Acta Scientiae Veterinariae, 2024. 52(Suppl 1): 973.

CASE REPORT
Pub. 973

ISSN 1679-9216

Splenophrenic Shunts in Cats - Surgical Management

Kihoon Kim[1], Jaehwan Kim[2] & Hwi-Yool Kim[3]

ABSTRACT

***Background*:** Portosystemic shunts are abnormal vessels that communicate with the portal and systemic venous circulation, causing bypass of blood from the gastrointestinal tract to the liver. Cats with portosystemic shunts may show clinical signs of neurological, gastrointestinal, and urinary tract diseases. The diagnosis is mainly based on computed tomography, and both medical and surgical treatment are recommended for patients with portosystemic shunts. However, the prognosis of long-term medical treatment without surgical management is unsatisfactory in cats. For surgical management of the shunting vessel, surgical correction using a gradual occlusion device, such as an ameroid ring constrictor, is indicated to redirect portal blood into the liver. However, surgical approaches for extrahepatic splenophrenic shunts require careful consideration since the terminating part of the shunting vessel is embedded in the central tendon of the diaphragm. This study presents the outcomes of ameroid ring constrictor placement on the shunting vessel caudal to the diaphragm in 3 cats with splenophrenic shunts.

***Cases*:** All 3 patients showed hypersalivation during the physical examination. On serum biochemical analyses, all patients showed increased ammonia and postprandial bile acid concentrations, indicating a portosystemic shunt. Based on blood tests, radiography, ultrasonography, and computed tomography, an extrahepatic splenophrenic shunt originating from the splenic vein and entering the caudal vena cava at the level of the diaphragm was diagnosed. Placement of an ameroid ring constrictor caudal to the diaphragm was performed to gradually attenuate the shunting vessel. In *Case 3*, additional cystostomy was performed to remove the urinary bladder calculus. All 3 cats recovered uneventfully from anesthesia. In *Case 1*, the ultrasonographic examination conducted during the postoperative period revealed mild abdominal effusion, which spontaneously resolved within a few days. In *Case 3*, the patient showed seizure activity, which resolved spontaneously. The clinical signs in all patients disappeared after surgery. Follow-up studies were performed on days 7, 14, 28, and 56 after surgery. All cats recovered after surgery without major complications.

***Discussion*:** Portosystemic shunts are generally classified as intrahepatic or extrahepatic. In cats, a single extrahepatic shunt originating from the left gastric vein and entering the caudal vena cava is the most frequent type of congenital portosystemic shunt. Intrahepatic portosystemic shunt in cats is much less described, but the most commonly reported type is the left divisional vessel. Ligation of the shunting vessel around the vessel as close to its insertion into the caudal vena cava as possible is generally recommended. If the ligation is placed between the tributaries and portal vein, the tributary still acts as a shunting vessel, resulting in hepatofugal flow. However, if the ligation is placed between the tributaries and caudal vena cava with complete occlusion, the tributary no longer acts as a shunting vessel, resulting in hepatopetal flow. In patients with a splenophrenic shunt, the shunting vessel terminates in the middle of the diaphragm, making it difficult to isolate the shunting vessel without any attachment to the surrounding tissues. Therefore, in the current study, the ameroid ring constrictor was placed caudal to the diaphragm. As shown in the current study, placement of the ameroid ring constrictor in the splenophrenic shunt in 3 cats yielded satisfactory outcomes.

Keywords: ameroid ring constrictor, cat, splenophrenic shunt.

DOI: 10.22456/1679-9216.138513

Received: 23 March 2024 Accepted: 30 June 2024 Published: 29 July 2024

[1]Department of Surgery, Jeil Referral Animal Medical Center, Busan, South Korea. [2]Department of Veterinary Medical Imaging & [3]Department of Veterinary Surgery, College of Veterinary Medicine, Konkuk University (KU), Seoul, South Korea. CORRESPONDENCE: H-Y. Kim [hykim@konkuk.ac.kr]. Department of Veterinary Surgery, College of Veterinary Medicine - Konkuk University (KU). 05029 Seoul, South Korea.

INTRODUCTION

A portosystemic shunt (PSS) is an abnormal vessel that communicates with the portal and systemic venous circulation, which results in blood bypassing from the gastrointestinal tract to the liver. Shunting vessels originate congenitally or are acquired due to portal hypertension caused by severe liver malfunction [1]. They are the most frequently reported congenital vascular anomalies of the hepatobiliary system and the most commonly described cause of hepatic encephalopathy in companion animals [3].

