ISSN: 0250-6882

Volume 5

EMA
جمعية الإمارات الطبية
EMIRATES MEDICAL ASSOCIATION

NEW EMIRATES MEDICAL JOURNAL

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Emirates Med J 2024; 5: e02506882295744

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REVIEW ARTICLE

Congenital Abdominal Aortic Aneurysm: Presentation, Etiology, Diagnosis and Management

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Abstract:

Aortic aneurysms are common in adults due to atherosclerosis but are rare in children and young adults, often overlooked due to infrequent reporting. Acquired aneurysms are usually linked to factors like umbilical artery ligation, connective tissue diseases, or vasculitides. In contrast, the causes of congenital abdominal aortic aneurysms (AAA) remain unknown due to their extreme rarity. Only a few cases have been reported. Prompt diagnosis is essential when symptoms such as abdominal distention, vomiting, or abdominal pulsatility occur. Diagnosis is typically confirmed through ultrasonography and multi-slice spiral computed tomography angiographies (MCSTA). After detection, a comprehensive investigation is necessary to rule out acquired AAA causes. Managing congenital AAA requires a highly personalized approach, with early surgical repair using grafts as a recommended option. After an extensive analysis of numerous academic sources, we have comprehensively understood the epidemiology, clinical features, and diagnostic and treatment techniques for congenital abdominal aortic aneurysms.

Keywords: Aortic aneurysms, Abdominal, Congenital, Children, Young adults, Atherosclerosis.

Article History	Received: November 29, 2023	Revised: February 07, 2024	Accepted: February 09, 2024

1. INTRODUCTION

Abdominal Aortic Aneurysm (AAA) is recognized as an isolated pathological enlargement of the abdominal aorta. Simplifying this, we classify circumstances under AAA when there's an exceedance in the diameter of the aorta beyond 30 mm or any area along the abdominal aorta that undergoes local enlargement beyond fifty percent compared to another area along the abdominal aorta [1]. It is crucial to distinguish between an aortic aneurysm and aortic dissection. Aortic dissection involves a tear in the intimal layer of the aorta, allowing blood to pass through and creating a false lumen that separates the tunica intima from the tunica media or the tunica adventitia. This process leads to the propagation of blood in the false lumen if it ends in a blind sac with subsequent enlargement. On the other hand, in a true aortic aneurysm, all three layers of the arterial wall (intima, media, and adventitia) contribute to aneurysm formation without any disruption to these layers [2, 3]. The occurrence of abdominal aortic aneurysms in newborns is exceedingly rare. A mere thirty-three cases involving children have been recorded, with the first instance documented in 1967 by Howarth *et al*. - establishing its exceptional infrequency [4 - 6]. A minor fraction of these aneurysms originate from inherent flaws in the arterial wall. In most instances, their origin continues to be unknown [7].

This review article addresses congenital abdominal aortic aneurysms (cAAA), focusing on their causes, prevalence, and optimal management. The aim is to enhance awareness and understanding among healthcare practitioners in how to deal with cAAA.

2. ETIOLOGY

While aortic aneurysms are quite uncommon in younger populations, instances of them do occur. Amongst these occurrences within pediatric demographics specifically noted for their rarity, the congenital type or ones that present at birth consistently rank as second highest in overall frequency [8]; they are classified as isolated truncal vascular defects per the Hamburg Classification of Congenital Vascular Malformations [9], but the cause of these defects is still unknown. Most of these instances are probably the result of unknown genetic alterations. Some exhibit peculiar physical findings and hindered developmental progression, while others may seem normal in children. Localized abdominal aortic narrowings may lead to poststenotic turbulent blood flow and eventual

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aneurysmal dilatation in this arterial abnormality, which is thought to have been most likely produced by a developmental arrest along the arterial system during the latter stages of embryogenesis. The visceral and extremity arteries may occasionally be affected by numerous aneurysms connected to these AAAs. An underlying undiscovered genetic abnormality often contributes to a severe error in developing and synthesizing collagen or elastin [8, 10]. The Transforming Growth Factor-Beta (TGF-) pathway regulates various connective tissue functions and processes linked to cell proliferation, differentiation, and growth. Recently, genetic abnormalities in individuals with aortic aneurysms have been linked to agents or components of TGF-signaling pathways, such as fibrillin-1. Loeys-Dietz syndrome and Marfan's syndrome are both rare hereditary connective tissue diseases that share patterns of cardiovascular abnormalities (such as thoracic aortic aneurysms). Mutations in the genes that code for TGF-2 or TGF-receptor (TGFBR) I or II cause these diseases [11 - 13]. Another genetic component linked to an aneurysm is the congenital defect of protease inhibitors. This defect leads to the deterioration of elastic fibers, a process not fully understood yet [14, 15]. These molecular and genetic processes are crucial for understanding congenital AAA etiology. Unfortunately, no previous instances of congenital AAA have documented these genetic abnormalities. As a result, the connection between these genetic defects and congenital AAA is uncertain. Additional research will be needed to determine their true link.

