure, only 7 (44%) were identified correctly on the basis of CT findings. Of the 6 patients with clinical signs of portal hypertension, 4 (67%) were identified correctly on the basis of CT and all 4 patients had an arterioportal shunt. Overall, CT findings determined the clinical type of disease in 13 (54%) of 24 patients.

Discussion

Liver disease in patients with HHT is a complex process because of the myriad of vascular shunts and associated clinical symptomatology. On the basis of past studies, it appears that most patients are asymptomatic. Ianora et al. [11] evaluated 70 patients with HHT for liver disease using multiphasic CT, and 52 had abnormal liver findings on CT. However, only 4 of the 52 patients were symptomatic. Similarly, Buscarini et al. [4] screened 40 patients with HHT for liver disease using Doppler sonography and found that only 3 of the 13 patients with HHT liver disease were symptomatic. Nonetheless, in affected patients, HHT liver disease can lead to significant morbidity and mortality, with the only cure being liver transplantation for severe cases [14–16].

Symptomatic liver involvement by HHT has been categorized into three distinct clinical patterns by Garcia-Tsao et al. [9]. The most common clinical type is high-output cardiac failure. These patients usually have shunts from the hepatic artery to the hepatic veins (arteriovenous shunt), causing excessive return of blood to the heart and eventually leading to right-sided heart failure. The next most common clinical type is portal hypertension. This type can result from hepatic artery to portal vein (arterioportal) shunts but is probably more commonly due to increased intrahepatic resistance as a result of nodular regenerative hyperplasia. The arterioportal shunt leads to portal hypertension and consequently to splenomegaly, ascites, and gastroesophageal varices.

The biliary disease subtype of liver involvement by HHT is characterized by biliary strictures and dilation and bile cysts. These patients have clinical symptoms related to biliary obstruction or sepsis [9, 17]. Abnormalities are due to biliary ischemia that results from arteriovenous shunting [4, 9]. Other

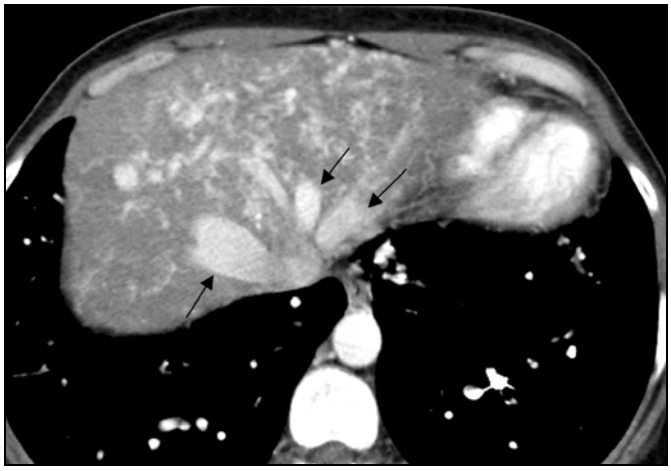
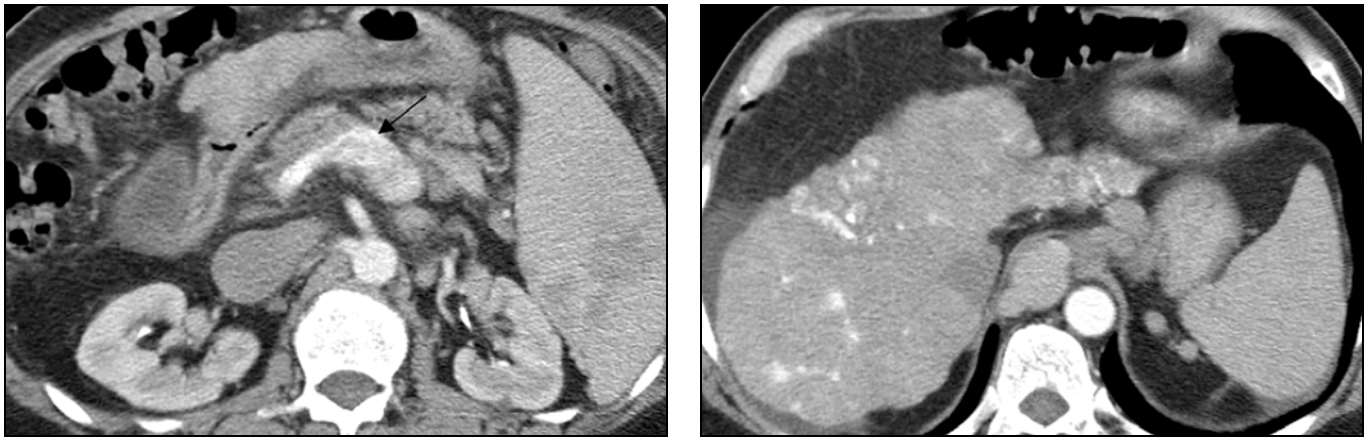


Fig. 4—Axial CT angiography image in 48-year-old woman with symptomatic hereditary hemorrhagic telangiectasia liver disease. Early filling of right, middle, and left hepatic veins (*arrows*) during arterial phase of study is consistent with arteriovenous shunt.



