Hepatic artery aneurysm: Factors that predict complications

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Objective: **We reviewed the Mayo Clinic experience with management and outcome of hepatic artery aneurysms (HAA).**
Methods: **Retrospective review of charts for 306 patients with true visceral aneurysm diagnosed from 1980 to 1998 enabled identification of 36 patients (12%) with HAA.**
Results: **Patients with HAA included 23 men and 13 women, with mean age of 62.2 years (range, 20-85 years). Most aneurysms were extrahepatic (78%) and single (92%). Mean aneurysm diameter at presentation was 3.6 cm (range, 1.5-14 cm). Five aneurysms had ruptured (14%), and four were symptomatic (11%). Mortality from rupture was 40%. Of the 9 patients with ruptured or symptomatic aneurysms, 2 patients had multiple HAA, 3 patients had fibromuscular dysplasia, and 2 patients had polyarteritis nodosa. All five HAAs that ruptured were of nonatherosclerotic origin ($P = .001$). Fourteen patients (39%) underwent elective procedures, including excision with vein graft (n = 7), excision with dacron graft (n = 3), excision alone (n = 2), and percutaneous embolization (n = 2). Two vein grafts and one dacron graft became occluded within 1 year. Nonoperative management was elected in 22 patients (61%) with mean aneurysm diameter 2.3 cm (range, 1.5-5 cm). No complications related to the aneurysm occurred during mean follow-up of 68.4 months (range, 1-372 months). Aneurysm growth was identified in 27%, the greatest being 0.8 cm over 34 months.**
Conclusions: **HAA are at definite risk for rupture (14%). Risk factors for rupture include multiple HAA and nonatherosclerotic origin. Patients with symptomatic aneurysms or any of these risk factors should be considered for intervention. (J Vasc Surg 2003;38:41-5.)**

Hepatic artery aneurysms (HAA) are rare and represent approximately 20% of all visceral aneurysms.[1-6] Historically, most aneurysms had ruptured at presentation or were incidentally discovered at autopsy. The recent advances in and rapid proliferation of cross-sectional imaging has enabled identification of HAA earlier in their natural history. Over the last two decades the treatment options for ruptured or symptomatic HAA have evolved to include embolization. However, because of the rarity of these aneurysms, optimal management of asymptomatic HAA remains controversial. The risk-benefit ratio of treating asymptomatic HAA is difficult to assess in the absence of knowledge about the risk factors associated with rupture. In addition, most data available on HAA are derived from series with a small cohort of subjects with a combination of both true and false aneurysms.

We previously reported 12 HAA treated surgically at Mayo Clinic Rochester between 1979 and 1991.[7] In the current study we report our experience with true HAA managed operatively and nonoperatively at our institutions over the last two decades.

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PATIENTS AND METHODS

A retrospective review was undertaken of charts for all patients with documented HAA seen at the Mayo Clinic (Rochester, Minn; Scottsdale, Ariz; Jacksonville, Fla) between January 1, 1980, and December 31, 1998. The study was reviewed and approved by our institutional review board. Included for review were charts for patients with HAA diagnosed with imaging studies, surgical exploration, or autopsy evaluation. Excluded from review were charts for patients with hepatic artery pseudoaneurysms. The records for all patients were analyzed with regard to demographic data, comorbid conditions, clinical presentation, aneurysm characteristics, imaging studies, management including operative intervention, and outcome. Long-term follow-up was obtained via chart review or by direct phone contact of the patient or relative. No specific protocol was followed for management of these aneurysms; surgeon preference and judgment dictated all management decisions.

The rate of atherosclerosis and of other variables of interest, ie, nonatherosclerotic aneurysm origin, was compared between two groups: ruptured versus nonruptured aneurysms. Statistical significance was assessed with the Fisher exact test. Margin of error was determined by generating 95% confidence intervals around the difference of group rates.