Medical or surgical management is recommended for PSS patients. Medical management can reduce the factors associated with hepatic encephalopathy. However, without surgical management, the prognosis of long-term medical treatment is unsatisfactory in cats [5]. The recommended surgical treatment includes placement of an ameroid ring constrictor (ARC), which is designed for gradual occlusion of a shunting vessel in cats [7,12].

An extrahepatic splenophrenic shunt (EHSPS), originating from the splenic vein and entering the caudal vena cava (CVC), differs from other types of PSS in the isolation of shunting vessels from surrounding tissues. This is because the shunting vessel passes through the diaphragm at the point of entry into the CVC. Only 1 study has described the placement of an ARC in a dog with a splenophrenic shunt [16]. To the best of the author's knowledge, placement of an ARC caudal to the diaphragm in cats with EHSPS has not yet been reported. The objective of this study was to report a case series describing successful surgical treatment by placement of the ARC on the shunting vessel caudal to the diaphragm in 3 cats with EHSPS.

CASES

Case 1. A 5-year-old spayed female domestic shorthair cat weighing 2.48 kg was referred for the evaluation of anorexia and hyperammonemia. A physical examination revealed hypersalivation and lethargy. Serum biochemical analysis revealed increased ammonia concentration (212 μg/dL; reference range [RR]: 0-95 μg/dL) and alanine aminotransferase (ALT) concentration (180 u/L; RR: 12-130 u/L). Low BUN concentration (8 mg/dL; RR: 16-36 mg/dL) was also identified. Preprandial and postprandial bile acid concentrations were 12.8 μmol/L (RR: 0-6.9 μmol/L) and 49.9 μmol/L (RR: 0-14.9 μmol/L), respectively. The combination of increased ammonia levels, high postprandial bile acid concentrations, and low BUN concentrations indicated possible PSS. On ultrasonographic examination, the portal vein (PV) was significantly smaller than the CVC, resulting in a PV/CVC ratio of 0.60 on the transverse image obtained at the porta hepatis level. Multiple anechoic cysts were also observed in the left kidney. On computed tomography (CT) examination, an abnormal vessel originated from the splenic vein and terminated in the phrenic vein cranial to the liver, inserting into the CVC from the left side (diameter, approximately 2.5 mm at the level of insertion), confirming as an EHSPS. As a preanesthetic medication, butorphanol[1] [Butophan® - 0.5 mg/kg, intravenous (iv)] and ampicillin-sulbactam[2] [Ubacilin® - 30 mg/kg, iv] was administered. Anesthesia was induced with alfaxalone[3] [Alfaxane® - 0.3 mg/kg, iv] and maintained with isoflurane[4] [Isoflurane®]. An ARC5 was placed in the shunting vessel caudal to the diaphragm (Figure 1). The abdominal wall and skin were closed routinely. Recovery from anesthesia was uneventful. During hospitalization, clinical signs such as anorexia, hypersalivation, and lethargy resolved. The patient was discharged after 4 postoperative (PO) days, and a protein-restricted diet was recommended at the time of discharge. On day 7, mild abdominal effusion was identified on routine ultrasonographic examination and resolved spontaneously within a few days. The client reported that the patient was doing well after discharge and showed no clinical signs. Preprandial and postprandial bile acid concentrations 8 weeks after surgery were 5.2 μmol/L (RR: 0-6.9 μmol/L) and 14.1 μmol/L (RR: 0-14.9 μmol/L), respectively.

