Multidetector Computed Tomography Findings of Splenic Artery Aneurysms Associated With Liver Involvement in Wilson's Disease

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ABSTRACT

Purpose. The purposed of this study was to examine the incidence and multidetector computed tomography (MDCT) findings of splenic artery aneurysms (SAAs) in patients with liver involvement related to Wilson's disease.

Methods. Eighteen patients with clinically and/or pathologically proven Wilson's disease underwent triphasic MDCT. Arterial, portal, and equilibrium phase images were obtained. The analysis of the CT features included the presence and characteristics of the SAA, splenic artery (SA) diameter, the presence and size of the portosystemic collateral vessels, and spleen volume.

Results. SAAs were detected in 11 patients (61.1%). Eight (72.7%) patients had multiple aneurysms. In 6 (54.5%) patients, the SAAs were located in the distal third of the SA and the intraparenchymal part of the SA. In 3 (27.3%) patients, the SAAs were located only in the distal third of the SA. In 1 (9.1%) patient, the aneurysms were located in the intermediate, distal third, and intraparenchymal part of the SA; in another (9.1%) patient, the aneurysms were located only in the intraparenchymal part of the SA. There were significant differences between the patients with SAA and those without SAA with respect to SA diameter, portosystemic collateral vessel diameter, and spleen volume ($P = .007$, $P < .001$, and $P = .006$, respectively).

Conclusions. The incidence of SAAs seems to be higher in patients with liver involvement related to Wilson's disease compared with patients with other causes of cirrhosis and portal hypertension. Large portosystemic collaterals, increased SA diameter, and spleen volume were significant factors for the presence of SAAs.

THE SPLENIC artery (SA) is the third most common site of intra-abdominal aneurysms aside from the abdominal aorta and the iliac arteries [1]. The true prevalence of splenic artery aneurysm (SAA) varies from 0.2% to 10.4% among the healthy population [2,3]. Although its pathogenesis is not clearly understood, the risk factors include trauma, hormonal and hemodynamic changes in pregnancy, chronic liver disease with portal hypertension, focal arterial inflammatory processes, and atherosclerosis [4,5]. The incidence of SAA is higher in patients with portal hypertension and chronic liver disease and has been reported to range from 2.97% to 50% [4,6–11]. The significance of the early detection and treatment of SAAs stems from the risk of rupture, which increases up to 3%–4% after liver transplantation [1,11,12].

Digital subtraction angiography (DSA) has been reputed to be the gold standard for the detection of SAAs. This technique has the disadvantage of being invasive and is

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often associated with complications [4,5,13,14]. However, recent advances in multi-detector computed tomography (MDCT) technology, including multiplanar reconstructions and three-dimensional volume-rendering technique, allow us to evaluate the SAAs noninvasively [14,15].

Wilson's disease, which is one of the risk factors for the development of portal hypertension and liver cirrhosis, is a rare inherited metabolic disorder that leads to copper accumulation in the liver and brain [16]. The incidence, characteristics, and clinical features of SAAs associated with various types of liver cirrhosis, as determined using different diagnostic modalities, have been reported previously [6–11]. In these studies, the patient population mainly consisted of patients with virus-induced cirrhosis. The number of patients with Wilson's disease is also very restricted. Thus, the purpose of this article is to determine the incidence and to describe MDCT findings of SAAs associated with liver involvement in the context of Wilson's disease.

MATERIALS AND METHODS

Between January 2010 and February 2012, 18 patients (9 men and 9 women; age range, 15–40 years; mean age, 29.4 ± 7.1 years) with clinically and/or pathologically proven Wilson's disease were included in the study. This study was approved by the ethics committee at our hospital, and written informed consent was obtained from all patients.

All patients underwent triphasic MDCT, which was used to characterize nonenhanced, arterial, portal venous, and equilibrium phases. Two scanners were used. Six patients underwent imaging with a 16-MDCT scanner (LightSpeed, GE Medical Systems, Milwaukee, Wis, United States), and 12 patients were evaluated with a 64-MDCT scanner (Aquillion 64; Toshiba Medical Systems, Tokyo, Japan). The examination protocols included the following scan parameters: for the 16-MDCT scanner, we used 16 × 1.25 mm collimation, 1.25-mm reconstruction thickness, 1.25-mm reconstruction interval, 120 kVp, and 135–250 mA. For the 64-MDCT scanner, we used 64 × 0.5 mm collimation, 1-mm reconstruction thickness, 0.5-mm reconstruction interval, 120 kVp, and 110–320 mA.