A **B**

Fig. 5—Axial CT angiography images of 62-year-old man with symptomatic hereditary hemorrhagic telangiectasia liver disease.
A, Opacification of main portal vein (*arrow*) is seen during arterial phase, which is consistent with arterioportal shunt. Splenomegaly is also present.
B, Contour nodularity and atrophy of right hepatic lobe are consistent with cirrhosislike changes.

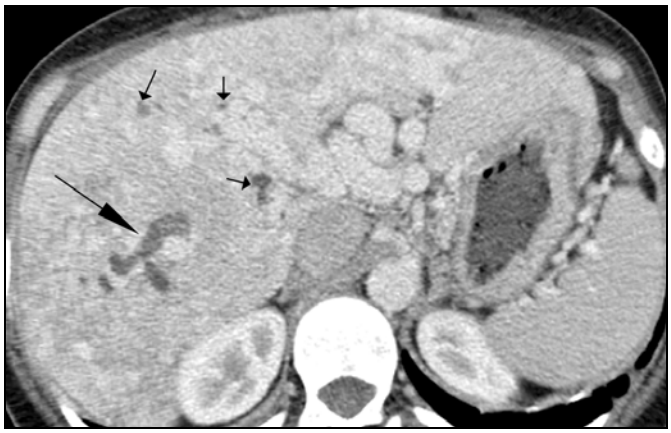


Fig. 6—Axial portovenous phase CT angiography image in 48-year-old woman with biliary subtype of hereditary hemorrhagic telangiectasia liver disease. Dilation of peripheral biliary branch in right hepatic lobe (*large arrow*) is seen. Smaller areas of biliary dilation are also present (*small arrows*).

TABLE 1: CT Findings in 24 Patients with Symptomatic Liver Involvement by HHT, Classified by Clinical Presentation

Patient No.	Age (y)	Sex	Clinical HHT Subtype	Predicted Subtype by CT	Hepatic Artery Diameter (mm)	Dilated Portal Vein	Type of Shunt	Cardiomegaly	Splenomegaly	Biliary Cysts or Dilatation
1	70	F	HF	HF	6			+		
2	61	F	HF	HF	8	+	AV	+		
3	81	F	HF	HF	9			+		
4	56	F	HF	HF	9			+		
5	70	F	HF	HF	12		AV	+		
6	77	F	HF	HF/B	13	+		+		+
7	63	M	HF	HF/PHT/B	13	+	AV/AP	+	+	+
8	62	M	HF	PHT	7		AP		+	
9	66	F	HF	PHT	14	+	AV/AP			
10	74	F	HF	B	9		AP			+
11	35	F	HF	B	10		AV			+
12	62	F	HF	B	10		AV			+
13	57	F	HF	B	18		AV			+
14	72	F	HF	NA	8					
15	64	F	HF	NA	12		AV			
16	38	F	HF	NA	13		AV			
17	66	F	PHT	B	9	+				+
18	54	M	PHT	PHT	12	+	AP		+	
19	46	F	PHT	PHT	6	+	AV/AP/PV			
20	64	M	PHT	PHT/B	15	+	AP		+	+
21	77	M	PHT	PHT/B	16	+	AV/AP			+
22	60	F	PHT	NA	8					
23	48	F	B	HF/B	13		AV	+		+
24	57	F	B	HF/B	17		AV	+		+

Note—HHT = hereditary hemorrhagic telangiectasia, HF = high-output heart failure, AV=arteriovenous, B = biliary, PHT = portal hypertension, AP = arterioportal, PV = portovenous, NA = unable to predict based on CT findings. Plus sign (+) indicates present.

symptoms that have not been classified and that occur as a consequence of shunting are hepatic encephalopathy, which results from portal vein to hepatic vein (portovenous) shunting, and mesenteric ischemia, which results from mesenteric steal of blood through the hepatic artery [18–21].