Case 2. A 9-year-old castrated male Scottish Fold cat weighing 3.6 kg presented with hypersalivation, dullness, head shaking, and ataxia. The patient's history included a diagnosis of hyperammonemia 1 month prior to presentation at the local animal hospital. On physical examination, the patient's mental state was depressed. The complete blood count was unremarkable. On serum biochemical analysis, increased ammonia concentration (265 μg/dL; RR: 0-95 μg/dL), hyperglycemia (199 mg/

dL; RR: 71-159 mg/dL), and hyperglobulinemia (6.0 g/dL; RR: 2.8-5.1 g/dL) were identified. The preprandial and postprandial bile acid concentrations were 24.1 µmol/L (RR: 0-6.9 µmol/L) and 111.5 µmol/L (RR: 0-14.9 µmol/L), respectively. On ultrasonographic examination, the PV was significantly smaller than the CVC, resulting in a PV/CVC ratio of 0.51 on the transverse image obtained at the porta hepatis level. On CT examination, an abnormal vessel arising from the splenic vein and inserting into the CVC from the left side (diameter, approximately 2.5 mm at the level of insertion) was confirmed to be an EHSPS (Figure 2). The preanesthetic medication, anesthesia, surgical procedure, postoperative care, and treatment were the same as those in Case 1. The patient was discharged after 6 days PO without any clinical signs. Preprandial and postprandial bile acid concentrations 8 weeks after surgery were 7.6 µmol/L (RR: 0-6.9 µmol/L) and 65.2 µmol/L (RR: 0-14.9 µmol/L), respectively.

Case 3. A 7-month-old female domestic shorthair cat weighing 2.5 kg presented with hypersalivation and anorexia. At the time of presentation, the patient was alert and showed severe hypersalivation. Laboratory serum analysis revealed a severely increased ammonia concentration (513 µg/dL; RR: 0-95 µg/dL) and a slightly low BUN concentration (14 mg/dL; RR: 16-36 mg/dL). The preprandial and postprandial bile acid concentrations were 8.2 µmol/L (RR: 0-6.9 µmol/L) and 36.9 µmol/L (RR: 0-14.9 µmol/L), respectively. The increased ammonia and postprandial bile acid concentrations and low BUN concentration indicated the possibility of PSS. On radiologic examination, microhepatica and radiopaque material were identified in the urinary bladder (UB) region (Figure 3A). Ultrasonographic examination revealed a hyperechoic material (4.3 mm in diameter) with an acoustic shadow within the UB, suggesting a cystic calculus. Urinalysis revealed the presence of ammonium urate crystals. On CT examination, an abnormal vessel was found to originate from the splenic vein and terminate at the CVC cranial to the liver, inserting on the CVC from the left side (diameter, approximately 3.66 mm at the level of insertion), which was confirmed to be an EHSPS. The preanesthetic medication, anesthesia, surgical procedure, postoperative care, and treatment were the same as in *Case 1*, except for the removal of calculi in the UB during the surgery (Figure 3B). The patient was discharged 3 days PO. However, the patient was re-hospitalized due to seizure activity 4 days PO, which disappeared 7 days PO, and was subsequently discharged. Preprandial and postprandial bile acid concentrations 8 weeks after surgery were 3.8 µmol/L (RR: 0-6.9 µmol/L) and 8.9 µmol/L (RR: 0-14.9 µmol/L), respectively. The patient showed no neurological signs 6 months after surgery.

