의학석사 학위논문

간 이식 수혜자에서 발견되는 비장동맥류의 임상적 특징 및 예후

Clinical Characteristics and Outcomes of Incidental Splenic artery aneurysms in Liver Transplanted Recipients

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이 논문을 의학석사학위 논문으로 제출함

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ABSTRACT

Background. PHTN is a known risk factor for SAA development and rupture. This study aimed to identify LT recipients who incidentally have SAA and PHTN, and analyze their clinical characteristics and treatment outcomes.

Methods. We conducted a retrospective review of LT recipients at our institution from January 2016 to December 2020. Patients with concurrent SAA were identified using 4-phase dynamic CT angiography. We collected data on SAA characteristics, SAA treatment methods, patient demographics, and liver disease-related features. We assessed changes in SAA size through the most recent CT scans performed at least 6 months after the SAA treatment.

Results. Of 2,127 LT recipients, 104 patients had incidental SAA, with a prevalence rate of 4.8%. The final analysis included 97 patients with 142 SAA occurrences. In our study, SAAs were predominantly located near the hilum and the distal third portion. Most SAAs were of the saccular type with an average diameter of 1.82 cm. An analysis of the patients divided by SAA diameter showed that those with larger SAA sizes had a higher prevalence of hypertension (p=0.013) and a longer duration of liver disease (p=0.005). The average follow-up period was 52.4 months, and only one patient experienced SAA-related complications.

Conclusions. Our results show that SAA accompanied by PHTN mainly exhibits the characteristics of saccular types located distally and at the hilum. Hypertension and the duration of liver diseases were associated with an SAA size.

Keywords: Splenic artery aneurysm; Portal hypertension; Liver transplantation recipients

Contents

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ABBREVIATIONS

BMI, body mass index; CAD, coronary artery disease; CDLT, cadaveric donor liver transplantation; carotid a. ds, carotid artery disease (stenosis or occlusion); CT, computed tomography; CTP, Child-Pugh score; DM, diabetes mellitus; HBV, hepatitis B virus; HCC, hepatocellular carcinoma; HCV, hepatitis C virus; HTN, hypertension; LC, liver cirrhosis; LDLT, living donor liver transplantation; LT, liver transplantation; MELD, Model for End-Stage Liver Disease; PAOD, peripheral artery occlusive disease; PHTN, portal hypertension; SAA, splenic artery aneurysm; SBP, spontaneous bacterial peritonitis

INTRODUCTION

Splenic artery aneurysm (SAA) has a low prevalence rate in the general population, at 0.8%.[1] However, among splanchnic artery aneurysms, it is the most common type.[2] The pathophysiology of SAA is not clearly elucidated. According to Stanley et al.,[3] the pathophysiology is classified into arterial dysplasia, portal hypertension (PHTN) with splenomegaly, focal arterial inflammatory processes, hormonal and hemodynamic events in parous women, and unrecognized pathogenic factors such as suspected arteriosclerosis. In Western populations, it is more prevalent in parous women,[4] while studies in Japan have often observed a significant association with the occurrence of PHTN.[5] Most cases are asymptomatic, and they are often discovered incidentally.[2] According to recent guidelines, in patients with SAA who are asymptomatic and show stable size, observation is recommended for those with a diameter of less than 3 cm.[6] The rupture of an SAA is a life-threatening complication with a high mortality rate.[7, 8] Known risk factors for the occurrence and rupture of SAA include pregnancy and PHTN.[4, 9] In the case of women of childbearing age, treatment is recommended regardless of size, but no specific treatment criteria are recommended for patients with PHTN. Management of SAA is recommended for patients undergoing liver transplantation to prevent rupture as a fatal complication.[6, 10, 11] In this study, we aimed to identify patients who had incidental SAA with PHTN, analyze their demographics and clinical characteristics, and investigate whether there are differences in outcomes based on treatment. Our objective was to offer clinical data that could support the development of treatment guidelines for individuals with SAA who have PHTN.