Table I. Frequency of associated aneurysms

	All aneurysms (n = 36 patients)		*Asymptomatic aneurysms (n = 27 patients)*		*Symptomatic aneurysms (n = 9 patients)*	
	n	*%*	*n*	*%*	*n*	*%*
Visceral aneurysms	11	31	10	37	1	11
Splenic	7	20	7	23	0	
Celiac	4	11	4	13	0	
SMA	3	8	2	7	1	11
Gastric	1	3	1	3	0	
Gastroduodenal	1	3	1	3	0	
Nonvisceral aneurysms	15	42	10	37	5	55
Abdominal aorta	7	20	5	19	2	22
Iliac	6	17	6	20	0	
Renal	6	17	3	11	3	33
Thoracic aorta	4	11	3	11	1	11
Carotid	2	6	1	3	1	11
Cerebral	1	3	0		1	11
Family history of aneurysm	1	3	1	3	0	

Table II. Method of initial diagnosis

	Patients	
Diagnostic method	*n*	*%*
Computed tomography	14	38
Arteriography	9	25
Ultrasound	5	14
Plain radiography	3	8
Incidental finding at operation	2	6
Operation because of rupture	2	6
Finding at autopsy	1	3

RESULTS

Demographic data and associated conditions. Of 306 patients with a diagnosis of visceral artery aneurysm seen at our institutions between 1980 and 1998, 36 patients (12%) had HAA. Twenty-three patients (64%) were men, and 13 patients (36%) were women. Mean patient age at presentation was 62.2 years (range, 20-85 years).

Hypertension was the most common comorbid condition in patients with HAA (72%), followed by malignancy (33%), peptic ulcer disease or gastritis (28%), coronary artery disease (22%), peripheral vascular disease (14%), chronic obstructive pulmonary disease (11%), and obesity (11%). Fibromuscular dysplasia was present in 6 patients (17%), polyarteritis nodosa in 2 patients (6%), and scleroderma in 1 patient (3%). Tobacco and alcohol use was documented in 67% of patients.

Incidence of concomitant aneurysms in patients with HAA is summarized in Table I. Nonhepatic visceral artery aneurysms were present in 31% of patients, with splenic artery aneurysm being the most common type (20%). Nonvisceral aneurysms were documented in 42% of patients; abdominal aortic aneurysm occurred most frequently (20%).

Clinical presentation and diagnosis. Twenty-seven of 36 patients with HAA (75%) had no symptoms. Five patients (14%), 3 men (13%) and 2 women (15%), had ruptured HAA at presentation. Mean age of patients with ruptured aneurysms was 57.6 years (range, 20-85 years). In the group with ruptured aneurysms, 2 patients each had multiple hepatic aneurysms, polyarteritis nodosa, or fibromuscular dysplasia, and 1 patient had a history of endocarditis; 3 patients in this group had concomitant nonhepatic aneurysms. All five ruptured aneurysms were of nonatherosclerotic origin. Rupture did not occur in the 26 patients with atherosclerotic HAA ($P = .001$). Two of 5 patients with ruptured aneurysms had polyarteritis nodosa (40%), whereas none of 31 patients without ruptured aneurysms had polyarteritis nodosa ($P = .01$).

Four patients (11%), 3 men and 1 woman, had symptomatic but nonruptured aneurysms at presentation. Mean age of patients with symptoms was 49.3 years (range, 20-74 years). Three patients had abdominal pain, and 1 patient had obstructive jaundice. In this group with symptoms, fibromuscular dysplasia was present in 1 patient, and 3 patients had concomitant aneurysms.

Methods used for the initial diagnosis are summarized in Table II. The diagnosis was established at computed tomography in 14 patients, arteriography in 9 patients, and ultrasound scanning in 5 patients. In 2 patients HAA was diagnosed incidentally during abdominal surgery performed because of another condition. In 2 patients ruptured HAA was documented during emergent exploration. One patient died on arrival at the clinic, and the diagnosis of ruptured HAA was confirmed at autopsy.

Aneurysm characteristics. Anatomic characteristics of HAA are summarized in Table III. A solitary aneurysm was present in 33 patients (92%). Multiple hepatic aneurysms were present in 3 patients (8%); 2 of these patients had ruptured aneurysms at presentation (67%), and both had polyarteritis nodosa. An extrahepatic aneurysm was

present in 28 patients (78%). Seven patients (20%) had mixed aneurysm, with both intrahepatic and extrahepatic components. Only 1 patient had an isolated intrahepatic aneurysm. The size and degree of calcification of the aneurysm was documented in 30 patients (83%). Calcification was noted in 14 patients (47%). Mean HAA diameter was 3.6 cm (range, 1.5-14 cm). Aneurysm diameter (10 cm) was available for only one ruptured aneurysm. In the symptomatic group, mean HAA diameter was 3.5 cm (range, 2-4.5 cm).