3. METHODOLOGY

We conducted a comprehensive literature search utilizing PubMed and Google Scholar electronic databases for pertinent English-language articles from January 1967 to July 2023. The search criteria incorporated specific keywords, such as: (“Pediatric congenital Aortic Abdominal Aneurysm”) AND (“Pathophysiology” OR “clinical manifestation,” OR “diagnosis” OR “screening” OR “differential diagnosis” OR “management” OR “etiology” OR “treatment” OR “graft” OR “prognosis” OR “follow-up”). After removing duplicates, the obtained results were organized using citation management tools (Zotero and Microsoft Excel). Methodological articles were independently and systematically screened during title, abstract, and full-text reviews. All categories of scientific articles were considered to compile insights on congenital abdominal aortic aneurysms (cAAA).

4. EPIDEMIOLOGY

There is no epidemiological data on cAAA; only 33 cases have been reported as of July 2023 [4, 6, 16]. Males display almost double the likelihood of developing congenital AAA as compared to females. Neonates and infants made up half of these patients. Furthermore, only 5 out of 33 individuals had been diagnosed with this condition when they turned three years old [6, 16]. Concerning positioning, in about 66.67% of cases, AAAs were found below the kidneys (infrarenal). Remarkably, though, around one-third remained asymptomatic. Wang and Tao *et al*. [17] in their study documented the details of all these cases.

5. PATHOPHYSIOLOGY

When it comes to congenital abdominal aortic aneurysms, our comprehension of the disease's pathophysiology is still incomplete. When looking into cases involving cAAA, related histologic irregularities have been recognized within the aorta's wall structure, the key abnormality identified being fibromuscular dysplasia [18 - 21]. Medial dysplasia is Most commonly observed in cAAA, defined as one type of fibrodysplasia characterized by collagen fiber accumulation in the media and the internal and external elastic lamina decrease [22]. A defect in protease inhibitors [14, 15] may cause the composition of elastin to change, leading to the fragmentation of elastic fibers in aneurysmal wall media and changes in their arrangement [23]. It was suggested that such aneurysms form during the fetal stage due to a marked secondary surge in fibroblasts and collagen strands within the adventitia [23].

6. PRESENTATION

Most congenital AAA instances, precisely 75.76%, are identified within the time frame starting from the second trimester up to one year of age. The incidence rate for AAA shows male predominance, with roughly twice as many males affected as females - maintaining approximately a 19:11 ratio in incidence rates among genders. The clinical manifestations of congenital AAA differ from no symptoms to rupture of the aneurysm and death. A more severe presentation may happen as AAA grows. Most manifestations usually seen involve a pulsating abdominal mass, abdominal distention, feeding difficulties and vomiting. For toddlers and adolescents, irritability and abdominal tenderness were manifestations [17]. Renal aneurysms and renal artery thrombosis might result in renovascular hypertension or renal failure, which should be taken seriously [17]. Only one recorded instance had an accompanying mass in the lower back with a subcutaneous vascular malformation [24]. The latest reported case was an incidental finding in a patient who suffered from tuberous sclerosis, highlighting the association between cAAA and systemic conditions such as inflammatory conditions, vasculitis or connective tissue disorders [6]. Infrarenal aneurysms are the most frequent kind, with 66.67%, followed by juxtarenal at 18.18%, noting that 3 out of the 33 reported cases did not mention the location. There were reports of associated aneurysms that included renal artery aneurysms, common iliac artery aneurysms, Descending thoracic aortic aneurysms, hypogastric artery aneurysms and intracranial aneurysms [4].

7. DIAGNOSIS

Congenital AAA is typically diagnosed using particular imaging modalities. AAA should be considered if a clinical manifestation such as abdominal pulsatile mass, abdominal vascular murmurs, or tremors are found. AAA can be identified by employing imaging methodologies such as ultrasonography, multi-slice spiral computed tomography angiography (MSCTA), and Magnetic resonance angiography (MRA). Ultrasound scanning tops the frequency list for diagnosing AAA, subsequently trailed by MSCTA and MRA. Ultrasonography has helped diagnose abdominal aneurysms prenatally as it can detect pulsatile masses using color Doppler imaging, found incidentally during prenatal visits. The high-