MATERIALS AND METHODS

In this research, we aimed to identify individuals with SAA who were also affected by PHTN. To achieve this, we conducted a comprehensive analysis of adult patients aged 18 and older who underwent liver transplantation at our institution between January 2016 and December 2020, specifically focusing on those with concurrent SAA. We identified individuals with concomitant SAA based on 4-phase dynamic computed tomography (CT) scans performed during the preoperative and follow-up periods. (**Figure 1.**) Patients who underwent retransplantation during this period were excluded, as well as patients with a follow-up period of less than 6 months or those without dynamic CT images beyond 6 months after intervention. (**Figure 2.**) The criteria for defining SAA were a diameter increase of 50% or more in the artery, with the measurement taken along the longest axis in the coronal or axial view on a dynamic CT scan. We categorized SAA shapes as either saccular or fusiform, and in cases of multiple aneurysms, we included up to the three largest in our analysis. The locations of the aneurysms were classified as proximal, middle, distal, hilum, or intraparenchymal along the splenic artery. Additionally, we assessed SAA classification grades[12] and the presence of thrombus. We collected demographic data on patients, including age, gender, comorbidities such as hypertension, diabetes mellitus, coronary artery disease, and peripheral artery disease, blood type, ethnicity, medication history (antiplatelet and statin use). Furthermore, we explored the etiology of liver fibrosis, the presence of hepatocellular carcinoma (HCC), the duration of liver disease before transplantation, the MELD score, CTP score, and the presence of liver disease-related features like splenomegaly or splenorenal shunt. The presence of PHTN was defined as the occurrence of one or more complications, such as ascites, esophageal varices, hepatic encephalopathy, or hepatorenal syndrome, along with evidence of liver cirrhosis.

Splenomegaly was included for cases with a length greater than or equal to 12 cm on CT imaging, and splenorenal shunt was defined as the presence of splenorenal collateral vessel enlargement confirmed on CT or angiography. We evaluated the approaches used for SAA treatment, classifying them as preoperative, intraoperative, or postoperative interventions. Subsequently, we evaluated alterations in SAA observed in the latest follow-up CT scan, performed at least 6 months after the last SAA intervention. Additionally, we divided the patients into two groups based on the mean size value and examined whether there were differences in baseline characteristics between these two groups.

Descriptive statistics for both numerical and non-numerical data were provided. Results were expressed as means ± standard deviations (SD) (ranges) or as the number (proportion) of patients. The Student t-test was used for numerical comparisons, while Pearson's chi-square test or Fisher's exact test was employed for non-numerical data. A p-value of ≤0.05 was considered statistically significant. Statistical analyses were conducted using SPSS Statistics for Windows, version 22 (IBM Corp., Armonk, NY, USA). The study received approval from our institutional review board, and informed consent was not required.

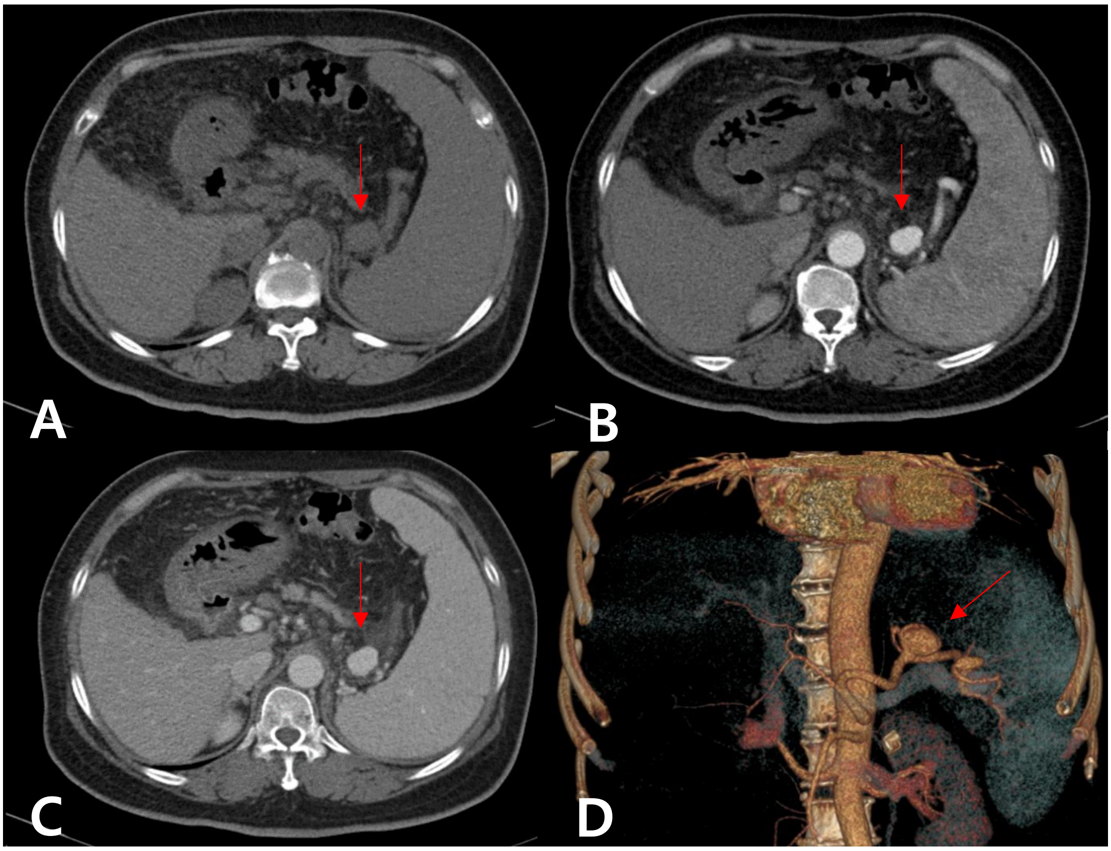

Figure 1. 2.2cm diameter Splenic artery aneurysm (arrow) on dynamic CT. (A; pre enhancement phase, B: arterial phase, C: portal phase, D: 3D reconstruction processed image)

