

CASE REPORT

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A rare phenomenon of spontaneous subclavian artery aneurysm rupture: A care report

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ABSTRACT

Subclavian artery aneurysm (SAA) only occurs in approximately 0.1% of all atherosclerosis aneurysms. Spontaneous rupture of subclavian artery is an extremely rare phenomenon which can lead to mediastinal hematoma and subsequent cardiac death from multiple complications. Proximal and mid clavicular SAAs have higher incidence of rupture. Thrombo-embolic phenomenon is mostly correlated with distal SAA, but it also occurs in <9% of proximal SAA as well. Therapeutic options of SAA repair are open surgical repair or endovascular treatment. We present a case of spontaneous rupture of subclavian artery aneurysm rupture leading to mediastinal hematoma with hypovolemic shock and death.

Keywords: Aneurysm, Rupture, SAA, Spontaneous subclavian artery

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INTRODUCTION

Spontaneous subclavian artery rupture is an extremely rare phenomenon that can lead to mediastinal hematoma and subsequent cardiac death from multiple complications [1]. These complications can be hypovolemic shock, superior vena cava syndrome, or acute respiratory failure from tracheal obstruction. The most common complaint in these patients is chest pain (approximately 71% of the cases) [2].

We present a case of spontaneous subclavian artery aneurysm rupture leading to mediastinal hematoma with hypovolemic shock and death.

CASE REPORT

A 74-year-old male with current smoking history of 50 pack years, coronary artery disease status post (s/p) coronary artery bypass graft (CABG), hypertension, and dyslipidemia presented to the emergency room (ER) with complaints of substernal chest pressure for one day. Upon arrival to the ER, the patient was hemodynamically stable and mentioned that “I am having chest pains and I can’t breathe” and then became unresponsive. We were unable to obtain a pulse, hence, cardiopulmonary resuscitation (CPR) was initiated. The patient had a difficult yet successful via fiberoptic intubation and return of spontaneous circulation (ROSC) was obtained after three rounds of CPR. On physical examination, the patient was found to have new swelling of the head and neck. He was started on levophed and vasopressin for pressure support. Initial lab work showed hemoglobin of 12.1 with international normalized ratio (INR) of 1.84 and negative troponin. Electrocardiogram (EKG) was insignificant and showed normal sinus rhythm without any acute changes (Figure 1). Chest X-ray was positive for soft tissue prominence/upper mediastinal widening (Figure 2). Emergent computed tomography (CT) neck and chest was obtained, and significant findings were made: Large 3.5 cm calcified proximal left subclavian artery with aneurysmal rupture and mediastinal hemorrhage. In addition, large dense fluid collection extending from the neck to the esophageal hiatus consistent with hemorrhage. There is a resulting

mass effect on the trachea and mainstem bronchi and left atrium (Figure 3).

Repeat lab work showed hemoglobin of 8.7. Massive transfusion was initiated to stabilize the patient, but we were unable to do so. In the meantime, interventional radiology (IR) was consulted and plan to do angiogram was initiated. However, the patient continued to decline, and he went to cardiac arrest again due to multiple etiologies—hypovolemic shock from continued blood loss and acute hypoxic/hypercapnic respiratory failure from increase intra-thoracic pressure causing bronchial collapse. Bedside bronchoscopy was initially attempted to open the trachea without any success. The patient was pronounced deceased shortly before reaching the IR suite.

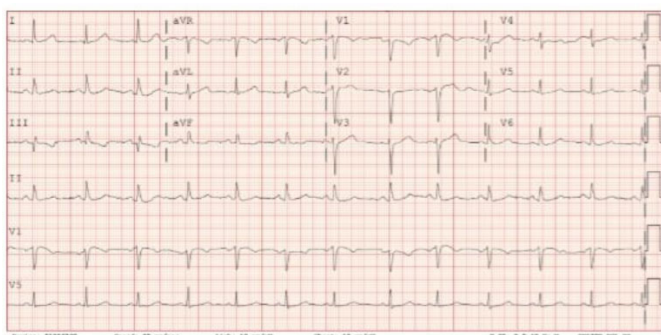


Figure 1: Normal sinus rhythm without any acute changes.

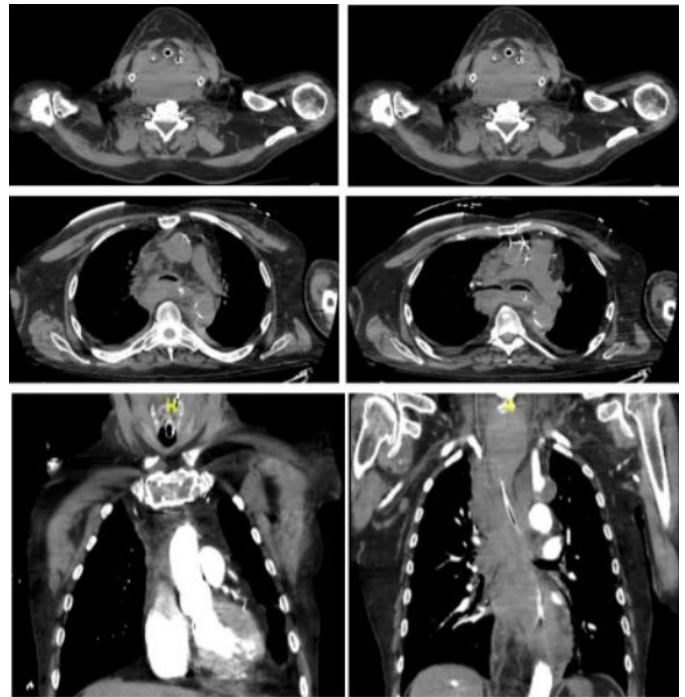


Figure 3: Large volume high density fluid collection and stranding extending from the lower neck to the esophageal hiatus. Peripherally calcified 3.5 cm aneurysm at the origin of the left subclavian artery with aneurysmal rupture and mediastinal hemorrhage. There is a resulting mass effect on the trachea and mainstem bronchi and left atrium.



