Intimomedial mucoid degeneration resulting in a dissecting infrarenal abdominal aortic aneurysm in a young Middle Eastern male: a case report

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Introduction and importance: Intimomedial mucoid degeneration is a rare vascular disorder first depicted in 1977. It involves mucin deposition in arterial layers. This will cause elastic tissue degeneration and aneurysm formation. This pathology predominantly affects the aorta. However, it could involve other smaller vessels. Surgical treatment could become complicated by a bleeding diathesis. Therefore, a precise surgical technique is necessary to avoid the ensuing complications.

Case presentation: We present the case of a previously healthy 27-year-old Middle Eastern male who presented to our surgical clinic following the incidental discovery of an infrarenal abdominal aortic aneurysm following a blunt trauma to the left flank incurred during a fall. Preoperative radiology unveiled a dissecting an infrarenal aortic aneurysm with a concurrent dissection flap at the left renal artery level. Furthermore, an additional dissection flap was observed at the abdominal aortic bifurcation devoid of thrombosis.

Clinical discussion: We planned to perform a bilateral aortoiliac bypass. However, due to the fragility of the artery wall, bleeding diathesis, and the tearing that occurred due to the anastomotic suture, the irreparable tear in the anastomosis complicated the situation, we decided to ligate the aorta and perform an axillary-bi-femoral bypass.

Conclusion: Intimomedial mucoid degeneration presenting as a dissecting infrarenal abdominal aortic aneurysm is an exceptionally rare pathology. This underscores the crucial need for extensive epidemiological research to document and raise awareness about these cases. Our literature review confirms that our case is the first documented instance in our country, and this emphasizes the significance of our findings.

Keywords: abdominal aortic aneurysm, case report, dissecting abdominal aortic aneurysm, intimomedial mucoid degeneration, vascular emergency, vascular surgery

Introduction

Decker et al.[1]. pioneered the depiction of Intimomedial Mucoid Degeneration (IMMD) in 1977. It represents a profoundly rare vascular pathology distinguished by the accumulation of mucin within the intimal and medial layers. In turn, this results in the degeneration of elastic tissue and the genesis of arterial wall aneurysms[1–4]. While the initial understanding confined IMMD

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HIGHLIGHTS

- The prevalence of aortic aneurysms stands at 1–2% and around 10% in the elderly.
- Intimomedial mucoid degeneration is a rare pathology that results in arterial aneurysms.
- The gold standard treatment method such aneurysms is open surgical repair.
- The definitive diagnosis is reached only after thorough histopathological analysis.
- Ours is the first ever documented case from our country of such an occurrence.

to the aorta, successive literature has extended its manifestation to encompass major aortic branches and lesser-sized blood vessels like the brachial and coronary arteries[1,5–8]. Predominantly, individuals with documented IMMD exhibit a notable prevalence among the black population (87%) and a predilection towards the female gender (74%). Notwithstanding, occurrences in men and other ethnicities have been documented[2,4]. Aneurysms in the context of IMMD typically assume either a saccular or fusi-form morphology. This elicits symptoms contingent upon their anatomical location[6,8]. IMMDs are characterized by a phenomenon of a bleeding diathesis. This could encumber the surgical interventions. Disparate from Disseminated Intravascular Coagulation (DIC), this phenomenon is amenable to resolution

following the surgical amelioration of the afflicted vessel[4]. Therefore, the execution of surgical procedures demands immaculate precision as a crucial prerequisite to optimal patient outcomes[4,8].

The work has been reported in line with the SCARE criteria and the revised 2023 SCARE guidelines[9].