Although the three possible predominant patterns of shunting (arteriovenous, arterioportal, and portovenous) lead to different clinical subtypes, it is unclear what mechanisms determine the expression of one subtype over another [8]. At least theoretically, heart failure would result from shunting that increases cardiac preload (arteriovenous or portovenous shunts). Portal hypertension results from either an increased flow into the portal system (arterioportal shunt) or hepatic anatomic abnormalities (nodular regenerative hyperplasia) leading to increased intrahepatic resistance. Finally, biliary symptoms result from shunting of the blood away from the peribiliary plexus (arteriovenous or arterioportal shunting). Most likely all three types of shunting occur concomitantly, with one predominant shunting pattern producing the main clinical subtype. However, the predominant type of shunting may change over time, which would explain the overlapping clinical presentations and the transition from one clinical presentation to another [9].

In this study, we sought to characterize the CT findings in patients with symptomatic liver disease. Previous studies have concentrated mostly on asymptomatic patients or small case reports of symptomatic patients [4–7, 10, 19, 21]. We report 24 consecutive symptomatic patients with well-documented liver involvement that could be categorized into one of the three clinical subtypes of liver involvement by HHT [9].

In our study, we found that all patients with symptomatic liver involvement have diffuse liver telangiectases that led to a markedly heterogeneous hepatic enhancement pattern. This appearance is characteristic of liver involvement by HHT. The common hepatic artery was dilated in all patients. Although other disease processes with increased hepatic blood flow, such as hepatocellular carcinoma, hemangiomas, highly vascular metastases and cirrhosis, can have common hepatic artery dilation, the hepatic artery in those cases is not as dilated as in HHT [3, 12]. In our study, 14 (58%) of 24 patients had marked dilation of the common hepatic artery (≥ 10 mm). The presence of telangiectases and dilation of the common hepatic artery as signs of HHT liver disease have been described in many other studies [5–7, 10–12, 18, 21–23]. The presence of these two findings is pathognomonic for HHT liver involvement, particularly in the presence of a compatible clinical history, and should obviate biopsy in suspected cases.