A total of 120 mL of nonionic contrast material (iopromide, Ultravist 370, Bayer Schering Pharma, Berlin, Germany) was injected into an antecubital vein at a rate of 4.0 mL/s with a power injector. Arterial-phase imaging was initiated within 5 seconds after reaching the enhancement of the descending aorta up to 180 HU (hounsfield unit), as measured by a bolus-tracking technique. The delay times for the portal venous phase and the equilibrium phase were 65 seconds and 120 seconds after the initiation of contrast infusion, respectively.

Axial images were transferred to a standard, commercially available workstation (Advantage Windows 3.1; GE Medical Systems, Milwaukee, WI, USA). All images were reviewed retrospectively by two radiologists (O.R.S. and K.F.) experienced in image processing. The decisions related to these findings were finalized by consensus. Arterial-phase images were used for the evaluation of SAAs. Analysis of the image data was based on axial images and two-dimensional (2D) and three-dimensional (3D) post-processing images (maximum intensity projection, multiplanar reformatting, and volume rendering). SAA was defined as an area of dilation with at least a 50% increase in diameter compared with the adjacent normal segments. Analysis of the CT features included location, number, and long diameter of the SAAs. The locations of the aneurysms were defined as follows: proximal, middle, and distal thirds of the SA and the intraparenchymal part of the SA. In addition, the diameter of the SA, the presence and size of portosystemic collaterals, and spleen volume were recorded. The equilibrium phase was used for CT volumetric measurement of the spleen. Using an electronic mouse, the contours of the spleen were manually isolated from the surrounding organs. Areas (cm^2) were calculated using software available on the scanner. The areas on each of the slices were added and multiplied by the slice thickness, which yielded a volumetric measurement.

All values are expressed as the means ± standard deviations. The SAA and non-SAA groups were compared using Fischer exact test, the Mann-Whitney U test and the t test. A P value $< .05$ was considered significant. All analyses were performed using SPSS 17.0 for Windows (Chicago, Ill, United States).

RESULTS

Eleven of the 18 patients with liver involvement related to Wilson's disease (61.1%) were found to have SAAs. The average ages of the patients with SAA and without SAA were 31.2 ± 6.9 years (5 men and 6 women) and 26.6 ± 7.1 years (4 men and 3 women), respectively. Of the 11 patients with SAA, 8 (72.7%) patients had multiple (more than 3) aneurysms. In 6 (54.5%) patients, the SAAs were located in the distal third of the SA and the intraparenchymal part of the SA. In 3 (27.3%) patients, the SAAs were located only in the distal third of the SA, and in 1 (9.1%) patient, the aneurysms were located in the intermediate, distal third, and intraparenchymal part of the SA. In 1 (9.1%) patient, the aneurysms were located only in the intraparenchymal part of the SA. The mean diameter of the SAAs was 11.8 ± 5.7 mm (range, 4.9–20.2 mm).

There were no significant differences between the patients with and without SAA in terms of age or gender ($P = .189$ and $P = 1$, respectively). All patients with SAA had visible collateral vessels; however, 71.4% of the non-SAA patients had visible collateral vessels ($P = .137$). There were significant differences between the SAA and non-SAA patients with respect to SA diameter, portosystemic collateral vessel diameter, and spleen volume. The mean diameters of the SA were 8.6 ± 1.7 mm in the patients with SAA and 6.2 ± 1.3 mm in the patients without SAA ($P = .007$). The mean diameters of the collateral vessels were 18.7 ± 8.05 mm in the patients with SAA and 5.7 ± 0.9 mm in the patients without SAA ($P < .001$). The mean spleen volumes were 1133.7 ± 666.9 mL in the patients with SAA and 560.4 ± 258.2 mL in the patients without SAA ($P = .006$; Table 1). According to our results, these 3 parameters were significant indicators for the presence of SAA.

DISCUSSION

SAAs represent an apparently rare but life-threatening condition and were first described by Beaussier in 1770 [17]. The actual incidence of SAA in patients with chronic liver disease and portal hypertension is higher than that in the general population [4,6–11]. The most important