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Case Report

Spontaneous Isolated Superior Mesenteric Artery Dissection (SISMAD): A Case Report and Review of the Literature

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Abstract

Introduction: Spontaneous isolated superior mesenteric artery dissection (SISMAD) is a rare vascular disease that lacks evidence-based guidelines regarding treatment options. In this paper, we present a case report perform a literature review of potential methods for the treatment of SISMAD.

Materials and Methods: A 60-year-old man presented with epigastric pain that had been aggravated by the consumption of food for two weeks. There was no history of smoking or alcohol consumption, and he had received previous treatment for hypertension and thyrotoxicosis which had lasted for over five years. After the detection of SISMAD, he was treated conservatively with antiplatelets for a follow up period of over 18 months. After this period of treatment, he no longer presented symptoms and there was a regression of the lesion which was demonstrated by interval CTA and duplex US.

Conclusion: Conservative treatment can sometimes be used for SISMAD in selected cases. Further investigations when bowel ischemia or mesenteric hemorrhage is suspected might be necessary as invasive treatment might be indicated.

Keywords: Superior mesenteric artery, Dissection, Hypertension

Introduction

The first case report of spontaneous isolated superior mesenteric artery dissection (SISMAD) was in 1947 by Bauersfield et al[1]. It is the second most frequent peripheral artery to be affected by dissection, after the internal carotid artery[2]. SISMAD most commonly occurs in people with an age falling in the mid-50s, and presents with epigastric pain after overeating and drinking[3,4]. This rare entity has a low correlation with atherosclerosis and atherosclerosis-occlusive heart disease. Clinical presentation can range from asymptomatic to catastrophic bowel ischemia or the fatal rupture of the superior mesenteric artery (SMA) aneurysm. Recently, the number of reported cases has increased with the development of diagnostic imaging techniques, such as CT angiography (CTA), multidetector computed tomography (MDCT), and multiplanar imaging, during investigations of abdominal pain or incidentally detected. The underlying actual course of SISMAD and its subsequent management strategies have been poorly established. Therefore, both operative and conservative approaches have been employed.

We report a case of a 60-year-old man who presented with epigastric pain. An imaging study revealed

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SISMAD type 2A without evidence of bowel ischemia or mesenteric hemorrhage and he was treated successfully with antiplatelets for more than 18 months.

Case Report

A man aged 60 years presented with epigastric pain for two weeks that worsened following taking of meals. However, there were no associated symptoms of fever, nausea, vomiting, constipation, or diarrhea. He had a history of previous treatment for hypertension and thyrotoxicosis that lasted for more than five years. A physical examination indicated mild tenderness at the epigastrium without guarding or rigidity, and no bruit was audible. The provisional diagnosis was the suspected early onset of acute cholecystitis or pancreatitis. However, ultrasound examination of his abdomen showed a fairly large proximal superior mesenteric artery at the origin, and demonstrated flow outside of the lumen of its native vessel. CT angiography of the aorta revealed isolated dissection of the superior mesenteric artery type 2A (Figures 1 and 2), in which the false lumen entry site began at 2.4 cm from the SMA origin (Figures 3 and 4), and the aneurysm sac extended for a length of 3.1 cm with distal false lumen thrombosis. The take-off origin for the replaced common hepatic artery from the SMA was 2.4 cm distal to the entry site of SMA dissection No thrombosis was seen in the true lumen distal to the dissection sac. Radiologic evidence of ischemia, such as bowel wall thickening, ascitic fluid or abnormal enhancement was not present. The entire aorta was of normal size and caliber but a fusiform isolated right common iliac artery aneurysm (2.5 cm in diameter) was found coincidentally, with no atheromatous plaque or calcifications. Therefore, conservative treatment with antiplatelets and close follow-up using ultrasonography and CTA was chosen for this patient.