Operative management and outcome. Fourteen patients (39%) underwent interventions, emergency procedures because of HAA rupture in 3 patients and elective operations in 11 patients. Mean aneurysm diameter in the elective group was 5.7 cm (range, 1.5-14 cm). In the group who underwent elective procedures, 5 patients underwent repair because of aneurysm size, 4 patients underwent interventions because of symptoms, and 2 patients underwent aneurysm repair during abdominal operations performed because of other indications.

The most common procedure was excision of the aneurysm with saphenous vein grafting (n = 7), followed by excision with dacron grafting (n = 3) and excision and ligation of the aneurysm (n = 2). Successful embolization was undertaken in 1 patient with a ruptured aneurysm and 1 patient with a symptomatic aneurysm. Operative mortality was 33% in the group with ruptured aneurysms and 0% in group who underwent elective surgery. Overall complication rate was 43% (100% in the rupture group vs 28% in the elective group). In the rupture group, 1 patient had multiple organ failure, with gangrenous cholecystitis, and died; 1 patient had a duodenal leak, intrabdominal abscess, short gut syndrome, and deep venous thrombosis of the lower extremities; and 1 patient had a urinary tract infection. In the elective group, 1 patient had ileus, 1 patient had acute myocardial infarction and pneumonia, and 1 patient had pneumonia and catheter sepsis.

Mean follow-up was 47.6 months (range, 1-192 months) in the surgical group. Ten patients (77%) were alive and well at last follow-up. Three patients died during the study, 1 each of esophageal carcinoma, ovarian carcinoma, and ruptured carotid artery aneurysm (initial presentation, ruptured HAA). Graft occlusion was documented in 2 patients with a saphenous vein graft (28.6%) and 1 patient with a dacron graft (33.3%). In 1 patient with saphenous vein graft occlusion repeat surgery was performed, with successful reconstruction with the splenic artery. No risk factors for graft occlusion were identified.

Nonoperative management and outcome. Nonoperative management was chosen in 22 patients (61%). Included in this group were 2 patients with ruptured HAA at presentation. One patient was dead on arrival; the other remained stable, with a contained hematoma due to intra-abdominal scarring from previous gastric resection, and was alive at 3-month follow-up. Mean follow-up in this group was 68.4 months (range, 1-372 months). Data regarding aneurysm size were available for 18 patients (82%). Four of these had aneurysms 3 cm or greater in diameter, ie, 3.0, 3.0, 3.5, and 5.0 cm, respectively; in the remaining 14 patients aneurysm diameter was 2 cm or less. Mean diameter of nonruptured HAA was 2.3 cm (range, 1.5-5 cm). Of the 11 patients with aneurysm size documented at follow-up, HAA interval growth was noted in 3 patients (27%), ie, 0.8 cm in 34 months, 0.5 cm in 57 months, and 0.3 cm in 70 months, respectively. In all 3 of these patients management was nonoperative, because aneurysm diameter was less than 2.5 cm in 2 patients and because of chronic lymphocytic leukemia and high operative risk in the other patient, with aneurysm diameter of 3.5 cm. Three patients died of causes unrelated to the aneurysm, 1 each of complications of scleroderma (at 132 months), pancreatic cancer (at 6 months), and gastrinoma and pulmonary embolism (at 17 months). The remaining patients were alive at follow-up, with no rupture or other complications related to the aneurysm.

Table III. Anatomic characteristics of HAA

	All patients	
HAA characteristic	*n*	*%*
Single	33	92
Multiple	3	8
Extrahepatic	28	78
Intrahepatic	1	3
Extrahepatic and intrahepatic	7	20
Calcification	14	47
Diameter (cm)		
Mean	3.6	
Range	1.5-14	

HAA, Hepatic artery aneurysms.