speed scanning system, multi-slice spiral computed tomography angiography (MSCTA), has a strong post-processing workstation and can perform thin-layer and volume scanning. Using multi-planar reconstruction, MSCTA may accurately and directly visualize the lesion and any nearby impacted organs or lumens [25]. Because it can provide information on the structural makeup and the possibility of endovascular treatment, MSCTA scanning has a high sensitivity for aneurysms and is useful for preoperative planning. MRA is proven to be an effective technique for the preoperative assessment of patients undergoing elective repair of abdominal aortic aneurysmal disease. Important preoperative information includes the type and location of the aneurysm, its diameter and cranial-caudal extension, the aneurysmal wall structures and periaortic space, the connection to the mesenteric and renal arteries, and the presence of an iliac aneurysm or stenosis [26]. The application of contrast-enhanced MRA methods has evolved the purpose of MR in imaging the abdominal aorta. The T1 shortening attributes of gadolinium chelates can assist contrast-enhanced imaging techniques. These techniques are particularly well-suited for aortic imaging with MRA since they are typically independent of blood flow velocity. The coronal 3D T1-weighted gradient echo imaging sequence is employed for contrast-enhanced approaches. The image obtained by these acquisition settings displays the abdominal aorta, iliac arteries, and common femoral arteries. The images provide for preoperative evaluation of iliac and common femoral artery diseases [27, 28]. Histopathological analyses can be utilized to reveal cases of fibromuscular dysplasia, in which the normal aortic architecture will be severely distorted with medial aortic fibrosis and loss of the majority of the medial smooth muscle and elastic fibers. The impact of this altered structure on function should be further investigated in light of these findings, as doing so could potentially inform clinical therapy [19, 29, 30]. For instances involving thoracoabdominal aortic aneurysms and coronary artery aneurysms, one might opt for CT angiography. An injection of contrast material is injected into the arteries, and a CT scan is used in computed tomography angiography (CTA) to help identify and assess blood vessel disease or related disorders, including aneurysms or blockages. Computed tomography angiography (CTA) can help isolate and determine the diameter of an AAA [20].

8. DIFFERENTIAL DIAGNOSIS

Knowing the differential diagnosis is crucial for directing a physician's investigation and treatment because many infant illnesses can present with abdominal pulsatile mass and murmurs on auscultation. It's important to highlight the higher occurrence of acquired abdominal aortic aneurysms (AAAs) compared to congenital ones. Thus, correctly identifying a congenital AAA necessitates first excluding the likelihood of an acquired AAA [31]. Mycotic aneurysms often result from traumatic insertion of umbilical artery catheters, infection, or thrombosis in umbilical arterial lines [32]. Other causes of acquired AAA include vasculitis attributed to Kawasaki's syndrome, Takayasu's disease, polyarteritis nodosa, neurofibromatosis, or Bourneville's tuberous sclerosis. Aneurysms caused by connective tissue disorders such as Marfan or Ehlers-Danlos syndrome occur from the degeneration of the aorta's medial layer. Still, they are more often discovered in the thoracic aorta and generally appear later in childhood, between the ages of 4 and 15 [33, 34]. People diagnosed with congenital AAA may also exhibit types of Ehlers-Danlos syndrome, with type IV being the most prevalent. This condition arises from abnormalities in type III procollagen within artery walls. Those with Ehlers-Danlos syndrome type IV rarely demonstrate significant skin laxity or joint hypermobility, complicating clinical recognition. Thus, confirming the diagnosis often requires skin biopsy and collagen III analysis [35]. Finally, children and adolescents with Loeys-Dietz syndrome, a connective tissue disease associated with mutations in the transforming growth factor β receptor, develop progressive arterial aneurysms and aortic dissection [36].

9. MANAGEMENT AND TREATMENT

Managing congenital AAA poses unique challenges, as the treatment plans often borrow from adult case studies and don't offer a uniform solution. Options can widely differ from adopting a wait-and-see strategy with an end goal of eventual correction to more critical measures such as surgical intervention.

10. CONSERVATIVE MANAGEMENT

Until recently, AAA's conservative management had largely gone unnoticed. As an adjunct to surgical treatment, conservative treatment aims to prevent AAA advancement and rupture, decrease the long-term need for surgery, and limit cardiovascular events. Conservative therapy is advised for patients for whom surgical repair carries a high risk of mortality and is unlikely to improve life expectancy [37]. There is no universal approach for cAAA management. Although steroids, cyclophosphamide, nonsteroidal anti-inflammatory (NSAIDS) medications, and statins have some therapeutic advantages, conservative therapy has a recorded mortality rate of 57.14%. To avoid hypertension, blood pressure should be managed, and antiplatelet therapy should be taken to reduce the risk of blood clot formation [6]. Other precautionary measures include ultrasonography monitoring and internal medicinal therapy such as statins, beta-blockers, matrix metalloproteinase inhibitors, angiotensin-converting enzyme inhibitors (ACEI), and antiplatelet medications. However, such drugs' effectiveness in treating pediatric AAA has not been studied [38].