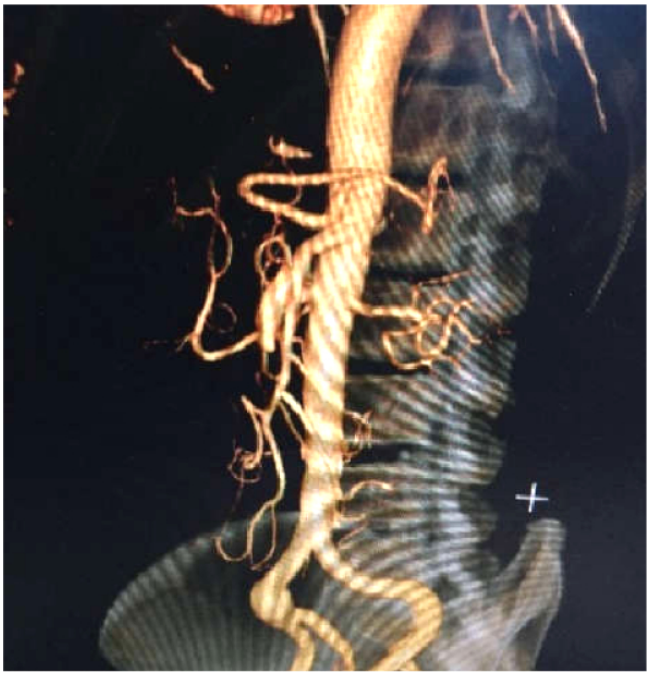


Figure 1 Coronal view 3D CTA of abdominal aorta

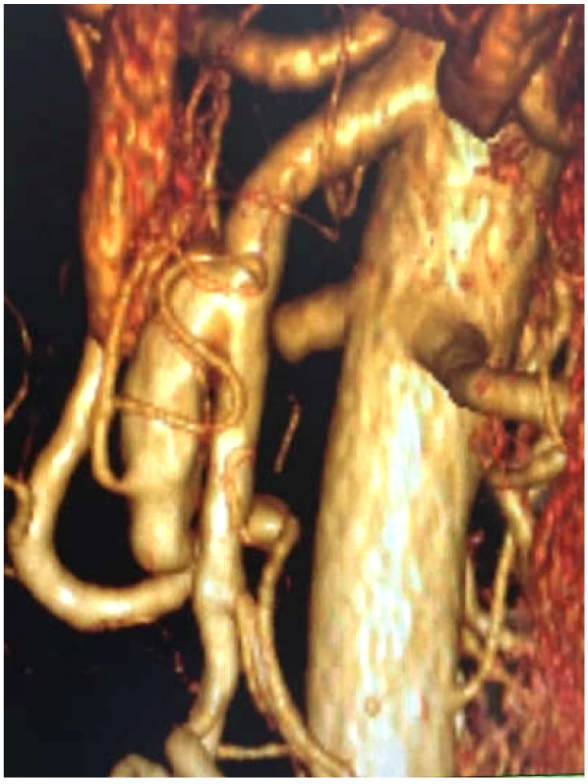


Figure 2 CTA of the SMA pseudoaneurysm

After 3 months of follow-up, the patient had no epigastric pain and the size of the dissection sac had slightly decreased, although the intimal flap was still present, as seen in the CTA in Figure 5.

After another 10 months, in December of 2018, the duplex ultrasound (shown in Figure 6) indicated that the dissection sac had a maximal diameter of 1.4 cm, with a small proximal thrombosis of the false lumen.

Finally, at the last follow up in April of 2019, the patient had no symptoms, the size of the dissection sac had decreased to a maximal diameter of 1.1 cm of the false lumen with a significant decrease of maximal flow velocity to 68.6 cm/sec and total thrombosis of false lumen as shown in Figures 7 and 8. The fusiform isolated right common iliac artery aneurysm did not change.