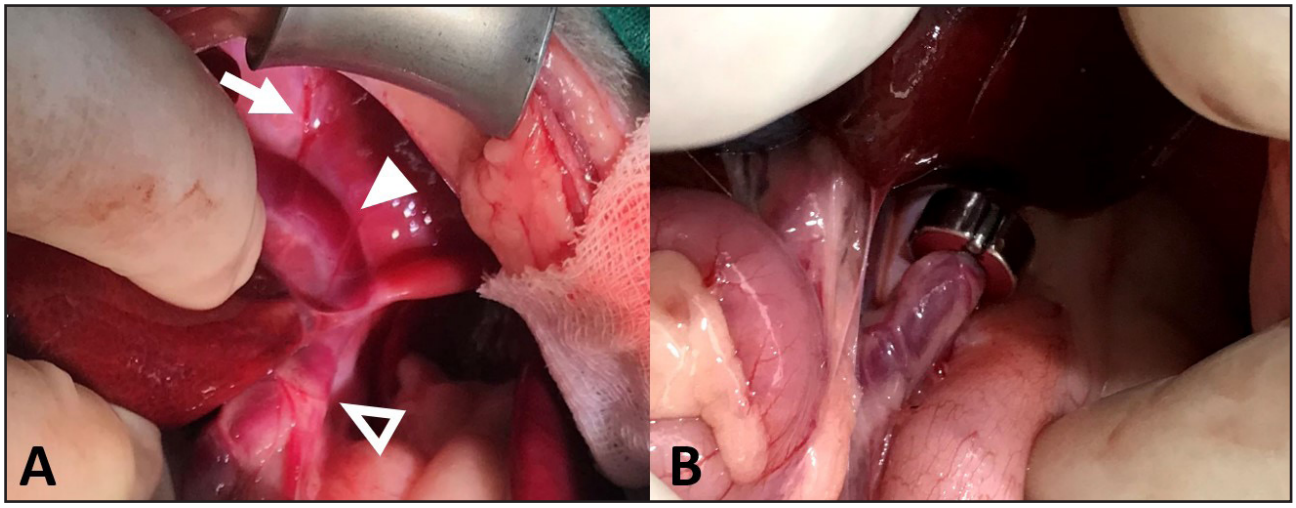

Figure 1. Surgical view of *Cat 1*. A- Phrenic vein passing along the central tendon of the diaphragm (arrow) and entering the shunting vessel (arrowhead) is identified. The ameroid ring constrictor (ARC) was placed caudal to the diaphragm (open arrow). B- The ARC was placed on the shunting vessel caudal to the diaphragm.

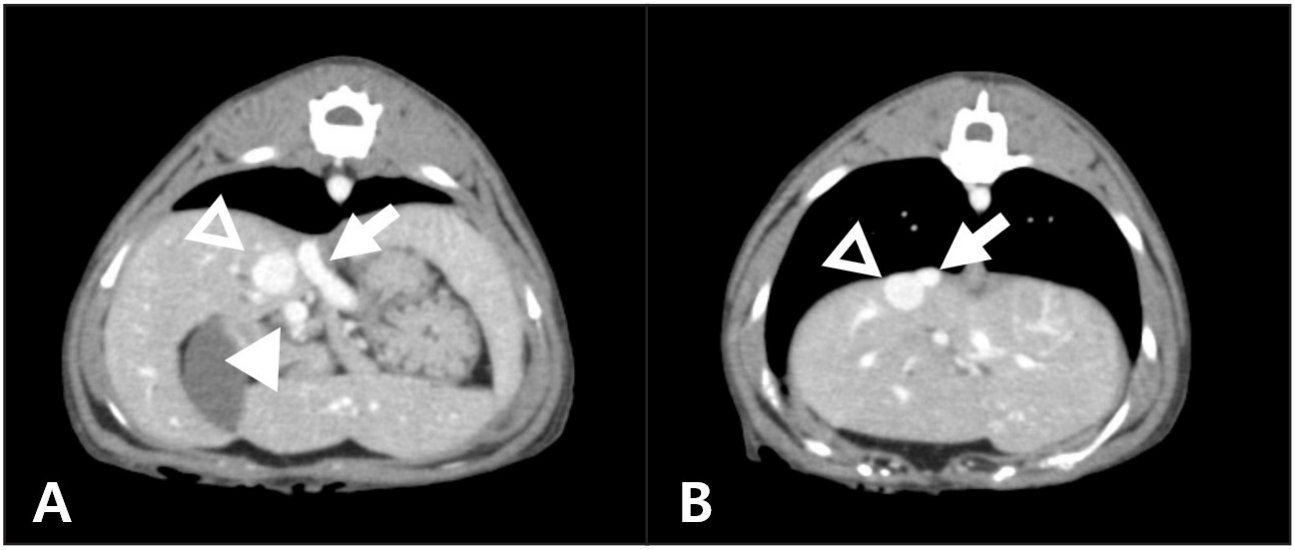

Figure 2. Computed tomography (CT) images of Cat 2. A- Caudal vena cava (CVC; open arrowhead), shunting vessel (arrow), and portal vein (PV; arrowhead) are identified. B- The shunting vessel (arrow) is entering the CVC (open arrowhead) at the level of diaphragm.

