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Complications in transcatheter closure of congenital portosystemic venous shunt using Amplatzer Vascular Plug

Jun Muneuchi*, Ryohei Matsuoka, Yusaku Nagatomo, Chiaki Iida, Seigo Okada, Hiromitsu Shirouzu, Mamie Watanabe

Department of Pediatrics, Japan Community Healthcare Organization, Kyushu Hospital, Japan

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ABSTRACT

Amplatzer Vascular Plug (AVP) has been utilized to occlude a congenital portosystemic venous shunt (CPSVS) that is a rare splanchnic venous anomaly. We present a case of a 12-year-old boy in whom device migration and portal vein thrombosis occurred after transcatheter closure of CPSVS using AVP type2. We reviewed the published literatures regarding with complications of CPSVS closure using AVP. Among 23 patients, we identified 5 complications including hypovolemic shock, hypoxia, liver dysfunction, device migration, and thrombosis. It is recommended to choose a device more than twice the diameter of the shunt, and to treat with an anticoagulant after closure.

1. Introduction

Congenital portosystemic venous shunt (CPSVS) is a rare congenital anomaly of splanchnic venous system that occurs in one per 30,000–50,000 children [1]. The management of CPSVS depends on the presence of hepatic encephalopathy, pulmonary arterial hypertension, cyanotic hepatopulmonary syndrome, and liver tumor (focal nodular hyperplasia), because these complications result in adverse outcomes [1–5]. Closure of the shunt is also recommended if the shunt ratio exceeds 60% even in asymptomatic patients [1]. The anatomy of CPSVS is classified as intrahepatic or extrahepatic types, and an intrahepatic portosystemic shunt arising from a portal branch is usually suitable for transcatheter closure [6]. A shunt needs to be evaluated with a balloon test occlusion, a measurement of portal venous pressure, and portography before attempting a complete closure of CPSVS. Shunt closure should be avoided when portal venous pressure rises above 30 mmHg [1]. Transcatheter closures of CPSVS include coil embolization, balloon occluded transvenous obliteration (BRTO), and embolization using Amplatzer Vascular Plug ™ (AVP) (St. Jude Medical Inc. Minnesota, USA) [1,4]. The AVP family consists of four models built with nitinol braids, and all plugs have self-expandable features. The device can be repositioned prior to deployment and is detached by unscrewing the device from the delivery, and is highly useful in transcatheter closure of CPSVS. Since Gillespine firstly described transcatheter closure of CPSVS using AVP [7], 23 cases have been reported so far. However, there is little information about complications associated with transcatheter closure of CPSVS using AVP. We demonstrate a 12-year-old boy who present with pulmonary arterial hypertension and hyperammonemia from a large intrahepatic CPSVS. Following the shunt closure using AVP II, there was migration of the device and the development of portal vein thrombosis. In addition, we performed a systematic review of published literatures regarding with complications of CPSVS closure using AVP.

1.1. Case report

A 12-year-old boy was referred to our hospital, because of cardiomegaly on chest X-ray and right ventricular hypertrophy on electrocardiogram on the school medical check-up program. Heart rate and blood pressure were 68 beats per minute and 116/52 mm Hg, respectively. Physical examination showed an accentuated pulmonary second heart sound and systolic heart murmur of grade 2 on the left lower sternal border. Echocardiography showed a

Abbreviations: CPSVS, Congenital portosystemic venous shunt; BRTO, balloon occluded transvenous obliteration; AVP, Amplatzer Vascular Plug.

* Corresponding author. Department of Pediatrics, Japan Community Healthcare Organization, Kyushu Hospital, 1-8-1 Kishinoura, Yahatanishi-ku, Kitakyushu City, Fukuoka 806-8501, Japan.
E-mail address: jmune@msn.com (J. Muneuchi).

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flattened interventricular septum on a short axis view and an increase in the peak velocity of tricuspid regurgitation of 4.0 m/s, which suggested that the estimated right ventricular systolic pressure was above 60 mmHg. Blood test revealed an increase in plasma ammonia of 146 μg/dl. Abdominal ultrasound and enhanced computed tomography of the abdomen showed a communication vessel between the intrahepatic left portal vein and the left hepatic vein. Brain magnetic resonance imaging showed the high intensity in the bilateral globus pallidus on T1-weighted images. Right heart catheterization demonstrated that mean pulmonary arterial pressure and pulmonary arterial resistance index were 37 mmHg and 4.3 Wood unit·m^2, respectively. We performed transcatheter closure of the CPSVS. Written informed consent was obtained before the procedure from the patient and his parents. Portal venography showed that the diameter of the shunt vessel was 11 mm. Balloon occlusion test of the shunt revealed that portal venous pressure was elevated from 8 mmHg to 15 mmHg, and portal venography during balloon occlusion (A 24-mm Sizing balloon, St. Jude Medical Inc. Minnesota, USA) showed intrahepatic portal vein, which allowed the closure of the shunt vessel (Fig. 1). We choose a 20-mm AVP II to close the shunt. Approaching from the right jugular vein, a 7-French, 120-cm sheath (TorqVue ™, St. Jude Medical Inc. Minnesota, USA) through 0.035 inch guidewire was introduced from the left hepatic vein into the portal vein through the shunt vessel. A 20-mm AVP II attached to the delivery wire was advanced within the sheath. After we confirmed the position of device by carefully and repetitive venography, the device was successfully deployed and detached (Fig. 2a). On the next day, however, chest X-ray showed that the device unfortunately migrated into the right pulmonary artery. We introduce a 10 French, 120-cm sheath (TorqVue ™, St. Jude Medical Inc. Minnesota, USA) from the right femoral vein and retrieve the device using two snare wires. We had discussed the indication of transcatheter closure for several months, and we again performed the transcatheter closure using a 22-mm AVP II (Fig. 2b). We deployed the device in the more proximal position than that in the first procedure, where was close to the bifurcation of the main portal vein (Fig. 3). At last, we successfully embolized the shunt vessel. Plasma ammonia level was decreased to 17 μg/dl. However, following ultrasound examination and contrast computed tomography showed the formation of a partial portal vein thrombus adjacent to the deployed device on the next day of the procedure (Fig. 4). He was treated with intravenous heparin for 3 days and oral warfarin for 3 months. Thereafter, the thrombus disappeared and no recurrent thrombosis occurred. Follow-up computed tomography showed the growth of intrahepatic portal vein and no thrombosis in the portal vein. Echocardiogram showed that right ventricular hypertrophy was improved and the peak velocity of tricuspid regurgitation was decreased to 3 m/s. He is doing well with the administration of oral ambrisentan in the outpatient clinic. These procedures were approved by our institutional review board.