Presentation of case

Patient information

We present the case of a 27-year-old male of Middle Eastern descent. This patient had no known previous medical illnesses, and he was referred to our university hospital's Vascular Surgery clinic with the chief complaint of abdominal pain. The onset of this pain traced back to an incident of falling from a height of 2 m 7 months prior. That inflicted significant blunt trauma to the left flank and shoulder. Afterwards, he underwent triage at the emergency room whereby the doctors noticed abdominal bruising over the left flank. During his preliminary evaluation, he underwent an abdominal ultrasonography scan that incidentally demonstrated the an infrarenal abdominal aortic aneurysm with a diameter of ~5.5 cm. The patient was therefore directed to our specialized clinic for further evaluation of the aneurysmal discovery. The abdominal pain was described as diffuse, dull, and intermittent, and was rated at 03/10 on the patient's numerical pain scale. Initially, it exhibited a partial response to over-the-counter analgesic. However, it became refractory to such interventions as time progressed. Notably, the pain did not radiate to other regions and lacked discernible triggering factors. Absent were symptoms such as intermittent claudication, coldness, lower limb swelling, pallor, or cyanosis. The patient denied any history of immune disorders or recent infections. Psychosocially, the patient had a 5-pack-year history of smoking, whereas the surgical, family, and allergic histories yielded no relevant findings. His BMI was 26 Kg/m².

Clinical findings

Upon physical examination, the patient exhibited stable vital signs. Commencing with inspection, the abdomen displayed symmetrical movement harmonizing with respiration, but with notable bruising in the left flank. Markedly, palpation revealed the presence of a pulsatile periumbilical abdominal mass accompanied by mild tenderness. Additionally, the arterial axes in both lower limbs exhibited no abnormal findings. Auscultation unveiled a localized bruit overlying the site of the aforementioned pulsatile mass. Beyond these notable findings, the remainder of the physical examination yielded no results.

Diagnostic assessment

Utilizing duplex ultrasound, we identified an infrarenal aortic aneurysm measuring 5.5 cm. It spanned from the renal arteries to the abdominal aortic bifurcation with no evidence of additional aneurysms. Subsequent to this, a detailed contrast-enhanced Multi-Slice Computed Tomography (MSCT) scan with three-dimensional reconstruction of the abdomen and pelvis corroborated the presence of the aforementioned aneurysm. It also revealed an associated dissection flap at the left renal artery level characterized by a true lumen. Additionally, another dissection flap was observed at the abdominal aortic bifurcation without

thrombosis (Fig. 1 A–G). The remainder of the arterial tree was free of any other abnormalities. A comprehensive laboratory panel encompassing coagulation, inflammatory, and infection markers yielded results well within normal ranges. Given this clinical profile, surgical intervention emerged as the preferred treatment modality. Prior to surgery, the patient underwent a nil-per-mouth nutritional regimen, had proper intravenous access for essential infusions set-up, and received the appropriate pre-operative antibiotics. Remarkably, no challenges or deviations from the established treatment plan were encountered throughout all perioperative phases.