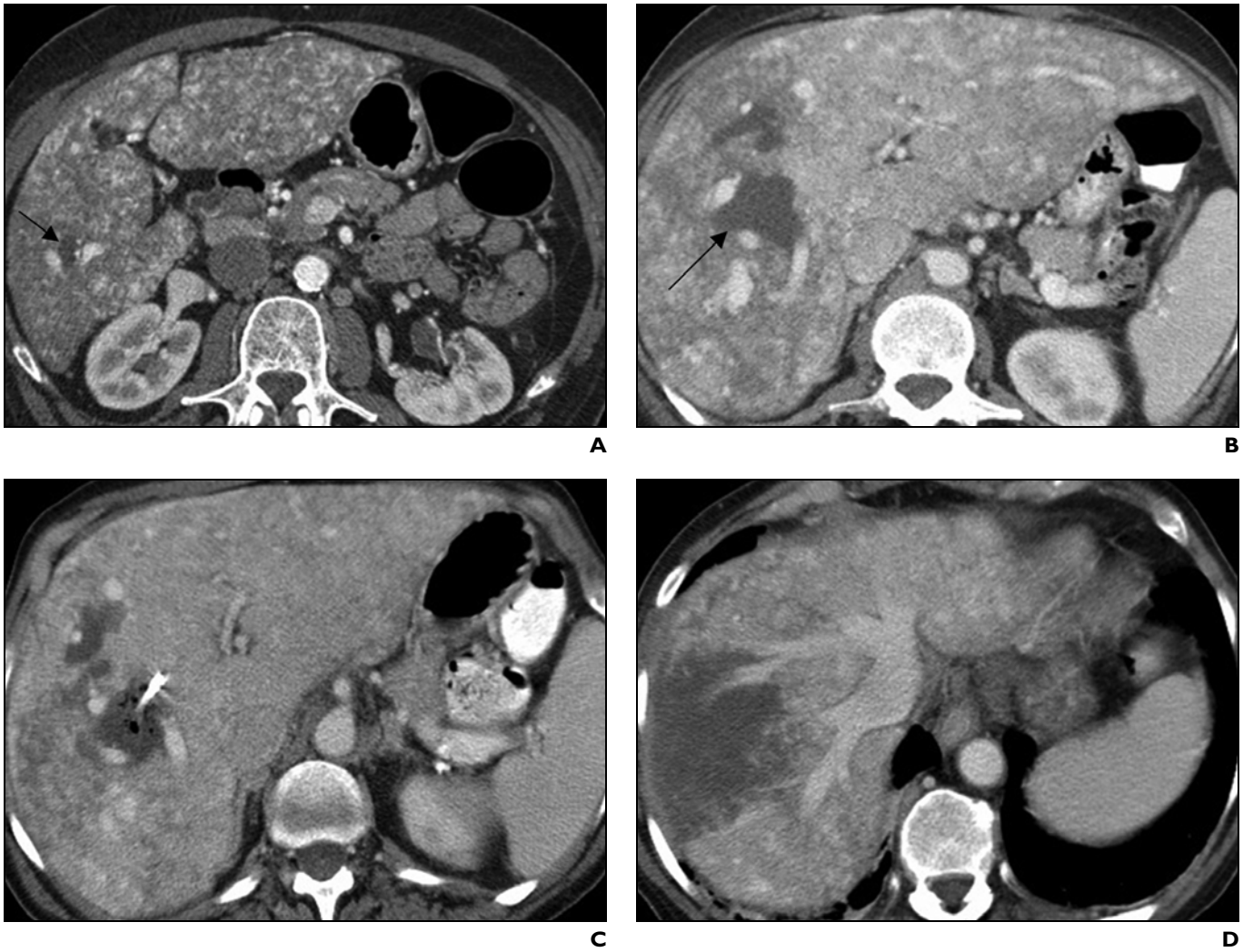


Fig. 7—Multiple axial CT angiography images in 70-year-old woman with hereditary hemorrhagic telangiectasia and liver disease show progression of biliary disease.
A, Baseline study shows characteristic heterogeneous hepatic parenchyma and small biliary cyst in right hepatic lobe (*arrow*).
B, One month after **A**, large biliary cyst lake (*arrow*) has developed.
C, One month after **B**, biliary collection has become infected despite biliary drainage and antimicrobial therapy.
D, Four days after **C**, biliary collection has markedly increased. Patient died shortly after last study.

A specific type of vascular shunt pattern could be visualized on CT in 71% of the cases. The most common shunt visualized was the arteriovenous type (13/24); however, it was not significantly different among the different clinical subtypes. The arterioportal shunt was observed more frequently in the portal hypertension clinical type (4/6 or 67%) than in the other types (4/16 of patients with heart failure and in neither of the patients with biliary symptoms). Nevertheless, no strong correlation between the predicted and actual clinical subtypes was seen, as determined on multiphasic CT. In only 13 patients (54%) did the predicted disease subtype match the clinical subtype.

Another interesting result is that 11/24 (46%) of our symptomatic patients had biliary imaging abnormalities that, except in two patients, were not accompanied by biliary symptoms (e.g., abdominal pain, jaundice, biliary sepsis). Biliary dilation and cysts have been poorly documented in prior imaging studies [9, 22], perhaps because most prior studies were performed in asymptomatic patients. Ianora et al. [11] and Ravard et al. [22] found no biliary abnormalities when evaluating 70 and 24 HHT patients, respectively, for possible liver disease with multiphasic CT. Only 4 of the 70 patients in the study by Ianora et al. and 4 of 24 patients in the study of Ravard et al. were symptomatic. Perhaps the presence of biliary abnormalities occurs later in the disease process at a time when the degree of liver ischemia is greater. Biliary disease is probably caused by the shunting of blood away from the peribiliary plexus, leading to biliary ischemia and biliary strictures (Fig. 7).

The limitations of our study are those inherent to any retrospective study performed in a relatively small number of patients. However, ours is a large series when considering the rarity of this entity. The shifting presentations that frequently occur in these patients may have also limited our results because we correlated only the initial presentation with the CT findings.

Our results indicate that although multiphasic CT may be limited in predicting the clinical subtype of HHT liver disease, it is useful in providing a diagnosis of liver disease secondary to HHT. The presence of both diffuse hepatic telangiectases and a dilated common hepatic artery can be considered pathognomonic of this disease process, particularly when accompanied by a compatible clinical picture. The presence of arterioportal shunting is more common in patients who have symptoms of portal hypertension. Finally, biliary imaging abnormalities appear to be more common in symptomatic than in reportedly asymptomatic patients and may be indicative of more advanced disease.