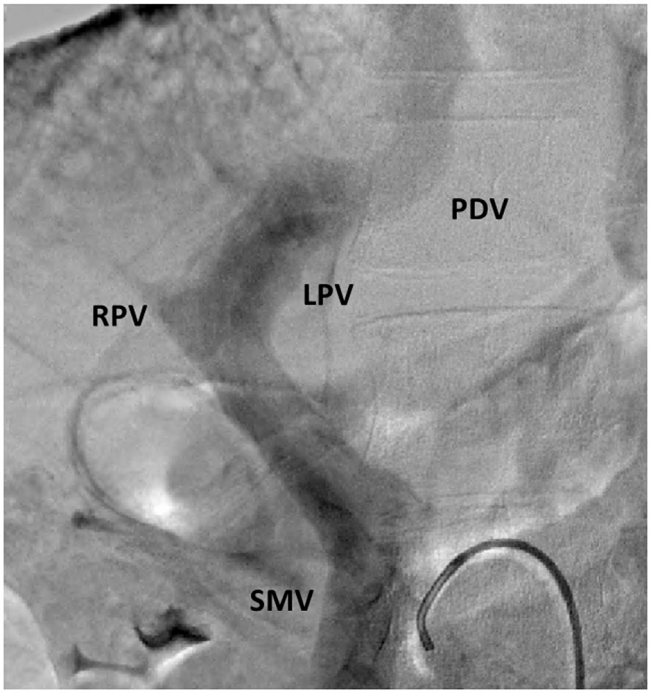

Fig. 1. Portal venous phase of a superior mesenteric artery angiogram shows the left intrahepatic portal vein (LPV) drained to the patent ductus venosus (PDV) in the diameter of 11 mm. The right portal vein (RPV) was hypoplastic. SMV; superior mesenteric vein.

2. Complications in transcatheter closure of congenital portosystemic venous shunt using Amplatzer Vascular Plug

2.1. Method

A literature search of PubMed and EMBASE was conducted for complications of transcatheter closure of CPSVS including the ductus venosus using Amplatzer Vascular Plug with no limitation in types of the article, language and geographical area. We enter the following search query: “transcatheter closure” or “vascular plug”, and “portosystemic venous shunt” or “ductus venosus”. We identified 16 articles regarding with transcatheter closure of CPSVS using AVP, and reviewed clinical data and complications.

2.2. Results

Of 16 articles, we identified 23 cases of transcatheter closure of CPSVS using AVP [4,7–21]. The summary of these cases is shown in Table 1. The first case of transcatheter closure of CPVS using AVP was described by Gillespie et al., in 2006 [7]. They addressed two cases of twin infant brothers with the patent ductus venosus who developed heart failure, in both of which the shunt vessels measured 5 mm in the diameters and occluded using with 8-mm AVP Is. Among 23 patients with CPSVS, the most common clinical manifestation was hepatopulmonary syndrome in 9 patients, following encephalopathy (n = 6), hyperammonemia (n = 2), dyspnea, and proteinuria. There was one asymptomatic patient. Alomari et al. reported a 6-year-old boy with a large patent ductus venosus who developed massive gastrointestinal bleeding as a rare manifestation of CPSVS [10]. The shunt vessel was successfully occluded by using a 16-mm AVP, resulting in an immediate cessation of bleeding. Transcatheter closure of CPSVS was performed at the median age of 6 years (ranging from 8 months to 67 years). Fourteen patients (61%) had intrahepatic CPSVS, and the other 9 patients (39%) had extrahepatic CPSVS. The Park classifications among 14 patients with intrahepatic CPSVS were type V in 7 (50%), type IV in 4 (29%), type III in 2 (14%), and type I in one patient, whereas the Morgan classifications were type II in 7 (78%) and type Ib in 2 patients among 9 patients with extrahepatic CPSVS. The median diameter of shunt vessel measured 10 mm, ranging from 2 to 22 mm (no description in 7 cases).Guneyli et al. reported a 9-year-old boy with a large shunt in the diameter of 20 mm that was occluded using a 22-mm AVP II, which was the similar to the present case [15]. There were 7 cases of the patent ductus venosus including the present case. The median diameter of the ductus venosus was 11 mm which seem to be as large as that of other type