Therapeutic intervention

The surgical intervention for our patient was executed at our tertiary university hospital and was conducted under general anaesthesia. The procedure was performed by two seasoned Vascular Surgery specialists, each with 17 years of extensive surgical experience. Based on the rheumatological consultation, our existing laboratory results, radiological signs, and the young age of the patient, we suspected a connective tissue disorder. We approached the aneurysmal sac (Figure 2A, B) by a longitudinal abdominal midline incision that opened the retroperitoneal space. The neck of the aneurysm was juxtarenal where there was less than 1 cm to renal arteries. We achieved a suprarenal exposure by ligation of the left renal vein and isolated the renal arteries. The superior mesenteric artery was about 1 cm above the renal arteries. Also, we isolated the aortal bifurcation. After that, we administered diuretics (Mannitol) and Heparin. Then, distal control of iliac arteries and suprarenal aorta was accomplished. We opened the aneurysmal sac; the arterial wall was extremely delicate, and no clots were found. The superior dissection flap was extended to suprarenal arteries, and the left renal artery was found to be originating from the false lumen. This countered the MSCT findings. At that point, the proximal anastomosis was done with DACRON reinforcement patches utilizing Proline 3/0 sutures and surrounding the neck of aorta with a first clamping time of about 28 min. However, when we opened the aorta to flush the anastomosis, there were two points of leakage which could be held with little compression in atherosclerotic arteries. Lacerations developed in these points, and that accelerated with every heartbeat and extended to more points from the anastomosis that could not be dealt with by tamping or reinforcement sutures even despite the attempts to do so. The anastomosis could not be repaired. Therefore, we replaced the clamp over the renal arteries, and we tried to sow the aorta, but we were unable due to the tear in the wall and its fragility. Therefore, it was decided to perform the aortic ligation. The ligation was made above the left renal artery with a second clamping time of 20 min. It was sufficient to control the bleeding. We took into consideration the suprarenal clamping time, the volume of blood lost, the short aortal neck below the renal arteries, and the tenuous and friable artery. In such circumstances, ligation of the aorta and common iliac arteries and extra-anatomical bypass was decided. We ligated the aorta above the left renal artery and kept the right renal artery intact. The axillary artery and femoral arteries were found to be as fragile and delicate as a leaf, similar to the aorta. Therefore, a right axillo-bi-femoral bypass was performed using a reinforced ePTFE bypass graft
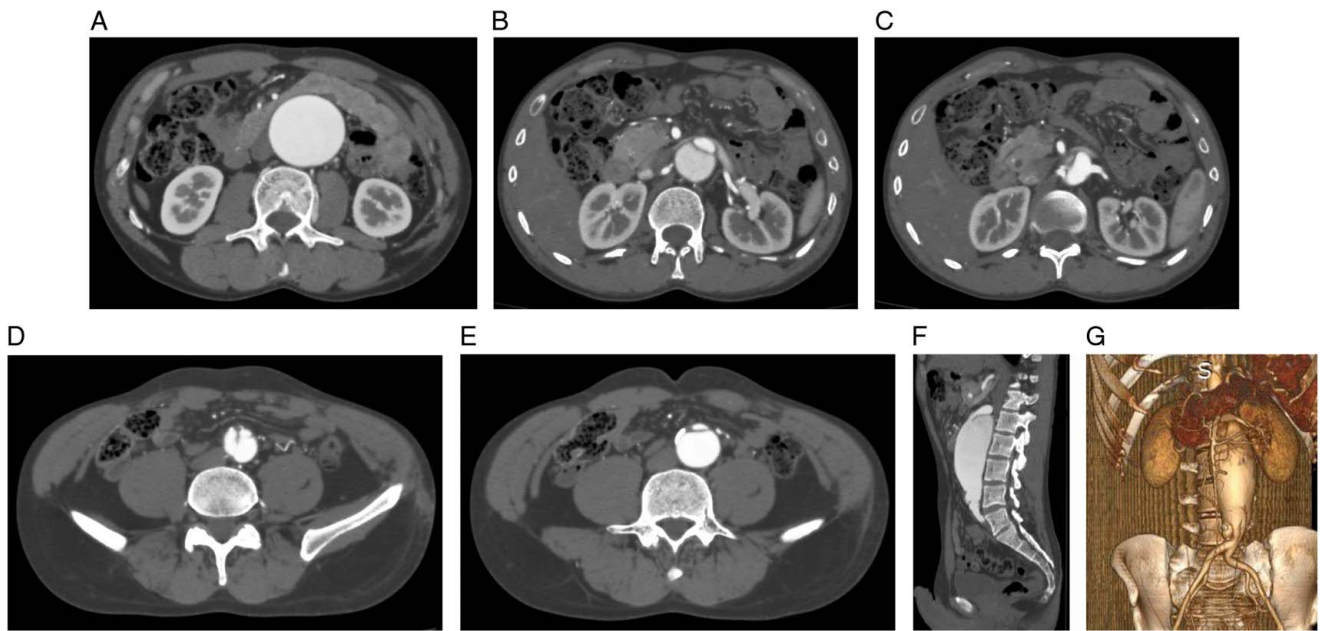
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Figure 1. (A) Preoperative contrast-enhanced Multi-Slice Computed Tomography (MSCT) image of the abdomen and pelvis in axial view. It demonstrates the infrarenal abdominal aortic aneurysm measuring 5.5 cm whereby its outer surface appears extremely thin. (B, C) Preoperative contrast-enhanced MSCT image of the abdomen and pelvis in axial view. It reveals a dissection flap at the neck section of the aneurysm at the level of origin of the renal arteries. (D, E) Preoperative contrast-enhanced MSCT image of the abdomen and pelvis in axial view. It reveals a dissection flap at the end of the abdominal aorta prior to the origin of the iliac arteries. (F) Preoperative contrast-enhanced MSCT image of the abdomen and pelvis in sagittal view. It demonstrates both of the dissecting flaps mentioned in the previous figures and shows how the flaps are free as we have a true arterial lumen. (G) Preoperative contrast-enhanced MSCT image of the abdomen and pelvis in coronal view with three-dimensional reconstruction showing the infrarenal abdominal aortic aneurysm.