References

1. Guttmacher AE, Marchuk DA, White RI. Hereditary hemorrhagic telangiectasia. *N Engl J Med* 1995; 333:918–924
2. Megbie ME, Wallace GM, Shovlin CL. Hereditary haemorrhagic telangiectasia (Osler-Weber-Rendu syndrome): a view from the 21st century. *Postgrad Med J* 2003; 79:18–24
3. Larson A. Liver disease in hereditary hemorrhagic telangiectasia. *J Clin Gastroenterol* 2003; 36:149–158
4. Buscarini E, Buscarini L, Danesino C, et al. Hepatic vascular malformations in hereditary hemorrhagic telangiectasia: Doppler sonographic screening in a large family. *J Hepatol* 1997; 26:111–118
5. Bernard G, Mion F, Henry L, Plauchu H, Paliard P. Hepatic involvement in hereditary hemorrhagic telangiectasia: clinical, radiological, and hemodynamic studies in 11 cases. *Gastroenterology* 1993; 105:482–487
6. Naganuma H, Ishida H, Niizawa M, Igarashi K, Shioya T, Masamune O. Hepatic involvement in Osler-Weber-Rendu disease: findings on pulsed and color Doppler sonography. *AJR* 1995; 165:1421–1425
7. Buscarini E, Buscarini L, Giuseppe C, Arruzzoli S, Bossalini G, Piantanida M. Hepatic vascular malformations in hereditary hemorrhagic telangiectasia: imaging findings. *AJR* 1994; 163:1105–1110
8. Sawabe M, Arai T, Esaki Y, Tsuru M, Fukazawa T, Takubo K. Three-dimensional organization of the hepatic microvasculature in hereditary hemorrhagic telangiectasia. *Arch Pathol Lab Med* 2001; 125:1219–1223
9. Garcia-Tsao G, Korzenik JR, Young L, et al. Liver disease in patients with hereditary hemorrhagic telangiectasia. *N Engl J Med* 2000; 343:931–936
10. Henderson JM, Liechty EJ, Jahnke RW. Liver involvement in hereditary hemorrhagic telangiectasia. *J Comput Assist Tomogr* 1981; 5:773–776
11. Ianora AA, Memeo M, Sabba C, Cirulli A, Rotondo A, Angelelli G. Hereditary hemorrhagic telangiectasia: multidetector row helical CT assessment of hepatic involvement. *Radiology* 2004; 230:250–259
12. Caselitz M, Bahr MJ, Bleck JS, et al. Sonographic criteria for the diagnosis of hepatic involvement in hereditary hemorrhagic telangiectasia (HHT). *Hepatology* 2003; 37:1139–1146
13. Weinreb J, Kumari S, Phillips G, Pochaczevsky R. Portal vein measurements by real-time sonography. *AJR* 1982; 139:497–499
14. Miller FJ, Whiting JH, Korzenik JR, White RI. Caution with the use of hepatic embolization in the treatment of hereditary hemorrhagic telangiectasia. *Radiology* 1999; 213:928–930
15. Whiting JH, Korzenik JR, Miller FJ, Pollak JS, White RI. Fatal outcome after embolotherapy for hepatic arteriovenous malformations of the liver in two patients with hereditary hemorrhagic telangiectasia. *J Vasc Interv Radiol* 2000; 11:855–858
16. Azoulay D, Precetti S, Emile JF, et al. Liver transplantation for intrahepatic Rendu-Osler-Weber's disease: the Paul Brousse Hospital experience. *Gastroenterol Clin Biol* 2002; 26:828–834
17. Ralls PW, Johnson MB, Radin DR, Lee KP, Boswell WD. Hereditary hemorrhagic telangiectasia: findings in the liver with color Doppler sonography. *AJR* 1992; 159:59–61
18. Martini GA. The liver in hereditary haemorrhagic telangiectasia: an inborn error of vascular structure with multiple manifestations—a reappraisal. *Gut* 1978; 19:531–537
19. Pepper GM, Brenner SM, Rodriguez C, Sprayregen S, Burack B. Portosystemic encephalopathy resulting from liver involvement in hereditary hemorrhagic telangiectasia. *N Y State J Med* 1981; 81:209–212
20. Wanless IR, Gryfe A. Nodular transformation of the liver in hereditary hemorrhagic telangiectasia. *Arch Pathol Lab Med* 1986; 110:331–335
21. Feizi O. Hereditary hemorrhagic telangiectasia presenting with portal hypertension and cirrhosis of the liver. *Gastroenterology* 1972; 63:660–664
22. Ravard G, Soyer P, Boudiaf M, et al. Hepatic involvement in hereditary hemorrhagic telangiectasias: helical computed tomography features in 24 patients. *J Comput Assist Tomogr* 2004; 28:488–495
23. Memeo M, Stabile Ianora AA, Scardapane A, Buonamico P, Sabba C, Angelelli G. Hepatic involvement in hereditary hemorrhagic telangiectasia: CT findings. *Abdom Imaging* 2004; 29:211–220