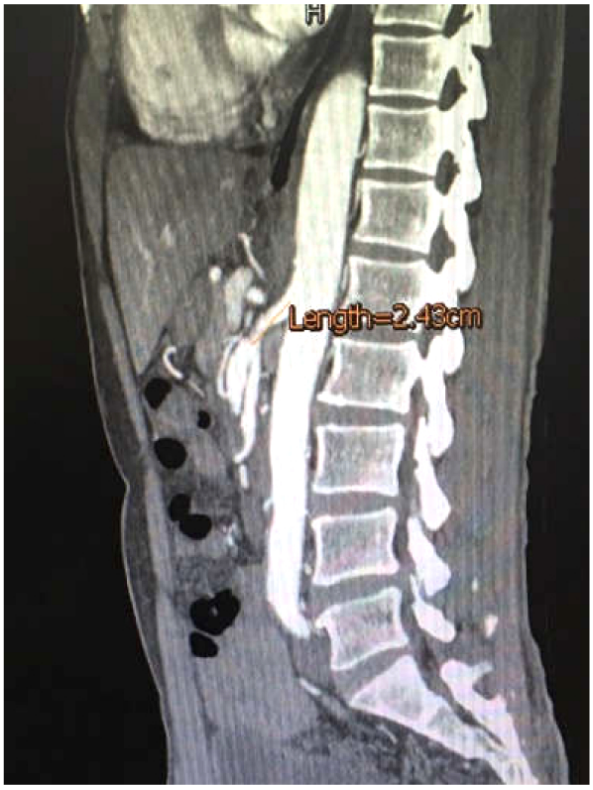


Figure 3 Dissection point of SISMAD is 2.4 cm from origin pseudoaneurysm sac is 3.1 cm

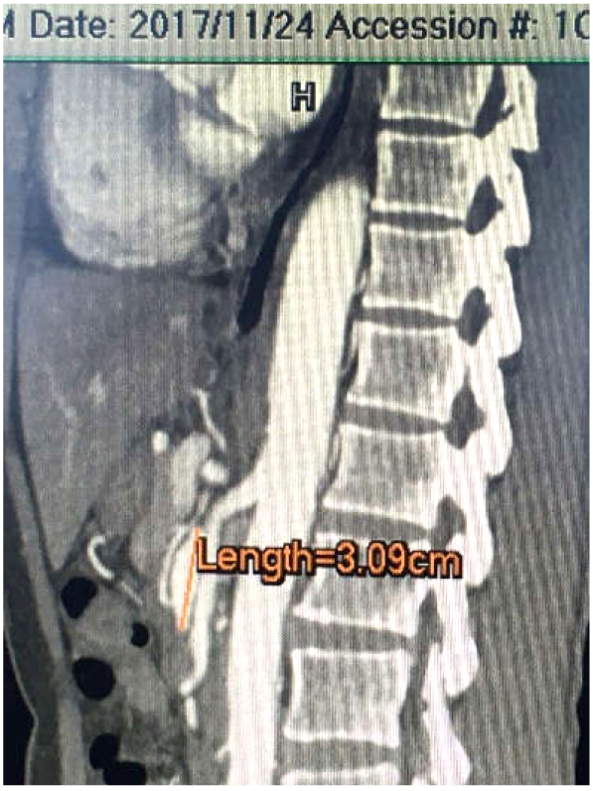


Figure 4 Length of SMA

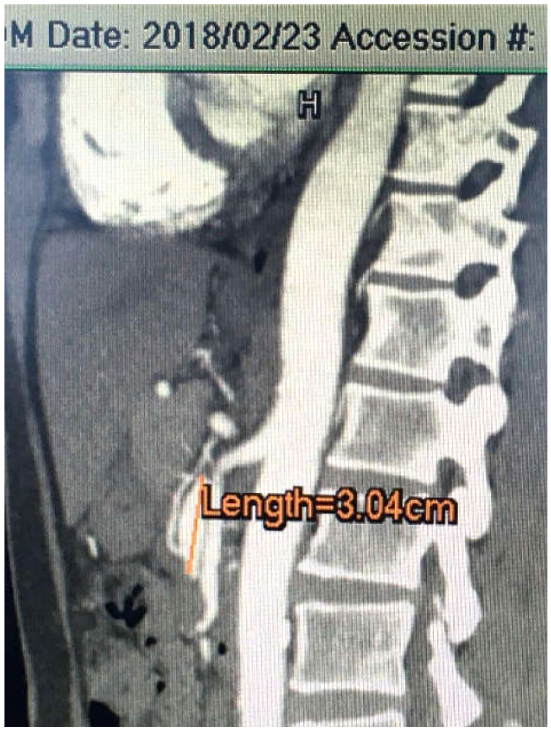


Figure 5 Length of SMA pseudoaneurysm sac is 3.04 cm, after 3 months of conservative treatment