7×80 mm from the right axillary artery to the right common femoral artery, and a femoral-femoral bypass was done to secure the arterial flow to the left inferior limb. The pelvis perfusion was secured by the retro-flow through the external iliac artery and internal iliac artery of both sides. The arterial flow was restored to the inferior limbs. Postoperative viability
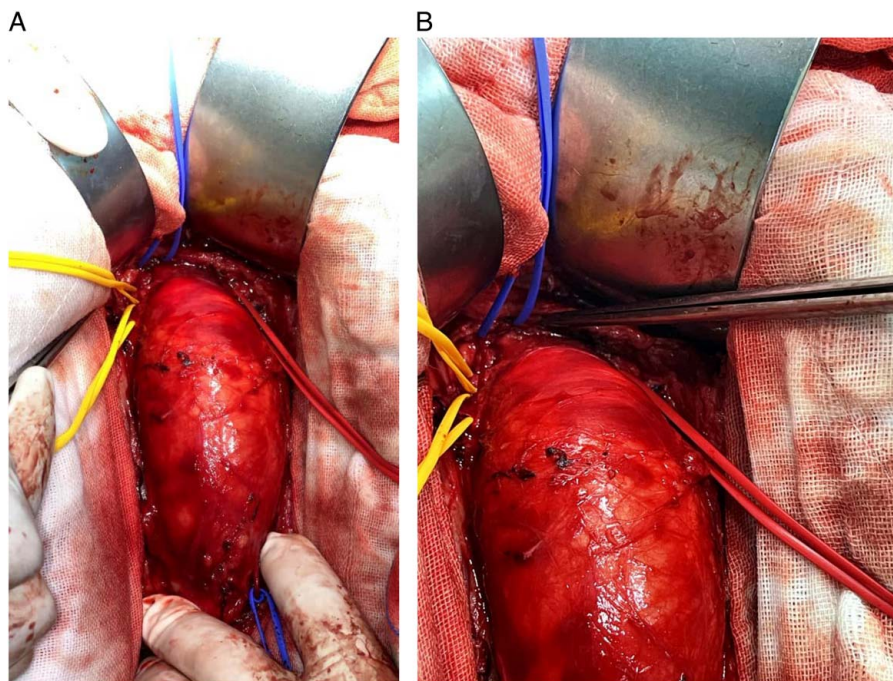