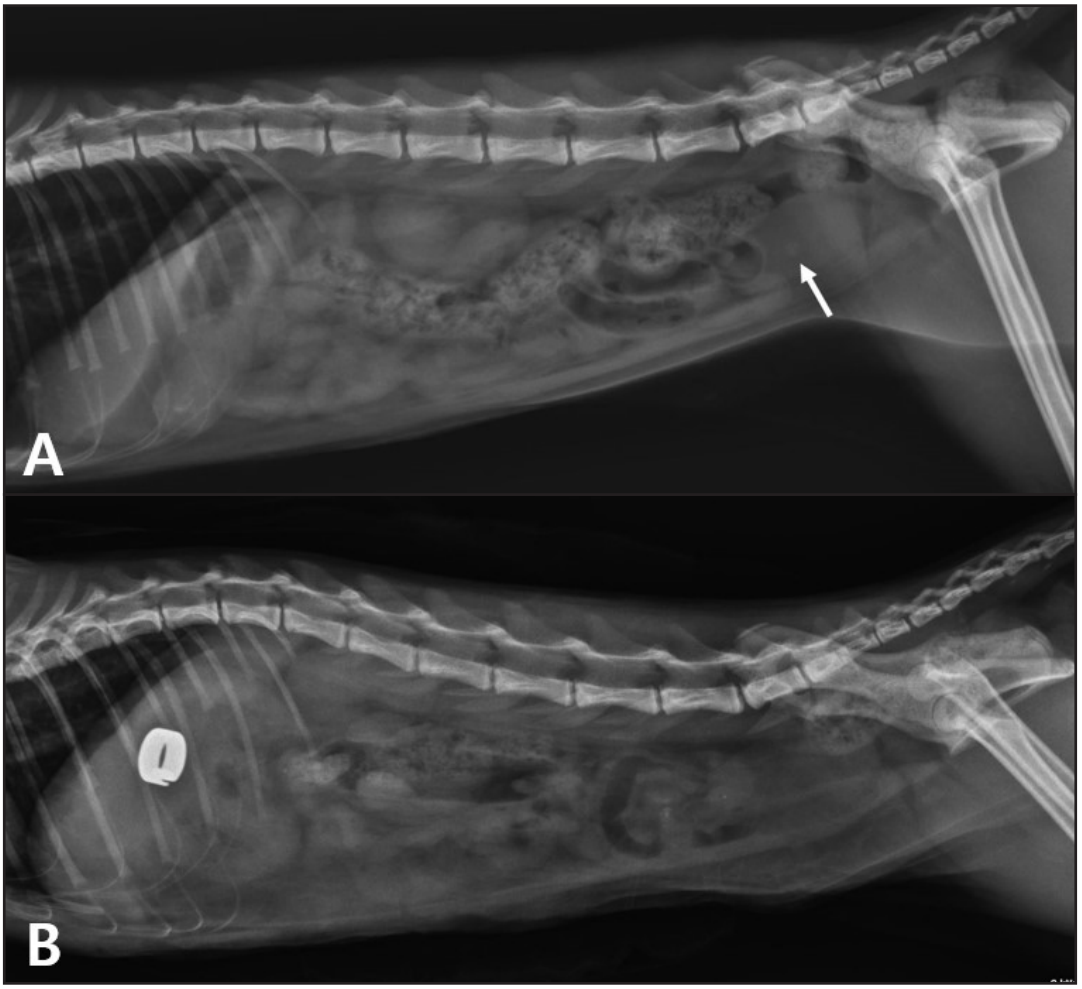

Figure 3. Pre- and postoperative radiographic images of *Cat 3*. A- Cranially displaced gastric axis, suggesting microhepatica, and calculi (arrow) in urinary bladder are identified on the preoperative lateral view. B- The ameroid ring constrictor (ARC) placed caudal to the diaphragm is identified.

DISCUSSION

PSSs are generally classified as intrahepatic or extrahepatic [2]. In cats, a single extrahepatic shunt originating from the left gastric vein and entering the CVC is the most frequent type of congenital PSS [9]; however, extrahepatic portointernal thoracic vein, portoazygos, and other types of portocaval shunts between the colonic or splenic vein and CVC have also been reported [1,8]. Intrahepatic PSS in cats is much less described, but the most commonly reported type is the left divisional vessel [14]. Affected cats may show clinical signs of neurological, gastrointestinal, and urinary tract diseases [9].

Ligation of the shunting vessel around the vessel as close to its insertion into the CVC as possible [4,6,10,11] is recommended for the following reasons: if the ligation is placed between the entry point of the tributaries to the shunting vessel and the PV, the tributary still acts as a shunting vessel, resulting in the entry of hazardous toxins into the systemic circulation. However, if ligation is placed between the tributaries and CVC with complete occlusion, the tributary no longer acts as a shunting vessel, resulting in hepatopetal flow.