DISCUSSION

Incidence. Although rare, HAA is the second most common type of visceral aneurysm after splenic artery aneurysm.[1,2,8] The true incidence of HAA is unknown. Of 2,091,965 patients seen at the Mayo Clinic between 1980 and 1998, HAA was identified in only 36 patients, yielding an incidence of 0.002%. HAA represented 12% of all visceral aneurysms seen at our institutions during that period. This aneurysm is more common in men.[2,5-11] In our study, most HAA were extrahepatic, a finding consistent with the reported incidence of 75% to 80%.[1-4,6,7,9,10,12,13] Most HAA are solitary[1,8]; we found multiple HAA in only 3 of our patients (8%).

Cause of HAA. The precise cause of HAA remains unclear. In the early twentieth century, most HAA were mycotic, with bacterial endocarditis the most frequent cause.[2,12] Today mycotic HAA are seldom seen, because of earlier antibiotic treatment of infections.[9,12] Only 1 patient in our cohort had a history of infective endocarditis. Several conditions have been associated with HAA, including atherosclerosis, arterial fibrodysplasia, vasculitis, polyarteritis nodosa, and systemic lupus erythematosus.[1,2,13-15] Rarely

HAA has been associated with disorders such as Takayasu arteritis, Kawasaki disease, von Recklinghausen neurofibromatosis, and Wegener granulomatosis.[11,13,16,17] Long-term oral amphetamine use has been implicated as a possible cause of HAA.[18] Congenital causes of HAA include Marfan syndrome, Ehlers-Danlos syndrome, Osler-Weber-Rendu syndrome, and hereditary hemorrhagic telangiectasia.[10,19]

Risk for rupture. Previous studies have indicated that most patients with HAA have symptoms or rupture at presentation.[1] Aneurysm rupture has been reported in 21% to 80% of all HAA.[1-3,6,8,10] In our series the overall incidence of rupture was 14%, lower than that in previous reports. Furthermore, the incidence of symptomatic aneurysm without rupture in our study was 11%. Most of our patients (75%) had no asymptomatic aneurysms.

Risk factors associated with rupture of HAA were poorly defined in the past. Establishment of a relationship between aneurysm size and risk for rupture has been difficult because of the rarity of HAA. In our study the smallest symptomatic HAA was 2 cm in diameter (mean, 3.5 cm). Size data were available for only one aneurysm in the rupture group (10 cm). However, rupture of HAA in the 2 cm range has been reported previously.[10]

An important finding of our study was increased risk for rupture or symptoms in patients with HAA of nonatherosclerotic origin. Of the 9 patients with ruptured or symptomatic HAA at presentation, 3 patients had fibromuscular dysplasia, 2 patients had polyarteritis nodosa, and 1 patient had a history of endocarditis. All ruptured aneurysms were of nonatherosclerotic cause. Therefore there was a 50% rupture rate in the 10 aneurysms with a cause other than atherosclerosis. Also, we noted that patients with polyarteritis nodosa were significantly more likely to have ruptured aneurysms at presentation. Several reports have previously documented ruptured or symptomatic HAA in patients with fibromuscular dysplasia, polyarteritis nodosa, von Recklinghausen neurofibromatosis, and Wegener granulomatosis.[10,11,14,17,20] Another important finding was that 2 of 3 patients with ruptured aneurysms had multiple HAA, and both patients had polyarteritis nodosa. Therefore we do not know whether polyarteritis nodosa or the presence of multiple aneurysms was the more important risk factor for rupture. These observations have been reported by others.[9,11,14,20] Mortality from rupture in our study was 40%, consistent with other studies (21%-43%).[1,6,8,21]

Indications for treatment. In the past, most authors have recommended repairing HAA, whether symptomatic or not, because of the risk for rupture and associated death.[10,12,22,23] While symptomatic and ruptured HAA require intervention, not all asymptomatic aneurysms warrant repair. Our study demonstrated that a selective approach can be used to manage HAA. On the basis of our findings, we recommend intervention for all nonatherosclerotic aneurysms and for multiple hepatic aneurysms. Intervention in these subgroups is warranted because of the high incidence of rupture and symptoms documented in our study. While no consensus exists regarding size criteria for intervention in asymptomatic atherosclerotic HAA, we believe it reasonable to consider surgical repair or embolization of aneurysms larger than 2 cm in a patient who is at reasonable operative risk and has a life expectancy of more than 2 years.