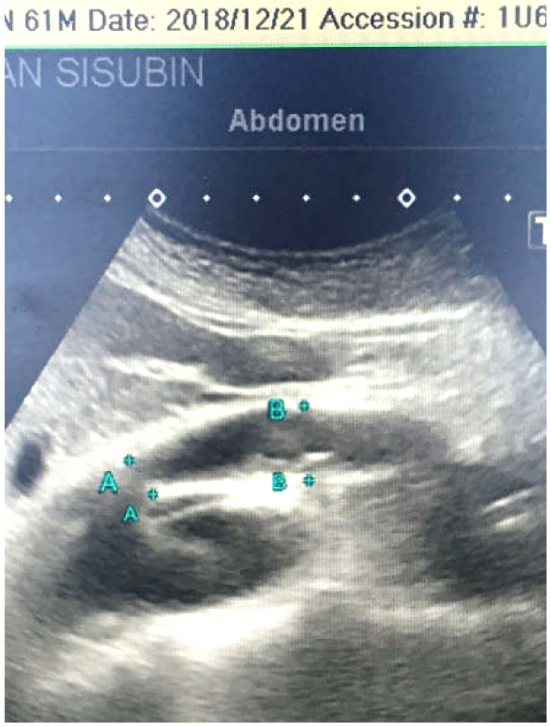
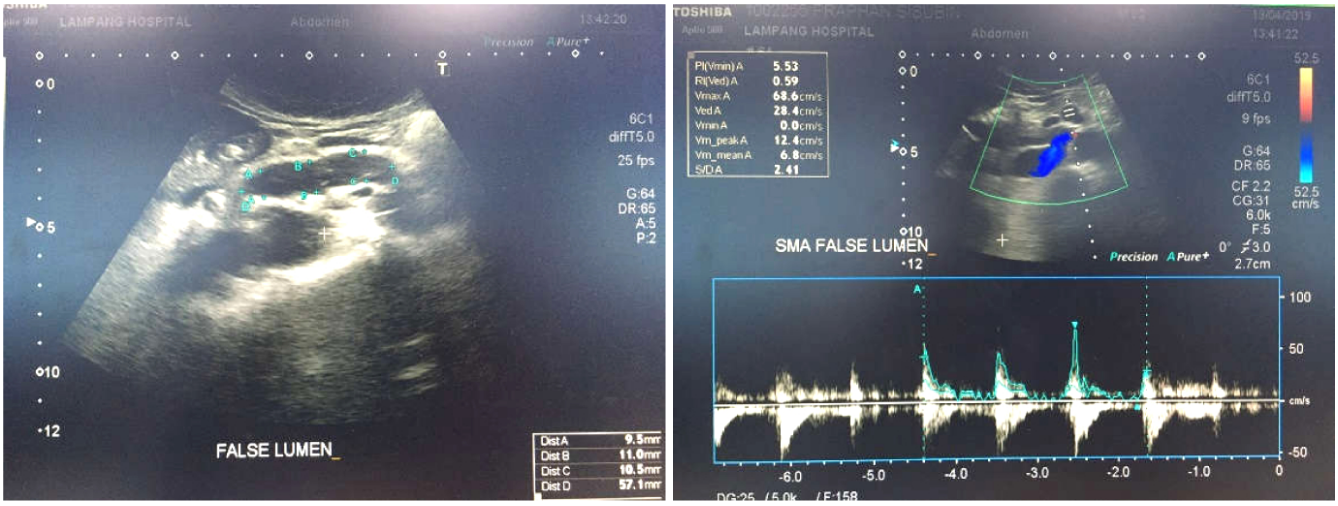


Figure 6 Duplex ultrasonography of the pseudoaneurysm of SMA after 13 months follow up showed thrombus in the aneurysmal sac and the sac size has decreased to 0.81 cm x 1.4 cm; distance A = 8.1 mm, B = 14.2 mm



Figures 7 and 8 Pseudoaneurysm of SMA after 17 months follow up; aneurysm sac size was decreased to 1.1 cm

