

# Giant Pseudoaneurysm of the Splenic Artery: Size/Rupture Correlation

Safi Khuri<sup>a, b, c</sup>, Mira Damouny<sup>a</sup>, Subhi Mansour<sup>a</sup>

## To the Editor

True arterial aneurysm is defined as a vascular lesion that involves the three layers of the arterial wall causing a vascular bulge. On the other hand, a false aneurysm, also known as pseudoaneurysm (PSA), is a vascular lesion that develops usually following a tear in one or two of the three arterial wall layers, with the developing lesion being contained by the outer adventitia or the local hematoma surrounding the PSA [1]. Giant PSA is defined as a vascular lesion greater than 5 cm in diameter. The splenic artery is the most common visceral artery to be involved in true and false aneurysms, with rates of 60% and 40%, respectively [2]. Although the prevalence rate of splenic artery aneurysm is reported to be 0.2-10.4%, the exact prevalence rate is yet to be known [3]. Splenic artery PSA is even rarer, with merely 300 cases reported in the English literature. Recently, due to the considerable increase in the use of the various imaging tests, such as abdominal ultrasound (US), computed tomography (CT) scan and magnetic resonance imaging (MRI), splenic artery aneurysms and PSA are detected with an increasing frequency [2]. Unlike true aneurysm, which is usually asymptomatic and incidentally discovered, splenic artery PSAs are usually symptomatic [2]. The most commonly reported symptoms are abdominal pain and hemorrhage, either intra-abdominal (into the peritoneal cavity) or intra-luminal (into the gastrointestinal (GI) tract) [4]. In a case series article, only 2.5% of splenic artery PSAs were incidentally found [2].

Etiologies for splenic artery PSA are diverse and include mainly pancreas-related pathologies (52%) (e.g., chronic pancreatitis - the most common, acute pancreatitis and pancreatic pseudocyst), post thoraco-abdominal trauma (29%), post-surgical complication (3%) and peptic ulcer disease (2%) [2, 5]. The mechanism behind splenic artery PSA due to pancreasrelated pathology is explained by the "autodigestion theory": leakage of pancreatic fluid, which includes proteolytic en-

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zymes, into the nearby structures, resulting in structural damage of the arterial wall with subsequent PSA formation [6]. Although any artery can be affected, the most common is the splenic artery, followed by the gastroduodenal artery (GDA). The major concern regarding true or false splenic artery aneurysm is rupture, which may be lethal if left untreated [2]. Mortality rates following untreated ruptures are reported to be as high as 90% [7]. Splenic artery PSAs possess a greater risk for rupture than true aneurysms, and thus, immediate management is almost always required for the treatment of PSAs regardless of size [5, 7]. The risk of rupture with consequent hemorrhage is high at about 37% for splenic artery PSA [7, 8]. For true splenic artery aneurysm, a strong relationship exists between the size of the aneurysm and the risk for rupture and bleeding. On the contrary, it has been claimed that this relationship does not exist for the PSA subgroup [9], and size is not included as a risk factor for rupture. The previously mentioned say is relatively an old one, which lacks scientific based studies and has never been statistically proven.

Due to the lack of reported studies about the aforementioned size/rupture risk correlation of splenic artery PSA, and following the hypothesis that larger PSAs have a greater potential for rupture, the aim of this article is to review the pertinent and available articles (mainly case reports and case series) in the English literature concerning splenic artery PSA, specifically the giant type, to find out if this subgroup of splenic artery PSAs have a higher risk for rupture and bleeding (due to larger diameter).

A search in PubMed was conducted, based on the "PI-COS" acronym. Headings and text words were used to identify studies (mainly case series and case reports) published regarding giant splenic artery PSA. The following search terms were included: "splenic artery pseudoaneurysm", "pseudoaneurysm of the splenic artery", "giant pseudoaneurysm of the splenic artery", "giant splenic artery pseudoaneurysm", and "giant visceral artery pseudoaneurysm".

All reported cases of giant splenic artery PSA were included, and data regarding patients' demographics, etiology, clinical presentation, diagnostic technique and therapeutic option were collected.