For asymptomatic atherosclerotic HAA 2 to 5 cm in diameter in patients with marginal health, treatment options are more controversial. We did not intervene in 4 patients with aneurysms 3 to 5 cm in diameter, and the aneurysms did not rupture. This suggests that intervention in these patients should probably be reserved for those with aneurysms that enlarge or become symptomatic. However, treatment decisions in this group of patients are difficult and must be made on an individual basis, because good data are lacking. In most patients with aneurysms larger than 5 cm some type of intervention to be undertaken to remove the risk for rupture.

Treatment options. In 1903 Kehr reported the first successful ligation of HAA[24]. Since then, several operative techniques have evolved for treatment of HAA. Appropriate therapy depends primarily on location of the aneurysm, presence of collateral flow, operative risk, and clinical status. Open surgical treatment options for HAA include ligation, excision, venous grafting, synthetic grafting, and hepatic resection.[1,2,4,8,13,23] Intrahepatic aneurysms can be treated with resection, ligation, or embolization.[3,10] Laparoscopic surgical techniques may also be useful for ligation of HAA, inasmuch as this minimally invasive technique has been used successfully for splenic artery ligation.[25]

Embolization is an alternative to open repair and has become increasingly popular.[5,15,17,22,26] The technique has an important role in management of intrahepatic aneurysms and in surgical candidates at high risk. Our limited experience with embolization in 2 patients was successful. This technique however, is associated with potential short-term complications, eg, hepatic ischemia, hepatic abscess, cholecystitis, and long-term concerns about recanalization of the aneurysm.[15,22,27,28]

CONCLUSIONS

Although rare, HAA are at definite risk for rupture. Not all HAA need intervention, and small (<2 cm) HAA may safely be observed. Risk factors for complications include multiple HAA or nonatherosclerotic origin, in particular, polyarteritis nodosa. Patients with symptomatic aneurysm or any of these risk factors should be considered for intervention. For patients with atherosclerotic HAA greater than 2 cm, the decision to intervene is more controversial. It is reasonable to recommend repair of HAA greater than 2 cm in a patient who is at good operative risk and has a life expectancy greater than 2 years. In patients with marginal or poor health with a 2 to 5 cm HAA, careful observation may be warranted, but the decision to intervene must be made on an individual basis. HAA greater than 5 cm should generally undergo repair, embolization, or ligation.