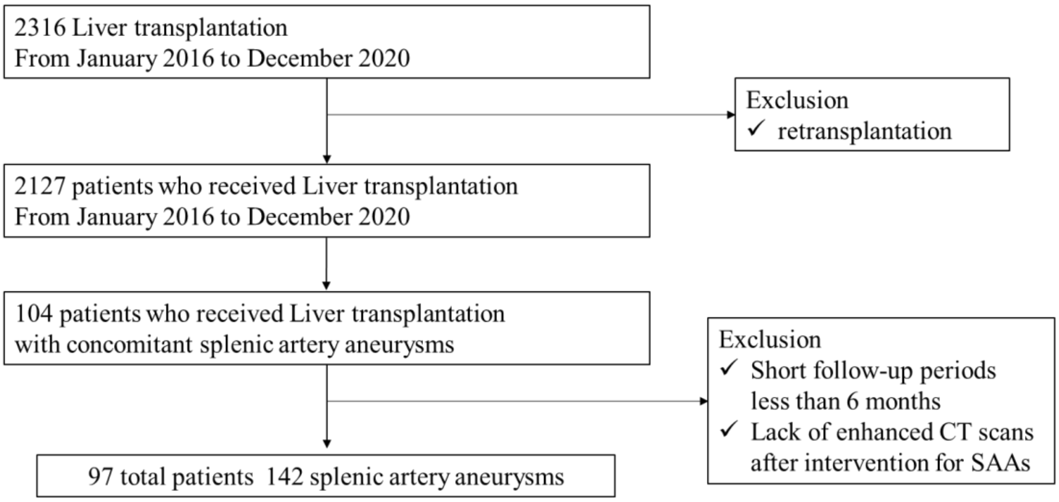

Figure 2. Flow diagram for the study population

RESULTS

Among a total of 2,127 recipients who underwent liver transplantation, excluding patients who underwent retransplantation, from January 2016 to December 2020, 104 patients were found to have incidental SAA, resulting in a prevalence rate of 4.8%. Seven out of the 104 patients were excluded due to a short follow-up period of less than 6 months or a lack of postoperative enhanced CT scans. A total of 97 patients were included in the analysis and presented with 142 SAA occurrences. The demographic characteristics and liver disease-related features of these patients are summarized in **Table 1**. The average age was 54.5 years, with females accounting for 50.5% of the cohort. Diabetes mellitus (DM) was present in 24% of patients, while hypertension (HTN) was observed in 11%. Only one patient had coronary artery disease (CAD), and there were no patients with peripheral artery disease or carotid artery disease. All patients were of Asian ethnicity, with 96 being Korean and one being Mongolian. The majority of patients (80.4%) had hepatitis B virus-related liver cirrhosis (HBV LC), followed by alcoholic liver cirrhosis in 10.3% of cases. Hepatocellular carcinoma (HCC) was concurrent in 34% of patients. The average duration of illness was 7.8 years, which was longer than the overall average of 3.76 years. The MELD score at the time of liver transplantation was 15.4 on average, and the CTP score was 8.4 on average, showing no significant difference compared to the average MELD score of 17.8 and the average CTP score of 8.53 for the entire recipient population. Ninety-five patients received living donor liver transplantation, while two received cadaveric donor liver transplantation. PHTN was present in all patients, and 82 individuals had esophageal varices, accounting for 84.5%, while 88 patients had splenomegaly, representing 88.0% of the cohort.

There were 65 patients with a single SAA, 19 with two SAAs, and 13 with three or more SAAs. Among the 97 patients, 142 SAAs were present; their characteristics are outlined in **Table 2**. Most SAAs were saccular type (130 out of 142, 91.5%), with a mean diameter of 1.82 cm. The distribution based on diameter is shown in the histogram in **Figure 3**. Most SAAs were located near the hilum (83 out of 142, 58.4%), followed by the distal third, middle third, and proximal third, and the least common was intraparenchymal SAA, which was observed in one patient as multiple aneurysms. Two SAAs had accompanying thrombus, and five had associated calcification.

