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SURGICAL RESOLUTION OF A MASSIVE, DEBILITATING LOW-GRADE INTRAMUSCULAR ARTERIOVENOUS MALFORMATION IN THE BACK: A LIFE TRANSFORMED

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ABSTRACT

Arteriovenous malformations (AVMs) are abnormal connections between arteries and veins, bypassing the capillary system, typically due to a failure in the differentiation process during embryonic development [1]. The size of AVMs increases as high-pressure blood flows directly from arteries to veins, following the path of least resistance [2]. This report presents a rare and remarkable case of a 45-year-old man with a massive, intramuscular AVM in his back. Despite the challenges posed by its size, volume, and potential risk to the thoracic cavity, the AVM was successfully treated with surgical excision alone. This intervention dramatically improved the patient's quality of life.

INTRODUCTION

Arteriovenous malformations (AVMs) are a rare congenital vascular anomaly characterised by abnormal connections between arteries and the veins bypassing the capillary system. AVMs can occur in various locations throughout the body. We present a case of a 45-year-old male presenting with a long standing, progressively increasing lump on the back. He was evaluated and diagnosed at a local tertiary centre as case of low flow AVM. He was treated conservatively with sclerosant for 5 years with no appreciable change in size of swelling. However, he developed skin ulceration at sites of sclerosant therapy.

Keywords: Arteriovenous malformations, MRA, ICD

Case Report :

A 45-year-old male presented with a mass on the anterior part of the left abdomen extending to the posterolateral aspect of the chest wall. It had gradually progressed in size over the last 6 months.

On examination there were 2 masses of which the smaller was of size 5 x 6cm that was present in the anterolateral aspect of the left abdomen and the latter was of size 25x 15cm present in the posterolateral

aspect of the left thorax. The masses were non tender, non-mobile, firm with ill-defined borders and had multiple dilated veins and bluish pigmentation on the surface of the skin (figure 1 a, b).

A doppler USG of the chest wall revealed tortuous network of dilated arteries and veins consistent with a low flow arteriovenous malformation. CT Angiography showed large ill- defined heterogenous lesion in subcutaneous left scapular region, posterolateral chest wall extending anteriorly with multiple cystic sinuses within it. On colour Doppler imaging it showed venous outflow pattern that was suggestive of the presence of the low flow AVM showing abnormal vascular flow and architecture involving the deep tissues of the chest wall and the muscles of anterior chest wall and abdomen.

Since it was a low flow AV malformation, Angio-embolization could not be done, hence surgical option was explored. The Intercostal muscles were spared and there was no intrathoracic extension of the AVM. The challenges we encountered during the surgery were:1) massive size and volume of the lesion, 2) intramuscular location, 3) skin involvement of the back, 4) no potential or feasible reconstruction apart from skin grafting, 5) potential thoracic breach.

The plan was 1) to address the anterior abdominal AVM first and identify the intramuscular plane for resection, 2) to resect the posterolateral wall lesion in the subcutaneous plane leaving a flap of skin over the large defect. The patient underwent successful surgical excision of the arteriovenous malformation under general Anaesthesia. Intraoperatively, the anterior AVM was found to be between external and internal oblique muscle and the posterior AVM was found to be involving the Latissimus Dorsi muscle that was carefully dissected out sparing the intercostal muscles. a prophylactic intercostal drainage procedure (ICD) insertion was done. (figure 2). Postoperative recovery was uneventful, the posterior

flap developed skin necrosis which was managed conservatively and the patient underwent skin grafting 1-month post-surgery with complete uptake. 6 months post-surgery follow up, the patient was asymptomatic with good range of movements on the operated side. The resected masses were sent for histopathological review that showed skeletal muscle and mature adipose tissue with diffuse vascular proliferation of varying calibre lined by flattened endothelial cells with no atypical cells seen (figure 3 a, b) and was consistent with the clinical diagnosis of AV malformation.

DISCUSSION

AV malformation can be defined as abnormal communication between an artery and vein, that is bypassing the capillary system. It is usually a congenital anomaly due to failure of differentiation into artery and vein from the common embryonic anlage [1]. These lesions enlarge owing to high phase of flow from the artery to vein following the path of lesser resistance [2]. Here we present a case of 45-year-old with a posterolateral chest wall AVM. Early identification and illustration of the involved vascular structures is important for the planning and effective management of AVMs. A more precise imaging helps to identify the feeding and draining vessels before resection even though a doppler ultrasound can be used to identify the existence of the AVMs. Contrast enhanced computed tomography can be used to delineate the associated vessels and to identify the surrounding soft tissue structure [3,4]. Magnetic Resonance Angiography (MRA) is able to identify multiple concurrent AV malformation and provides the added benefit of avoiding ionizing radiation and iodinated contrast. It is therefore recommended to monitor the postoperative response [5], however we MRA is expensive imaging and does not prove to be superior to CT angiography in delineating the vascular anatomy.

Treatment plan must cater to the individual and be customised to their vascular anatomy noting the feeding and the draining vessels. Surgical resection is considered as a gold standard procedure for the treatment of AVMs since decades, however the bleeding potential always pose a greater risk [6]. To avoid the above said risks catheter embolization has been describes as a less invasive procedure at AVM extirpation, unfortunately this procedure requires multiple setting and have high rate of recurrence or soft tissue necrosis due to ligated vessels [3,7]. Surgical resection was the preferred mode of management in our case thereby reducing the risk of recurrence in this giant chest wall AVM.

CONCLUSION

A Giant Chest wall AVM is a very rare lesion which poses interesting therapeutic dilemmas. There is a paucity of published case reports of Giant chest wall AVM being treated with surgical resection alone due to 1) massive size and volume of the lesion, 2) intramuscular location, 3) skin involvement of the back, 4) no potential or feasible reconstruction apart from skin grafting, 5) potential thoracic breach. making this case a rare and exciting case [8,9]. Early diagnosis and treatment of large symptomatic case is always needed to prevent the dangerous sequelae of bleeding, loss of function or even high output cardiac failure. Here we present a novel example of performing a surgical resection combined with reconstruction of the wound by skin grafting proving to be effective in the treatment of massive posterolateral chest wall AV malformation.

LEGENDS



Figure 1(a, b): preop pictures showing as large posterolateral chest wall AVM





Figure 2a : intraoperative picture showing the resected specimen of the AV malformation



Figure 2b : intraoperative picture showing the bare chest wall following resction



Figure 2c: postoperative photo of the resected area



Figure 3a,b: Histopathology Images

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