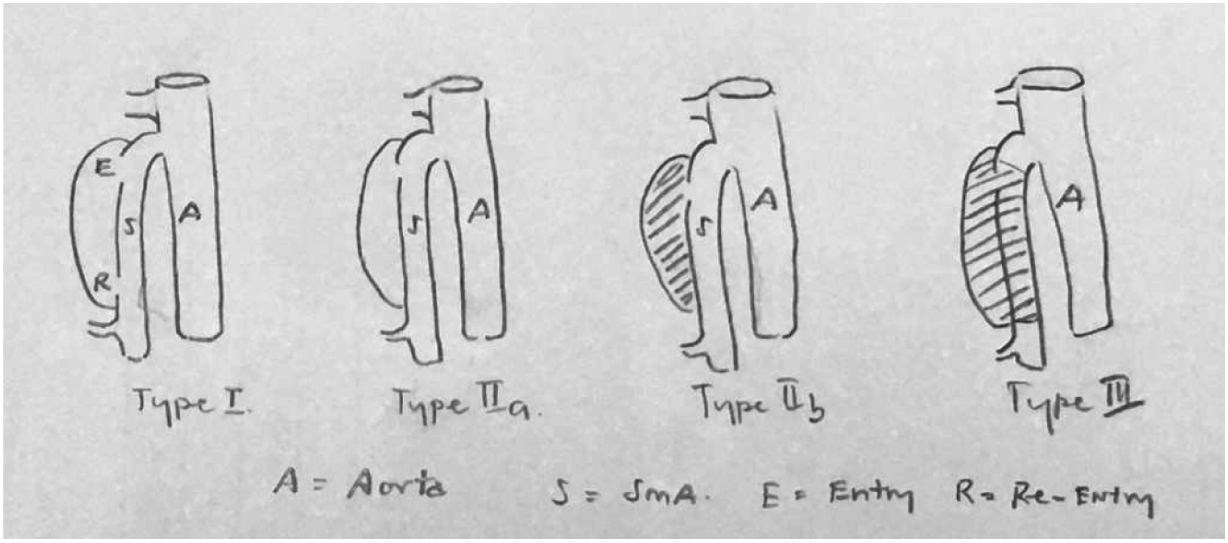


Figure 9 Types of SMA dissection: Type 1, patent true and false lumen with entry and re-entry sites; Type 2, patent true lumen but no reentry flow from the false lumen; Type 2a: visible false lumen (blind pouch of false lumen); Type 2b, no visible false lumen (thrombosed false lumen); Type 3, SMA dissection with occlusion of SMA

Discussion

Bauerfield described the first case of spontaneous isolated superior mesenteric artery dissection (SISMAD) in 1947, and the prognosis at the time was very poor[5]. The dissection was often associated with uncontrolled hypertension[6]. Hypertension was seen in 30% of SISMAD patients, and SISMAD was not associated with atherosclerosis. The origin of the dissection site corresponds to the inferior border of the pancreas, and the fixed retro-pancreatic portion is a continuation of the mobile mesenteric portion, which may result in shearing forces on the SMA wall.

In a previous case report, the patient's symptoms included acute epigastric pain with a duration lasting less than 4 weeks[7]. Associated symptoms, such as nausea, vomiting or abdominal distension, should raise concern for the presence of intestinal ischemia.

Laboratory investigations and plain radiography results are usually normal, but further ultrasonography should be done to evaluate the hepato-biliary system, pancreas, and the adjacent vessels. Any suspicious lesions seen on ultrasonography should be followed MDCT or CTA, specifically to confirm the diagnosis and also to categorize the SMA dissection, as recommended

by Sakamoto et al.[8] This classification was developed by Yun[11], which classifies SMA dissection into three types based on angiographic findings (Figure 9).

In these classifications, there was no relationship between radiological appearance and clinical course. currently there is no standard therapy for SISMAD, but treatment algorithms based on patient presentation and clinical course has been developed by Cho and Carter et al.[9-10] Symptomatic patients presenting with an acute abdomen (arterial rupture, intestinal ischemia, and necrosis) are managed surgically. Conservative approaches have been successful in the management of asymptomatic and even non-complicated symptomatic SMA dissection.