Following the treatment algorithm (**Figure 4.**) established by our institution, Treatment was administered to the majority of patients with SAA both before and after liver transplantation. Out of the 97 patients, intraoperative ligation or intraoperative embolization was performed in

54 patients, which accounts for approximately half of the patients. This was followed by 26 patients who underwent embolization or stent insertion before surgery to achieve exclusion. These were cases where a history of prior peritonitis or severe retroperitoneal edema was present, making it anticipated that accessing the splenic artery during surgery would be challenging. Also included were situations where the SAA was located in the distal portion. Eight patients received intervention after surgery; seven were managed with observation without additional treatment (**Table 3**), and the follow-up period averaged 52.4 months. Only one patient experienced complications related to SAA, which was a spleen abscess that occurred after preoperative embolization. The patient, who exhibited multiple aneurysms with a maximum diameter of 4.5 cm, experienced improvement following treatment for the spleen abscess with Pigtail catheter insertion and intravenous antibiotics.

After liver transplantation, most patients underwent CT follow-up at intervals ranging from 3 to 12 months. We reviewed each patient's latest CT scan to ascertain any changes in the SAAs. Among these, 74 SAAs (52.1%) were observed without any change in size, while 59 (41.5%) showed artifacts of embolic material after embolization, making it challenging to determine changes in the aneurysm's size or the maintenance of blood flow. In the last follow-up CT, three aneurysms showed complete disappearance, and four aneurysms demonstrated a reduction in diameter by more than 0.5 cm. There was no change in size in the eight patients who were observed, and their average size was 1.51 cm. No patients developed new SAAs or aneurysmal expansion during follow-up.

Based on an average diameter cutoff of 1.82 cm, 44 SAAs (30.9%) were found to be larger than this threshold and were present in 37 patients. When we divided the 97 patients into two groups based on SAA diameter for analysis, it was observed that the group with larger diameter SAAs had a higher prevalence of hypertension (p=0.013) and a longer duration of liver disease (p=0.005).

Table 1. Demographic characteristics and liver disease–related features in LT recipients with incidental SAA

Variables	SAA cohort N = 97	Dia<1.82cm N = 60	Dia ≥1.82cm N = 37	P value*
Age, mean (SD)	54.5 (7.88)	53.9 (7.90)	55.5 (7.85)	0.330 \
Female, n (%)	49 (50.5)	30 (50.0)	19 (51.4)	0.897
BMI, kg/m2 (SD)	24.1 (3.34)	23.9 (3.17)	24.4 (3.62)	0.549
DM, n (%)	24 (24.7)	15 (25.0)	9 (24.3)	0.940
HTN, n (%)	11 (11.3)	3 (5.0)	8 (21.6)	**0.019**
CAD, n (%)	1 (1.0)	1 (1.7)	0 (0.0)	1.000
PAOD, n (%)	0			
Carotid A. ds, n (%)	0			
Blood type, n (%)				
A	33 (34.0)	19 (31.7)	14 (37.8)	
B	32 (33.0)	19 (31.7)	13 (35.1)	
AB	11 (11.3)	6 (10.0)	5 (13.5)	
O	21 (21.6)	16 (26.7)	5 (13.5)	
RH, n (%)				1.000
+	96 (99.0)	59 (98.3)	37 (100.0)	
-	1 (1.0)	1 (1.7)	0 (0.0)	

Race, n (%)				1.000
Korean	96 (99.0)	59 (98.3)	37 (100.0)	
Asian (non-Korean)	1 (1.0)	1 (1.7)	0 (0.0)	
Medication, n (%)				
Antiplatelet	2 (2.1)	2 (3.3)	0 (0.0)	0.523
Statin	2 (2.1)	2 (3.3)	0 (0.0)	0.523
Etiology, n (%)				
HBV LC	78 (80.4)	47 (78.3)	31 (83.8)	
HCV LC	3 (3.1)	3 (5.0)	0 (0.0)	
Alcoholic LC	10 (10.3)	7 (11.7)	3 (8.1)	
Cryptogenic LC	4 (4.1)	1 (1.7)	3 (8.1)	
Autoimmune hepatitis	1 (1.0)	1 (1.7)	0 (0.0)	
Biliary atresia	1 (1.0)	1 (1.7)	0 (0.0)	
HCC, n (%)	33 (34.0)	22 (36.7)	11 (29.7)	0.484
Liver disease duration, year (SD)	7.81 (7.20)	6.23 (5.84)	10.37 (8.46)	**0.005**
MELD score, mean (SD)	15.4 (6.14)	15.5 (6.85)	15.24 (4.83)	0.797
CTP score, mean (SD)	8.4 (2.09)	8.4 (2.32)	8.2 (1.67)	0.639