Figure 2. (A, B) Intraoperative image demonstrating dissecting infrarenal abdominal aortic aneurysm that was precipitated by intimomedial mucoid degeneration.
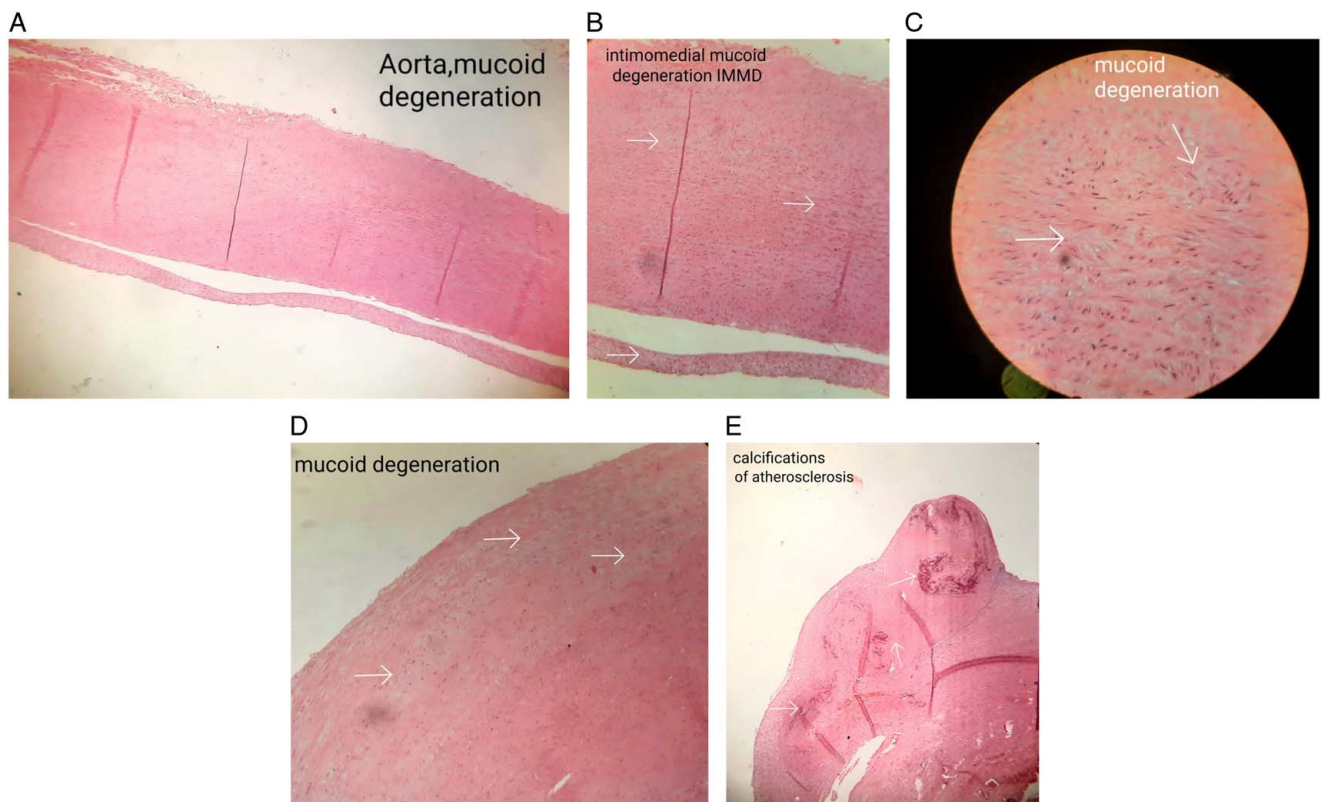
Figure 3. (A–E) Images from the histopathological analysis of the aneurysmal specimens via Hematoxylin and Eosin staining. In (A), we can see the aortic tissue along with mucoid degeneration (10× magnification). In (B), we can see the intimomedial mucoid degeneration as annotated by the white arrows (20× magnification). In (C), we can better visualize the mucoid degeneration as annotated by the white arrows (40× magnification). In (D), we can see the mucoid degeneration as annotated by the white arrows (20× magnification). In (E), we can see the atherosclerotic calcifications as annotated by the white arrows (10× magnification). we can see the elastic tissue degeneration, Mucin deposits in the media and intima, foci of calcification, and fibrin deposits.

of the lower extremities and patency of the bypass grafts were assessed by means of bedside duplex ultrasound. The results were normal as no free fluids or pseudoaneurysms were noticed, the graft was functioning as it should be, and the lower extremity pulses were normal. Meanwhile, the mean blood pressure dropped to 60 mmHg, and the patient lost about three liters of blood. More than 16 units of full blood count, fresh frozen plasma, and platelets were transferred. The arterial blood flow was diminished in the inferior limb for about 5 h. The acidosis that accompanied the restored flow of the inferior limbs, the volume lost, blood derivatives infusion, and the resultant DIC, and the acute kidney insufficiency due to volume loss put the patient at high risk of multi-organ failure. As a result, the patient left the operation room on pumps to support life. He remained in the intensive care unit for about 38 h in a shock state with pumps to support life. He
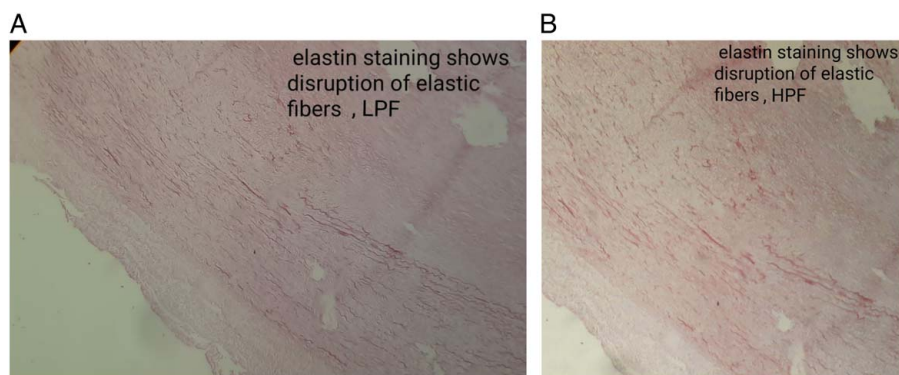