11. SURGICAL MANAGEMENT

AAA may be repaired using open surgery or endovascular aneurysm repair (EVAR). However, there are special concerns when treating congenital AAA in neonates, infants, or young children. Despite a fast-growing proficiency with EVAR in adults [39], there is no known instance yet where these devices have been utilized for children suffering from congenital AAA. Also critical to note is that carrying out EVAR on infants or children isn't feasible due to the unavailability of suitable endograft and the potential impact on their growth and development over time, making it unsuitable specifically for this demographic group. Therefore, currently, among younger

patients undergoing treatment for triple-A disorder, using endovascular aneurysm repair (EVAR) remains impracticable. Aneurysmorrhaphy has been implicated in a minute number of cases. However, preserving the dysplastic aortic segment raises the likelihood of recurrence. In children, an artificial graft was a sufficient vascular replacement with no influence on growth, development, or quality of life [40, 41]. On the other hand, using an artificial graft in neonates raises the danger of mismatching between the fixed graft diameter and the growing native vessel diameter. As a result, graft replacement ought to be considered as the child grows up. Artificial grafts and allografts were the most usually employed for revascularization, with previously mentioned instances including Dacron graft or polytetrafluoroethylene (PTFE) graft, allografts, and a case of native vasculature. Although allografts have the benefit of having a high long-term patency and a low risk of postoperative graft infection, the long-term use of immunosuppressants and allograft sources creates complications [42]. A standard cryopreserved allograft is not recommended since it causes an immunological response linked to increased fibrosis, calcification, and degradation. Recently, a new generation of cryopreserved decellularized allografts with decreased immunogenicity for treating congenital heart diseases has been demonstrated [43]. These grafts appear to be long-lasting and well-suited for infantile growth. Aortoiliac reconstruction using decellularized branching pulmonary allograft yielded favorable results in one study [44]. However, estimating the value of this surgical repair for instances with congenital AAA remains impossible due to a lack of data on the graft's patency, need for reoperation, and the patient's long-term survival.

12. PROGNOSIS AND FOLLOW-UP

Managing congenital abdominal aortic aneurysm (cAAA) presents a challenge due to its rarity and potentially life-threatening complications. Early identification through clinical presentations and multimodality imaging is crucial. However, there still exists ambiguity regarding future outcomes [45]. A study undertaken in June 2019 documented a case where an early elective open repair with tubular ePTFE graft proved beneficial. The patient was discharged thirteen days after their surgery and steadily returned to normal health according to evaluations made three months post-operation [46]. Notably, this case lacked long-term follow-up data. In contrast, a second case, reported in October 2022, provided insight into a 14-year follow-up of a patient with cAAA. This individual initially underwent primary graft replacement at 3 and experienced no complications until growth cessation. At that point, they presented with lower limb pain at the age of 17. An extra anatomical bypass was performed, proving to be one of the most successful treatments for cAAA and representing the longest-followed case in the literature [17]. Overall, the natural prognosis of aortic aneurysms during infancy is quite poor, highlighting the significance of surgical intervention [23]. Nevertheless, cAAA management necessitates a multidisciplinary approach and surgical repair with long-term follow-up is advised, even though the optimal timing and approach remain unclear [4].

CONCLUSION

Congenital Abdominal Aortic Aneurysm (cAAA) is a rare disease with unknown origins and minimal understanding of its molecular pathways. Studies show that there are only eleven women for every 19 men involved. Symptoms like pulsatile abdominal mass require immediate imaging techniques like ultrasound and Multi-slice CT Angiography (MSCTA) to confirm the presence of an Abdominal Aortic Aneurysm (AAA). Management strategies for congenital AAA are not universally standardized, with both conservative treatments and surgical interventions documented. Conservative treatment may not yield the desired outcome, so surgical repair options may be considered if the patient's overall health is favorable and revascularization is feasible. However, comprehensive reports on long-term post-operative follow-up are scarce, leaving vital details about graft patency longevity and potential surgical intervention requirements ambiguous.

LIST OF ABBREVIATIONS

AAA	=	Abdominal Aortic Aneurysms
ACEI	=	Angiotensin-Converting Enzyme Inhibitors
CTA	=	Computed Tomography Angiography
ePTFE	=	Expanded Polytetrafluoroethylene
EVAR	=	Endovascular Aneurysm Repair
MCSTA	=	Multi-slice Spiral Computed Tomography Angiographie
MRA	=	Magnetic Resonance Angiography
TGF	=	Transforming Growth Factor

CONSENT FOR PUBLICATION

Not applicable.

FUNDING

None.

CONFLICT OF INTEREST

The authors declare no conflict of interest, financial or otherwise.

ACKNOWLEDGEMENTS

Declared none.

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