CTP grade, n (%)				
A	16 (16.5)	12 (20.0)	4 (10.8)	
B	52 (53.6)	26 (43.3)	26 (70.3)	
C	29 (29.9)	22 (36.7)	7 (18.9)	
Operation type, n (%)				1.000
CDLT	2 (2.1)	1 (1.7)	1 (2.7)	
LDLT	95 (97.9)	59 (98.3)	36 (97.3)	
Portal HTN, n (%)	97 (100.0)	60 (100.0)	37 (100.0)	0.677
Esophageal varix, n (%)	82 (84.5)	50 (83.3)	32 (86.5)	0.523
SBP, n (%)	2 (2.1)	2 (3.3)	0 (0.0)	0.819
Ascites, n (%)	46 (47.4)	29 (48.3)	17 (45.9)	0.515
Splenomegaly, n (%)	88 (90.7)	52 (96.3)	36 (100.0)	
Splenorenal shunt, n (%)	25 (27.8)	12 (22.2)	13 (36.1)	0.150
Portal vein thrombus, n (%)	28 (28.9)	17 (28.3)	11 (29.7)	0.883

*Student t test for numerical variables, Pearson's chi-square or Fisher's exact test for non-numerical variables

Table 2. SAA characteristics (patients N=97, total aneurysms N= 142)

Variable	Description
Number of SAAs per patient, n (%)*	
1	65 (67.0)
2	19 (19.6)
Three or more	13 (13.4)
Types of SAA, n (%)**	
Saccular	130 (91.5)
Fusiform	12 (8.5)
SAA mean diameter, cm (SD)	1.82 (0.79)
≥1.82cm, n (%)**	44 (30.9)
<1.82cm, n (%)**	98 (69.1)
SAA location, n (%)**	
Proximal third	10 (7.0)
Middle third	17 (12.0)
Distal third	29 (20.4)
Hilum	83 (58.4)
Intraparenchymal	3 (2.1)
SAA thrombus, n (%)**	2 (2.1)
SAA wall calcification scoring, n (%)**	
0 (No calcification)	137 (96.5)
1 (calcification less than 40%)	2 (1.4)
2 (calcification more than 40%)	3 (2.1)

*The percentage was calculated with the denominator being the total number of patients.