Reviewing the current English literature revealed almost 300 cases of splenic artery PSA, 40 of which (13.3%) are of the giant type. Of the 40 patients, 25 were males and 15 were females (male/female ratio of 1.6:1). The average age at diagnosis was 48 years old (age range 8 - 78 years old). The most common precipitating etiological factor was chronic pancrea-

<sup>&</sup>lt;sup>a</sup>Department of General Surgery, Rambam Medical Center, Haifa, Israel <sup>b</sup>HPB and Surgical Oncology Unit, Rambam Medical Center, Haifa, Israel <sup>c</sup>Corresponding Author: Safi Khuri, Department of General Surgery, Rambam Medical Center, Haifa, Israel. Email: s\_khuri@rambam.health.gov.il

Characteristics	
Demographic features	
Age, mean (range), years	48 (8 - 78)
Male/female ratio	1.6/1
Etiology	
Chronic pancreatitis	66%
Unknown	15%
Acute pancreatitis	8%
Post-surgical	8%
Post-trauma	3%
Clinical presentation	
Rupture (bleeding)	50%
Abdominal pain	42.5%
Abdominal fullness and pulsation	5%
General deterioration	2.5%

**Table 1.** Demographic Features, Etiological Causes and Clinical Presentations of Patients With Giant Splenic PSA

titis (mainly alcoholic) at 66%, followed by acute pancreatitis (8%), post-surgical (8%) and post traumatic (3%). About 15% of patients were healthy with an unknown etiology. Of the three cases of giant splenic artery PSA following operation, one case was following splenectomy due to idiopathic thrombocytopenia [10], one following pancreatoduodenectomy procedure [11] and one case following cyst-gastrostomy operation [12]. Twenty patients (50%) presented with rupture of the PSA, manifested by bleeding either into the GI tract, into the abdominal cavity or by bloody discharge in the abdominal drains. Initial investigation of this group of patients was dependent on whether the patient is hemodynamically stable or not and whether the bleeding is intra-luminal or extra-luminal. Most stable patients who presented with signs of GI bleeding (intra-luminal hemorrhage) underwent endoscopy as an initial modality for investigation, while non-stable patients underwent abdominal CT scan following initial stabilization [13]. The most common presentation for a ruptured giant splenic artery PSA was upper GI bleeding (complaints of hematemesis, melena or both), reported in nine patients (45%). Upper endoscopic findings were diverse, ranging in terms of severity from diffuse gastritis [14] to non-pulsatile bulging of the proximal third of the greater curvature/posterior wall of the gastric corpus [15]. Five patients (25%) presented with lower GI bleeding, manifested as hematochezia. One case presented with bloody discharge in the abdominal drains following pancreatoduodenectomy [11]. The previously mentioned rupture percentage (50%) is by far higher than the known rupture risk for splenic artery PSA (37%). Seventeen patients (42.5%) presented with abdominal pain, two patients with upper abdominal fullness and pulsation and one patient with general deterioration (Table 1). Of the group of patients presenting with rupture, three cases of mortality (15%) were reported [12, 16, 17], two of which while waiting for endovascular intervention in the hospital at the surgical ward and the third [12] was due to massive GI re-bleeding weeks following endovascular intervention.

Treatment of splenic artery PSA, including the giant type, has undergone a significant evolution over the last two decades, from being a disease treated mainly by surgical means during the previous century to a disease mostly managed by a variety of non-surgical methods nowadays.

Surgical intervention is the therapeutic option of choice, in all cases of splenic artery PSA, if rupture and hemodynamic instability is the initial clinical presentation. Surgical methods used in the treatment of splenic artery PSA include but are not limited to suturing of the splenic artery proximal to the PSA, suturing and splenectomy or splenectomy in addition to distal pancreatectomy, especially in the case of chronic pancreatitis and pancreatic pseudocyst formation.

The non-surgical options available currently include endovascular, endoscopic or CT-guided interventions. Endovascular interventions, mainly with coils embolization of the splenic artery, have emerged over the years as an excellent therapeutic option, for both non-giant and giant subtypes, with high success rates on clinical and radiological follow-up [18-20]. Krueger et al [21] described a successful management of giant splenic artery PSA by CT-guided direct thrombin injection into the PSA lumen. This technique was also reported in other case reports with successful outcomes [7]. A different therapeutic option for direct thrombin injection into the PSA lumen is by endoscopic ultrasound (EUS). It was Rai et al [22] who reported the first case of EUSguided coil and glue injection for the management of splenic artery PSA.

In conclusion, the data mentioned above contradict the formal statement that there is no correlation between splenic artery PSA diameter and the risk for rupture. The risk for rupture is higher in the giant type splenic artery PSA than in the non-giant subgroup of patients. Hence, the previously mentioned claim should be re-examined, and further future studies are encouraged.

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#### **Conflict of Interest**

The authors declare that they have no conflict of interest concerning this article.

#### **Informed Consent**

Not applicable.

## **Author Contributions**

SM designed the research, and MD and SM collected and analyzed data. SK wrote and approved the final paper.

# **Data Availability**

The authors declare that data supporting the findings of this study are available within the article.

## Abbreviations

PSA: pseudoaneurysm; GI: gastrointestinal

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