Figure 4. (A, B) Images from the histopathological analysis of the resected specimens in low-power field (20× magnification) and high-power field (40× magnification), respectively. Elastin staining reveals the disruption of elastic fibres.

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was given antibiotics, high volume, and blood transfusion. Nevertheless, no diuresis or dialysis were performed due to his low blood pressure (even with the pumps). Eventually, he passed away due to multi-organ failure. Based on the previous intraoperative findings, tissue biopsies from the arterial walls of the abdominal aorta and the subclavian artery were taken to investigate for the potential presence of an underlying soft tissue degenerative disease. The subsequent analysis via Hematoxylin and Eosin (H&E) of both biopsies revealed elastic tissue degeneration with deposits of mucin in the media and intima in addition to the presence of foci of calcification with fibrin deposits (Fig. 3A–E). Further analysis via Elastin staining further depicted the disruption of elastic fibres (Fig. 4A, B). Findings conform to the diagnosis of Intimomedial Mucoid Degeneration that directly resulted in a dissecting aneurysm. Other biopsies were taken from the axillary and femoral arteries. They revealed similar findings.

Discussion

Within the general populace, the prevalence of aortic aneurysms stands at 1–2%. This percentage was seen to escalate with advancing age to ~10% among older individuals[10]. The history of IMMD can be traced back to its initial documentation in South Africa with Pepler's pivotal publication dating back to 1955[11]. However, the nomenclature *"Intimomedial Mucoid Degeneration"* was coined in 1977 by Decker *et al.*[1]. where it was elucidated in a case series involving a number of nine patients afflicted with aortic aneurysms. In terms of disease progression and origin, the pathophysiological keystones of IMMD entail the gradual accumulation of mucin within the intima and media. This takes place specifically within their extracellular compartment. This mucin accrual precipitates the breakdown of collagen fibres. Also, it precedes the deterioration and disintegration of the elastic fibres within the media[2,4,12]. Mucin vesicles are also discernible within the vascular smooth muscle cells. In this process, they instigate cellular necrosis in a progressive manner. Said necrosis initially occurs in the cytoplasm and subsequently within the nucleus[4]. In principle, cells undergo classical necrotic processes including karyorhexis, pyknosis, and karyolysis. The ensuing extracellular mucin accumulation infiltrates and relocates collagen fibres. In turn, this fosters connective tissue disbanding and jeopardizes the structural integrity of the cellular walls leading aneurysmal formation[13]. In terms of the affected anatomical locations, IMMD exhibits its lethal impact not only on the aorta but also extends its reach to encompass large and medium-sized arteries[2,4,8,12]. We must note though that the aorta and its principal branches including the subclavian, carotid, superior mesenteric, iliac, and femoral arteries, are the primary anatomical components affected by this pathology[8]. Nevertheless, recent accounts have delineated smaller vessels succumbing to IMMD, such as the coronary[6], brachial[5], dorsalis pedis, and temporal arteries[4]. In terms of the population-specific disease occurrence, this disease exhibits features consistent with its initial description in a predominantly South African population, primarily comprising black females. Yet, instances of this pathology presenting in individuals who are males and also from other ethnic groups (i.e. European and Indian descents) have also been reported in the contemporary literature[2–4,14]. On this note, Abdool-Carrim *et al.*[8]. reported an approximate prevalence of 3.5% of all non-traumatic aneurysms in the South African black population groups.