** The percentage was calculated with the denominator being the total number of aneurysms

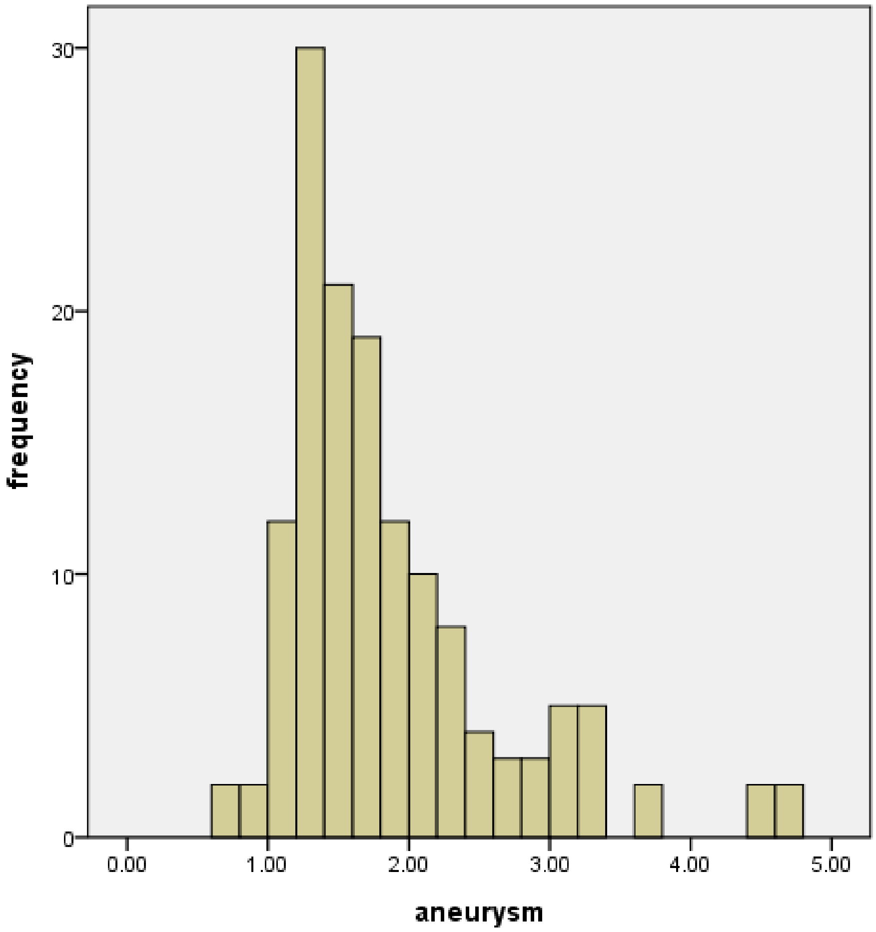

Figure 3. SAA size frequency distribution

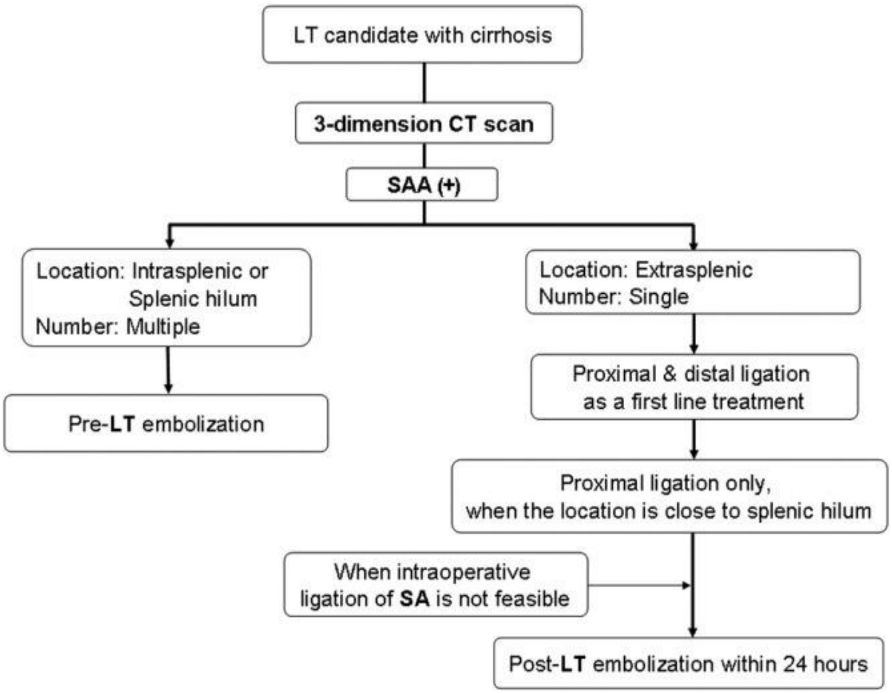

Figure 4. Treatment algorithm for SAAs in Liver transplantation with cirrhosis at the Asan Medical Center. [10]

Table 3. Treatment and fate of SAAs (patients N= 97, SAA total N=142)

Variables	Description
Treatment, n (%)*	
Preoperative intervention	27 (27.8)
- Embolization	26
- Stent insertion	1
Intraoperative intervention	54 (55.7)
- Ligation of splenic a.	51
- Embolization	3
Postoperative intervention	8 (8.2)
- Embolization	8
Observation	8 (8.2)
Follow-up period, months (SD)	53.6 (17.5)
Complication, n (%)*	1 (1.0)
Fate of SAA, n (%)**	
No interval change in size	74 (52.1)
Remain artifacts d/t Embolization	59 (41.5)
Disappearance	3 (2.1)
Decreased in size	4 (2.8)

*The percentage was calculated with the denominator being the total number of patients.

** The percentage was calculated with the denominator being the total number of aneurysms.

DISCUSSION

This study explored the features of coincidental SAA in individuals with PHTN. Small sample sizes have constrained earlier research on this subject due to the rare occurrence of this condition.[13] However, at our institution, we conduct a considerable number of liver transplantations. As the rupture of SAA can significantly affect the prognosis of liver transplant patients, we have adopted a more proactive approach to screening and intervention.[10] Despite the low prevalence of the condition due to its specificity, we were able to include a significant number of SAA patients.

Prevalence

In our study, the prevalence of SAA was 4.8%. This prevalence was approximately five times higher than that of the general population. However, in previous studies targeting liver transplant recipients, the prevalence of SAA ranged from 7% to 17%.[10, 14-16] The prevalence in our study was relatively low compared to previous studies. In a study conducted by Kóbori et al.,[14] (prevalence 13%, 45/337 patients), all patients received cadaveric liver transplantation, including pediatric cases (95 patients). Heestand et al.[16] (prevalence 13%, 9/70 patients) used different criteria to define aneurysms, classifying an increase of more than 25% in the splenic artery diameter as indicative of SAA. It is believed that these variations in definitions may contribute to differences in reported incidence. In our study, SAA measurement included only cases where the aneurysm was clearly visible and measurable on CT scans, potentially leading to an underestimation of the actual prevalence.