Figure 2: Soft tissue prominence/widening along the right aspect of the upper mediastinum.

DISCUSSION

Subclavian artery aneurysm (SAA) only occurs in approximately 0.1% of all atherosclerosis aneurysms

as reported by Dent et al. [3]. It is associated with hypertension, atherosclerosis, trauma, tumor, congenital disease (Marfan syndrome, Aberrant artery malformation, and Neurofibromatosis), infection, or thoracic outlet syndrome. Proximal SAA is correlated majority with atherosclerosis (19%), congenital collagen disorders (18%), trauma (15%), and infection (14%) [4]. Based on the past medical history (PMHx) of our patient, it is safe to say that his SAA was most likely caused by atherosclerosis. Atherosclerotic SAA usually occurs predominantly in males and in ages over 60.

Subclavian artery aneurysm is asymptomatic until it progresses where it ruptures or forms thrombosis or embolizes where it is associated with significant morbidity or mortality. Anderson et al. did a meta-analysis of 394 cases of SAA and approximately 9% of patients had SAA rupture with associated mortality of 19% [5]. Proximal and mid clavicular SAAs have higher incidences of rupture. Thrombo-embolic phenomenon is mostly correlated with distal SAA, but it also occurs in <9% of proximal SAA as well [5]. In our case, the patient suffered catastrophic rupture of proximal SAA.

Mainly, SAAs are repaired early to avoid complications or to treat symptoms. However, there are no standard of care guidelines regarding the timing of treatment. Lawrence et al. reported that SAA repair only comprises approximately 0.5% of all aneurysm repairs [6].

Therapeutic options of SAA repair are open surgical repair or endovascular treatment. Conventional open surgical SAA repair is associated with 8% mortality [5]. Hence, endovascular repair (EVSAR) and hybrid procedure are preferred due to lower mortality and morbidity.

CONCLUSION

In conclusion, our case report highlights a rare case of proximal SAA aneurysm rupture associated with chest pain as the only presenting symptom. These types of cases are extremely rare and only few cases are reported in the literature. It should always be in the differential diagnosis as this type of arterial lesion is clinically significant due to its high morbidity and mortality.

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Author Contributions

Waheed Abdul – Conception of the work, Design of the work, Acquisition of data, Interpretation of data, Drafting the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Guarantor of Submission

The corresponding author is the guarantor of submission.

Source of Support

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Consent Statement

Written informed consent was obtained from the patient for publication of this article.

Conflict of Interest

Author declares no conflict of interest.

Data Availability

All relevant data are within the paper and its Supporting Information files.

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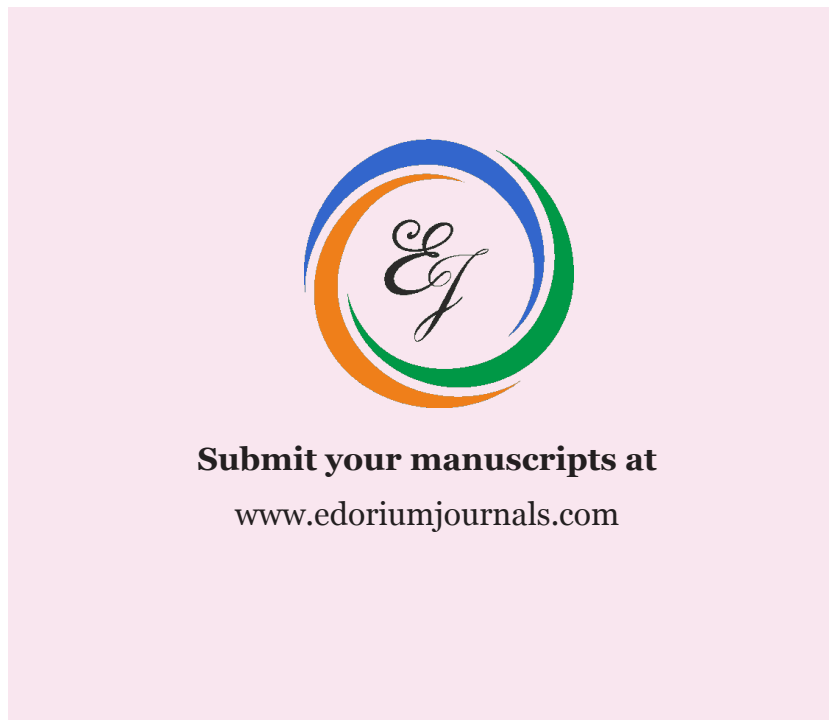
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