The surgical approach for EHSPS should be carefully considered in comparison with that for other types of shunts. Because the EHSPS vessel originates from the splenic vein and enters the CVC at the level of the diaphragm, separating the shunt vessel from the fibrous central tendon of the diaphragm without any tissue attachment is challenging [16]. Yoon *et al*. [16] previously identified the separation of the shunting vessel from the central tendon to be a challenging procedure and reported successful treatment of EHSPS by placing an ARC on the shunting vessel before entering the diaphragm in a dog. Thus, in this case, placement of the ARC with an internal diameter of 6 mm was performed around the shunting vessel right behind the shunting vessel entering the diaphragm. However, the phrenic vein between the CVC and ARC had to be inevitably left in all 3 cases, since the phrenic vein enters the shunting vessel along the diaphragm. Although the ARC was placed in a suboptimal location, the placement of the ARC in the EHSPS resulted in improvement of biochemical findings, imaging parameters, and clinical signs in association with EHSPS in all cases. The serum bile acid concentrations in *Cases 1* and *3* returned to their normal ranges. The diameter of the PV cranial to the insertion point of the shunting vessel was gradually increased. The increased diameter of the PV without any clinical signs might also imply improvement in liver function and structure [15].

In *Case 2*, the postprandial serum bile acid concentration at 8 weeks after surgery was higher than normal, indicating partial occlusion of the shunting vessel. However, the clinical signs resolved. Although partial ligation was achieved, hepatopetal shunt flow can still develop with a lower resistance to the PV than to the ligature [11]. Moreover, even if the function of the shunting vessel remained after partial occlusion, the blood of the splenic vein was less toxic than that of any systemic vein, resulting in normalization of the blood ammonia concentration.

In order to avoid acute fatal portal hypertension, 2 methods have been suggested during surgical procedures, either individually or in combination [11]. Measurement of portal pressure by direct catheterization of a portal tributary has been commonly used for direct quantitative assessment of portal hypertension. The other method is based on observation of qualitative signs (visual changes in the intestines) and indirect quantitative variables (magnitude of fluctuation in mean systemic arterial blood pressure and heart rate) to assess the degree of portal hypertension after ligation. However, in these cases, an intraoperative evaluation of portal hypertension was not performed. ARC causes gradual attenuation of the shunting vessel, resulting in complete shunt occlusion within 1 to several weeks [11]. Thus, an ARC does not require intraoperative assessment of portal hypertension because of its gradual attenuation.

However, in *Case 1*, the patient had abdominal effusion, which was assumed to result from portal hypertension. Moreover, in *Case 3*, the patient showed seizure activity 4 days PO, which spontaneously disappeared 7 days PO. Because the gradual occlusion of the ARC was uncontrollable, postoperative complications, including abdominal effusion or seizure were suspected to result from subacute or chronic portal hypertension, since the aforementioned signs were observed at 4 and 7 days PO, respectively. Subacute or chronic portal hypertension may develop when the hypoplastic portal system cannot adapt to increased blood flow at the same rate as the attenuation rate of the device [13]. This phenomenon was outlined as a rapid return to the normal range of postprandial serum bile acid concentrations in *Cases 1* and *2* at 8 weeks in comparison with that in *Case 3*, which does not reveal any clinical signs of portal hypertension after surgery.

A limitation of the present study is that follow-up assessments for each patient were performed up

to 8 weeks after surgery. Since the ARC occlusion process continues for more than weeks after surgery, complete occlusion of the shunting vessel could not be thoroughly identified, especially in *Case 2*.

In conclusion, placement of the ARC on the shunting vessel caudal to the diaphragm allowed successful management of patients with EHSPS without isolation of the shunting vessel from the central tendon of the diaphragm, which is known to be challenging.

MANUFACTURERS

[1]Myungmoon Pharmaceutical Co. Ltd. Seoul, Republic of South Korea.

[2]Whanin Pharmaceutical Co. Ltd. Seoul, Republic of South Korea.

[3]Jurox Pty Ltd. Rutherford, Australia.

[4]JW Pharmaceutical. Seoul, Republic of South Korea.

[5]Hippo Science. Seoul, Republic of South Korea.

***Declaration of interest*.** The authors alone are responsible for the content and writing of the paper. The authors report no conflicts of interest.

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