Sex

In the general population, SAA occurs four times more frequently in females than males.[2] In our study, the gender ratio among patients with concomitant SAA was relatively balanced. However, in the overall liver transplant recipient population, males outnumbered females by a ratio of 2:1. When we analyzed the entire male and female populations, the SAA incidence was approximately 3.1% in males (47 out of 1,516) and 7.0% in females (43 out of 611). The gender ratio difference was less pronounced than in the general population, and the results were similar to previous studies conducted on liver transplant recipients.[10] SAA incidence varies between men and women, and factors such as fertility and reproductive capacity are essential contributors. However, relevant factors, including fertility and reproductive capacity, were not investigated in the current study population, making it challenging to determine their impact.

Liver disease–related features

In terms of etiology, viral hepatitis was the most common, consistent with the results of the study by Heestand et al.[16] and a study conducted at the Mayo Clinic.[4] While the MELD and CTP scores did not show significant differences compared to the overall average, the duration of illness was relatively longer. However, statistically significant comparisons were challenging since the number of patients with SAA is very small compared to the overall patient population. Further analysis is needed through propensity score matching to compare patients with similar baseline characteristics.

SAA characteristics

In our study, SAAs were most commonly distributed near the hilum and the distal third portion. This pattern suggests that the increased blood flow into the splenic artery due to PHTN and increased pressure at the bifurcation site may contribute to the development of SAAs.[5, 9] Therefore, it can be inferred that PHTN becomes the primary pathological factor contributing to the occurrence of SAAs in this patient group. The average diameter of SAAs was 1.82 cm. Compared to previous studies, our study had a smaller average size when considering the entire population of SAA patients (ranging from 2.2 cm to 3.1 cm). [4, 14] However, when compared to studies focusing on patients with concomitant liver disease (average size of 1.5 cm),[17] the average size was similar. This was likely due to more frequent imaging examinations for liver diseases, enabling the diagnosis of SAA even at smaller sizes. When dividing the patient population into two groups based on a 1.82 cm cutoff, there are significant differences in the presence of hypertension and the duration of liver disease. In the case of hypertension, Stanley et al.[18] showed that it impacts SAA, yielding consistent results. As all patients had PHTN, interpreting the duration of liver disease as the time during which PHTN acted is plausible. This suggests that hypertension and PHTN may contribute to the increase in the size of SAAs over time. While wall calcification of SAA was not observed in the majority of cases, results align with previous studies reporting that eggshell-like calcification, observed in over 75% of cases, serves as a protective factor against aneurysm dilation but is not frequently observed in patients with concurrent PHTN.[18]

Outcome of treatment

One patient developed a splenic abscess, indicating a low risk of complications. The risk of SAA rupture after liver transplantation has been reported to be approximately 2-4%.[16]

The relatively low incidence rate found in this study, combined with results from prior research involving liver transplant patients or individuals with liver fibrosis, implies that treating SAAs can be advantageous.[19] Elsewhere, a 0.97% probability of new-onset visceral aneurysm development was determined among post-liver transplant patients, which is higher than the prevalence in the general population; larger aneurysms have also been observed in this population.[20] In contrast, our study did not identify any new cases of aneurysms in the entire cohort of 2,127 patients, and no patients showed an increase in aneurysm size. While no significant differences in outcomes were observed based on preoperative, intraoperative, or postoperative interventions, it is noteworthy that in cases where aneurysms disappeared or showed a remarkable reduction in size of 0.5 cm or more in follow-up CT scans, all of these cases had undergone intraoperative ligation. As analyzed in previous studies on surgical ligation,[10, 21] it is evident that surgical ligation effectively blocks the flow within the aneurysm. However, in the case of endovascular repair, where coil embolization is predominantly used, it can be challenging to assess the degree of aneurysm shrinkage through CT scans, making it difficult to compare the effectiveness. Especially in patients with concomitant PHTN and liver fibrosis, open surgical repair is associated with a higher risk of bleeding and complications.[22] Endovascular repair may be considered a preferred option. Complications such as spleen infarction or abscess more commonly accompany splenic artery aneurysms than the surgery itself. When considering treatment, it is necessary to weigh the advantages and disadvantages of surgery and endovascular interventions, considering the patient's condition and factors such as the location and size of the SAA. Further research is needed to determine if there are differences in outcomes between the treatment methods.

This was a single-center, retrospective analysis susceptible to selection bias. It focused on patients who underwent liver transplantation, and those with relatively severe liver fibrosis and portal hypertension were included. Factors other than PHTN may have influenced SAA's occurrence and size increase. Additionally, the varying follow-up periods among patients prevented an analysis related to prognosis.