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REFERENCES

1. O'Driscoll D, Olliff SP, Olliff JFC. Hepatic artery aneurysm. Br J Radiol 1999;72:1018-25.
2. Lal RB, Strohl JA, Piazza S, Aslam M, Ball D, Patel K. Hepatic artery aneurysm. J Cardiovasc Surg 1989;30:509-13.
3. Salcuni PF, Spaggiari L, Tecchio T, Benincase A, Azzarone M. Hepatic artery aneurysm: an ever present danger. J Cardiovasc Surg 1995;36: 595-9.
4. Srivastava DN, Chakravarti AL, Gupta RK, Gujral RB. Gastrointestinal bleeding from a false aneurysm of the hepatic artery after cholecystectomy. Am J Gastroenterol 1996;91:395-7.
5. Abboud B, Ghossain A, Tohme C, Atallah N, Abi Ghanem S, Farah P. Aneurisme de l'artere hepatique traite par embolisation [French]. J Chir 1994;131:252-6.
6. Liou TC, Ling CC, Pang KK. Liver abscess concomitant with hemobilia due to rupture of hepatic artery aneurysm: a case report. Hepatogastroenterology 1996;43:241-4.
7. Dougherty MJ, Gloviczki P, Cherry KJ, Bower TC, Hallett JW, Pairolero PC. Hepatic artery aneurysms: evaluation and current management. Int Angiol 1993;12:178-84.
8. Andrew DR, Vive JU, Macpherson DS. Successful resection of a massive hepatic artery aneurysm. J R Army Med Corps 1994;140:138-40.
9. Lewis DR, Kung H, Connon JJ. Biliary obstruction secondary to hepatic artery aneurysm: cholangiographic appearance and diagnostic considerations. Gastroenterology 1982;82:1446-51.
10. Erskine JM. Hepatic artery aneurysm. Vasc Surg 1973;7:106-25.
11. Den Bakker MA, Tangkau PL, Steffens TW, Tjiam L, van del Loo EM. Rupture of a hepatic artery aneurysm caused by Wegener's granulomatosis. Pathol Res Prac 1997;193:61-6.
12. Senocak F, Cekirge S, Senocak ME, Karademir S. Hepatic artery aneurysm in a 10-year-old boy as a complication of infective endocarditis. J Pediatr Surg 1996;31:1570-2.
13. Caputo AE, Roberts WN, Yee YS, Posner MP. Hepatic artery aneurysm in corticosteroid-treated, adult Kawasaki's disease. Ann Vasc Surg 1991;5:533-7.
14. Chan RJ, Goodman TA, Aretz TH, Lie JT. Segmental mediolytic arteriopathy of the splenic and hepatic arteries mimicking systemic necrotizing vasculitis. Arthritis Rheum 1998;41:935-8.
15. Hidalgo F, Narvaez JA, Rene M, Dominguez J, Sancho C, Montanya X. Treatment of hemobilia with selective hepatic artery embolization. JVIR 1995;6:793-8.
16. Koyama M, Tanaka M, Shimizu M, Nomura S, Kako N, Suzuki S, et al. Surgical treatment of mesenteric infarction, thoracoabdominal aortic aneurysm, and proper hepatic aneurysm in a middle-aged woman with Takayasu's arteritis. J Cardiovasc Surg 1995;36:337-41.
17. Hassen-Khodja R, Declemy S, Batt M, Castanet J, Perri C, Ortonne JP, et al. Visceral artery aneurysms in von Recklinghausen's neurofibromatosis. J Vasc Surg 1997;25:572-5.
18. Welling TH, Williams DM, Stanley JC. Excessive oral amphetamine use as a possible cause of renal and splanchnic arterial aneurysms: a report of two cases. J Vasc Surg 1998;28:727-31.
19. Cooper SG, Richman AH. Spontaneous rupture of a congenital hepatic artery aneurysm. J Clin Gastroenterol 1988;10:104-7.
20. Jones HJ, Staud R, Williams RC. Rupture of a hepatic artery aneurysm and renal infarction: two complications of fibromuscular dysplasia that mimic vasculitis. J Rheumatol 1998;25:2015-8.
21. Chen MF, Chou FF, Wang CH, Jang YI. Hematobilia from ruptured hepatic artery aneurysm. Arch Surg 1983;118:759-61.
22. Tarazov PG, Ryzhkov VK, Polysalov VN, Prozorovskij KV, PolyKarpov AA. Extraorganic hepatic artery aneurysm: failure of transcatheter embolization. HPB Surg 1998;11:55-60.
23. Mays ET, Conti S, Fallahzadeh H, Rosenblatt M. Hepatic artery ligation. Surgery 1979;86:536-43.
24. Guida PM, Moore SW. Aneurysm of the hepatic artery: Report of five cases with a brief review of the previously reported cases. Surgery 1966;60:299-310.
25. Arca MJ, Gagner M, Heniford BT, Sullivan TM, Beven EG. Splenic artery aneurysms: methods of laparoscopic repair. J Vasc Surg 1999;30: 184-8.
26. Tsai CH, Chiou CY, Ko QY, Hwang HS, Hwang MH, Yang CT, et al. Therapeutic embolization of post-cholecystectomy hepatic artery. Hepatogastroenterology 1992;39:158-60.
27. Lumsden AB, Mattar SG, Allen RC, Bacha EA. Hepatic artery aneurysms: the management of 22 patients. J Surg Res 1996;60:345-50.
28. Steiner Z, Brown RA, Jamieson DH, Millar AJW, Cywes S. Management of hemobilia and persistent biliary fistula after blunt liver trauma. J Pediatr Surg 1994;29:1575-7.

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