Additionally, it was noted that the affected individuals developed aneurysms at a younger age compared to atherosclerotic patients (52 compared to 65 years of age). In terms of patient presentation, we must bear in mind that the clinical manifestations in patients with IMMD come in the form of localized symptoms contingent upon the anatomical location of the aneurysms[5,11]. Such symptoms occur in the form of the presence of a pulsatile mass, back pain/discomfort, abdominal pain, extremity vascular claudication, and symptoms stemming from aneurysmal leakage due to rupture or dissection[8]. In this regard, the infrarenal aorta constitutes the most common site of involvement. It is then trailed by the thoracic aorta, subclavian, common carotid, and common iliac arteries[8]. Morphologically, it is worthy to note that aneurysms in IMMD patients typically exhibit fusiform or saccular characteristics[7]. In our case, the aneurysm was fusiform in appearance. In terms of obtaining a preoperative insight with clues toward the existing diagnosis, numerous diagnostic imaging methods are available and they include duplex ultrasound, computed tomography angiography, and magnetic resonance angiography. Moreover, they were found to be instrumental in ascertaining the disease extent and potential aneurysmal dissection[7,8]. The literature underscores a conspicuous characteristic of IMMD that provides us with a clue in the preoperative diagnostic phase. This refers to the nearly complete dearth of a luminal thrombus within the aneurysmal sac. This is witnessed intraoperatively during the aneurysmal repair stage. An intriguing hypothesis posits the existence of a potential fibrinolytic process instigating directly from the aneurysmal locus in IMMD. This theoretical framework not only clarifies the conundrum surrounding the rareness of luminal thrombus, but also provides a cogent rationale for the proclivity of affected patients to manifest haemorrhagic complications during the surgical restitution of IMMD-associated aortic aneurysms[2,4,12]. Rare pathologies like IMMD always pose diagnostic challenges for physicians. On that note, IMMD's lack of characteristic clinical presentations necessitates reliance on histological elements for the establishment of an accurate diagnosis[12]. In terms of microscopic features of IMMDs, the prime histological attributes encompass intimal and medial thickening due to mucin buildup. This process subsequently yields Elastin fibre fragmentation and pathological amalgamation[7,15]. This deteriorated cellular wall integrity ultimately precipitates the formation of an aneurysm[4,15]. A distinctive feature that guides pathologists in establishing a diagnosis during the histological analysis of IMMD is the conspicuous absence of any inflammatory reaction[1] as was evident in the analysis of the specimens in our case. Since the establishment of a definitive diagnosis is based on the merits of proper histopathological examination, we must bear in mind that distinction from other disorders needs to be made. This is especially important because IMMD share multiple pathological features and histological similarities with a select few other disorders. For example, the abnormal mucin deposition akin to IMMD could potentially lead to confusion with the more widespread vascular disease termed *"Mucoid Extracellular Matrix Accumulation"*, which is formerly known as *"Cystic Medial Necrosis"*[12,16]. Mucoid Extracellular Matrix Accumulation is linked to various genetic conditions, but differs from IMMD in its confinement to the media layer and exclusively affecting the aorta. Mucoid extracellular matrix accumulation's histology typically reveals an augmented mucoid extracellular matrix whether it is within the lamella or trans-lamellar enlargements of the media[16]. Another potential source of confusion arises from the close proximity of Intimomedial Mucoid Degeneration with another rare