From the largest clinical series of SISMAD, the natural history of SISMAD after conservative treatment, clinical features, the relations between wall shear stress at the SMA and anatomic features of SMA[11] correlating with SISMAD types on CTA, found that 41% of asymptomatic patients had morphologic improvement and some had complete remodeling of the aneurysm, which was more frequent in type 2 lesions than in types 1 or 3 lesions.

Certain symptoms, especially epigastric pain, may suggest visceral ischemia, according to Cho et al.[10] A study conducted by Yun et al., however, found no correlation between pain severity and CTA type, but found a positive correlation between dissection length and an increase in perivascular inflammation which can elicit more pain[11].

At present, there is no clear preference for conservative treatment or surgical intervention in SISMAD without rupture or intestinal ischemia. Nagai et al.[12] suggested that the disease pattern of SMA dissection is similar to that of the internal carotid artery and thus the possible usefulness of anticoagulants. Indications for surgery as suggested by Sparks et al. include of the increasing size of an SMA aneurysm, SMA thrombosis, and persistent symptoms during anticoagulant treatment. Possible surgical or interventional procedures[13-16] include aortomesenteric or ilio-mesenteric bypass, thrombectomy, ligation and resection, thrombolytic or endovascular stent placement.

Conclusion

In our case report, after conservative management and follow-up with duplex ultrasound examinations, there was a decrease in the size of the false lumen, the slowing of the flow within the false lumen compared to previous examinations, and a resolution of symptoms. These results suggest and support the effectiveness of a non-operative approach for selected patients with SISMAD.

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บทคัดย่อ **รายงานผู้ป่วยมีอาการปวดท้องที่เกิดจากการกัดเซาะผนังหลอดเลือดที่ไปเลี้ยงลำไส้ในช่องท้องแขนงที่ 2 ชนิดที่เกิดขึ้นเอง**

อนุวัชช์ จันทร์ทิพย์

กลุ่มงานศัลยกรรม โรงพยาบาลลำปาง

ความเป็นมา: การกัดเซาะผนังหลอดเลือดที่ไปเลี้ยงลำไส้ในช่องท้องแขนงที่ 2 ชนิดเกิดขึ้นเอง มีโอกาสเกิดน้อยมาก และยังไม่มีแนวทางการปฏิบัติหรือการรักษาอย่างชัดเจนเมื่อทำการตรวจพบ

รายงานผู้ป่วย: ผู้ป่วยชายอายุ 60 ปี มีอาการปวดท้องบริเวณลิ้นปี่หลังรับประทานอาหาร เป็นมาประมาณ 2 สัปดาห์ ก่อนมาโรงพยาบาล ไม่มีประวัติการสูบบุหรี่หรือดื่มแอลกอฮอล์ มีประวัติการรักษาโรคความดันโลหิตสูงและ คอพอกเป็นพิษมาประมาณ 5 ปี ภายหลังจากตรวจพบว่ามีการเซาะของผนังหลอดเลือดที่ไปเลี้ยงลำไส้ในช่องท้องแขนงที่ 2 ผู้ป่วยรายนี้ได้รับการรักษาแบบประคับประคองโดยการใช้ยาต้านเกล็ดเลือด และได้ติดตามหลังการรักษาเกินกว่า 18 เดือน พบว่าผู้ป่วยมีอาการลดลง สามารถตรวจพบการเปลี่ยนแปลงของรอยโรคโดยการตรวจอุลตร้าซาวด์ร่วมกับการตรวจคอมพิวเตอร์หลอดเลือดช่องท้อง

สรุปผลการศึกษา: การรักษาแบบประคับประคองสามารถทำได้ในผู้ป่วยบางรายที่มีการเซาะของผนังหลอดเลือดที่ไปเลี้ยงลำไส้ในช่องท้องแขนงที่ 2 ชนิดที่เกิดขึ้นเองได้ แต่อาจจำเป็นต้องได้รับการผ่าตัด ในกรณีที่ตรวจพบว่ามีเลือดออก หรือเกิดการขาดเลือดของลำไส้