CONCLUSION

Through this study, it was possible to confirm that SAA accompanied by PHTN mainly exhibits the characteristics of saccular types located distally and at the hilum. Additionally, the presence of hypertension and the duration of PHTN were associated with SAA diameter. Therefore, in patients with SAA accompanied by PHTN, especially those with a history of hypertension and a prolonged duration of liver disease, it may be worth considering SAA treatment to mitigate the risk of size increase. Additionally, it is advantageous to intervene for SAA in liver transplant recipients. However, further research is needed to establish the appropriate timing and methods for preventing rupture in SAA patients with concomitant PHTN.

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국문요약

서론

비장동맥류는 일반인구의 0.8%로 그 발생빈도는 낮으나 내장동맥류 중 가장 흔하며, 파열 시 사망률이 높은 질환이다. 문맥 고혈압은 비장동맥류의 발생과 파열의 위험인자로 알려져 있다. 그러나 문맥고혈압을 동반한 비장동맥류 환자에 대한 구체적인 치료 지침이 없다. 따라서 본 연구에서는 본원에서 비장동맥류와 문맥고혈압을 동반하는 간 이식 환자를 대상으로, 그들의 인구통계학적 및 임상 특성을 분석하고 치료방법에 따른 예후를 조사하였으며 이를 통해 문맥고혈압을 가진 비장동맥류 환자의 치료에 도움이 되는 임상정보를 제공하고자 하였다.

연구방법

2016년 1월부터 2020년 12월까지의 기간 동안 본원에서 간이식을 받은 환자들을 대상으로 한 후향적 연구로, 이식 전후에 시행하는 4상 조영증강 전산화단층촬영을 검토해 비장동맥류를 동반한 환자를 찾아 인구통계학적 특징 및 간 질환 관련 특성, 동반된 비장동맥류의 임상적 특징, 이식 전, 중, 후에 시행한 치료 방법에 대해 조사하였다. 또한 비장동맥류에 대한 치료 후 추적관찰 중 가장 마지막으로 시행한 전산화단층촬영 검사를 통해 비장동맥류의 변화를 평가하였다. 추가적으로, 환자군을 비장동맥류의 평균 크기를 기준으로 두 그룹으로 나누어 두 군 사이에 유의미한 차이를 보이는 특성이 있는지 분석하였다.

연구결과

5년기간 내에 2,127명의 간 이식 환자 중 104명의 환자가 비장동맥류를 동반하고 있었고 발병률은 4.8%였다. 7명의 환자는 후향적 조사 기간이 충분하지 않거나 수술 후 조영증강 전산화단층촬영 검사가 없어 제외되었고, 최종 분석에는 97명의 환자와 142개의 비장동맥류가 포함되었다. 연령 평균은 54.5세이며 여성은 인구의 50.5%를 차지하고 있었고 질병 지속 기간의 평균은 7.8년이었다. 비장동맥류를 동반한 모든 환자에서 문맥고혈압

관찰되었으며, 88%가 비장 비대를 가지고 있었다. 비장동맥류는 주로 비장문과 원위부에 위치하였다. 대부분의 비장동맥류는 낭종형(91.5%)으로, 평균 직경은 1.82 cm이었다. 평균 추적 기간은 52.4개월이었으며, 단 한 명의 환자만 SAA 관련 합병증을 경험하였다. 추적 기간 동안 새로운 동맥류나 크기 증가는 식별되지 않았다. 비장동맥류 직경의 평균값인 1.82 cm의 기준으로 환자들을 두 그룹으로 나누어 분석 시, 44개의 비장동맥류(30.9%)가 이 평균 이상의 크기였고, 37명의 환자가 해당되었다. 직경에 따라 나눈 97명의 환자를 분석한 결과, 비장동맥류의 크기가 큰 환자들에서 고혈압의 발병률이 더 높았으며, 간 질환 기간이 더 길게 나타났다.

결론

본 연구를 통해 문맥고혈압을 동반한 비장동맥류의 경우, 원위부와 비장문 근처에 있는 낭종형이 흔히 나타나는 특징을 보임을 알 수 있었다. 또한, 비장동맥류에 대한 치료를 시행하는 것이 파열의 합병증 발생 위험에 비해 안전한 것으로 보이며, 고혈압 및 간 질환 유병 기간이 비장동맥류의 크기와 관련성을 보여, 향후 문맥고혈압을 동반한 비장동맥류 환자에서 고혈압 병력과 간 질환 유병기간이 긴 환자의 경우에 크기 증가의 위험성을 고려하여 보다 적극적인 비장동맥류에 대한 치료를 고려해볼 수 있을 것으로 생각된다.