anomaly denominated as *"Cystic Adventitial Disease"*. Cystic Adventitial Disease exhibits a marked predilection for the male demographic and manifests as unilateral lesions affecting the adventitia of peripheral blood vessels. This pathology particularly involves the following vessels: Femoral, popliteal, external iliac, radial, and ulnar arteries[17]. When we discuss aetiology of this pathology, Cystic Adventitial Disease remains a subject of debate. This gives rise to various hypotheses that encompass degenerative processes, trauma, and systemic diseases. Conspicuously, histological examination of cystic adventitial disease unveils the presence of multi-locular gelatinous or mucinouscysts within the adventitial layer[18]. In terms of treatment of IMMDs, the established modality for addressing aneurysms associated with IMMD is grounded in open surgical repair as substantiated by the available scientific literature[7,8]. Intraoperatively, we can also attempt to differentiate IMMDs from other potential pathologies. For example, an intrinsic characteristic distinguishing IMMD is the marked scarcity of luminal thrombus within the aneurysmal sac[5]. Intriguingly, patients undergoing operative intervention for IMMD often encounter dangerous levels intraoperative bleeding[7]. This is a phenomenon exacerbated by surgical handling and promptly ameliorated upon the successful repair of the aneurysm. This diathesis lends credence to the hypothesis that a primary fibrinolytic process emanates from the diseased aneurysm, thereby clarifying the infrequent occurrence of thrombus in IMMD. This also explains the reason why IMMDs are extremely rare to be found with an occlusive vessel disease other than numbered cases documented in the available literature[2,6–8]. This characteristic sets IMMD apart from disseminated intravascular coagulation[4]. Patients afflicted with IMMD typically exhibit diminished platelet and fibrinogen levels alongside reduced levels of factors V and VIII[4]. This culminates in accelerated fibrinolysis as evidenced by a diminished euglobulin lysis time[4]. A distinguishing feature to look for when assessing IMMDs' bleeding diathesis is that, unlike DIC, positive fibrin monomer and elevated D-dimer levels are conspicuously absent in IMMD[4,7,8]. Cognizant of the proclivity for bleeding tendencies in IMMD patients, surgical operators must exercise heightened awareness during these procedures[8]. Additionally, the arterial wall in IMMD cases often presents as friable, and is known to have a predisposition to facile dissection during suture mending. Consequently, a meticulous suture technique becomes indispensable in mitigating potential complications[7,8]. The perioperative phase necessitates close monitoring and rectification of the coagulation profile and platelet dysfunction. Furthermore, the prognosis in IMMD hinges crucially upon the extent of the disease and the timing of its clinical presentation[7]. In terms of morbidity and mortality in patients with aneurysms associated with an IMMD, acute patient presentations featuring dissection correlate with heightened morbidity and mortality rates[7]. On this note, unfavourable consequences are intrinsically linked to major blood loss necessitating extensive transfusions and the concomitant complications thereof. Multi-organ failure as a result of shock further compounds the deleterious consequences of acute presentations in such patients[8].

Conclusion

IMMD represents an exceedingly rare pathological phenomenon. It is even rarer when it results in a dissecting abdominal aortic aneurysm. Encountering such a pathology is extraordinarily infrequent within the field of Vascular Surgery. That prompts shifting attention for thorough exploration in the epidemiological and research domains. This will take place via meticulous documentation of such cases which will foster awareness that can contribute to suspecting this diagnosis and the implementation of the necessary therapeutic interventions. It is vital to remember that there is increased intra- and postoperative risk of haemorrhaging due to friability of arterial walls because of the subsequent dysregulation of thrombotic factors that occurs during the course of an IMMD. Keeping IMMD as a possible differential diagnosis would help us circumvent that risk and manage it if it occurs. Our in-depth examination of the existing literature substantiates that our case stands as an unprecedented documentation within our country.

Ethics approval and consent to participate

Not applicable.

Consent of patient

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

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Author contribution

O.A.: Conceptualization, resources, methodology, data curation, investigation, who wrote, original drafted, edited, visualized, validated, literature reviewed the manuscript, and the corresponding author who submitted the paper for publication.
O.H.: Resources, methodology, data curation, investigation, who edited, visualized, validated, and literature reviewed the manuscript.
L.H.: Pathological analysis of resected specimens, assignment of the final histopathological diagnosis, validation, and review of the manuscript.
A.M., M.G.: Vascular Surgery specialists who performed and supervised the operation, in addition to validation, supervision, project administration, resources, and review of the manuscript. All authors read and approved the final manuscript.

Conflicts of interest disclosure

There are no conflicts of interest.

Research registration unique identifying number (UIN)

Not applicable.

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Availability of data and materials

The datasets generated during and/or analyzed during the current study are not publicly available because the data were obtained from the hospital computer-based in-house system. Data are available from the corresponding author upon reasonable request.

Provenance and peer review

Not commissioned, externally peer-reviewed.

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