

Participation of atrioventricular valves chordae tendineae pathology in a patient's clinical presentation

Udział zaburzeń strun ścięgnistych zastawek przedsionkowo-komorowych w obrazie klinicznym pacjenta

Rafał Bieś¹, Zuzanna Szewczyk¹, Tomasz Lepich²

¹Students' Scientific Association, Department of Human Anatomy, Faculty of Medicine, Medical University of Silesia, Katowice, Poland

²Department of Human Anatomy, Faculty of Medicine, Medical University of Silesia, Katowice, Poland

Medical Studies/Studia Medyczne 2024; 40 (2): 201–207

DOI: <https://doi.org/10.5114/ms.2024.140981>

Key words: congenital heart defect, papillary muscles, chordae tendineae, atrioventricular valves.

Słowa kluczowe: wrodzone wady serca, struny ścięgliste, mięśnie brodawkowate, zastawki przedsionkowo-komorowe.

Abstract

Introduction: The anatomically correct chordae tendineae originate from the apical part of the papillary muscles of the ventricles and attach to the corresponding free margins of the atrioventricular valves. Together with the papillary muscles, they contribute to the effective contraction of the ventricles, creating the so-called “annular-ventricular continuity”.

Aim of the research: This publication aimed to evaluate and analyse the impact of chordae tendineae pathology of the atrioventricular valves on the clinical presentation of a patient.

Material and methods: A systematic review of the literature was performed in the PubMed, Web of Science, and Scopus databases and according to the PRISMA guidelines (Preferred Reporting Items for Systematic Reviews and Meta-Analyses).

Results and conclusions: This review allowed us to distinguish 6 groups of characteristic pathological lesions of chordae tendineae such as thickening, elongation, shortening, fibrosis, complete absence, or histopathological changes of the chordae tendineae. Most of the cases resulted from congenital underlying diseases, and treatment mainly consisted of surgical correction.

Streszczenie

Wprowadzenie: Anatomicznie prawidłowe struny ścięgliste pochodzą z wierzchołkowej części mięśni brodawkowatych komór i są przymocowane do odpowiednich wolnych brzegów zastawek przedsionkowo-komorowych. Wraz z mięśniami brodawkowatymi przyczyniają się do skutecznego skurczu komór, tworząc tak zwaną ciągłość pierścieniowo-komorową.

Cel pracy: Ocena i analiza wpływu patologii strun ścięgnistych zastawek przedsionkowo-komorowych na obraz kliniczny pacjenta.

Materiał i metody: Systematyczny przegląd piśmiennictwa został przeprowadzony w bazach danych PubMed, Web of Science i Scopus zgodnie z wytycznymi PRISMA (*Preferred Reporting Items for Systematic Reviews and Meta-Analysis*).

Wyniki i wnioski: Przegląd ten pozwolił nam wyróżnić sześć grup charakterystycznych zmian patologicznych strun ścięgnistych, takich jak pogrubienie, wydłużenie, skrócenie, zwłóknienie, całkowity brak lub zmiany histopatologiczne w strunach ścięgnistych. Większość przypadków wynikała z wrodzonych chorób podstawowych, a leczenie polegało głównie na korekcji chirurgicznej.

Introduction

Tendinous chords, anatomically, originate from the apical part of the papillary muscles of the ventricles and attach to the corresponding cusps of the atrioventricular valves. Each papillary muscle has a large shaft from which 6 “heads” extend. From each of them, 12 primary fibrous tendons depart, and then they divide into 2 secondary fibres, which again divided into 2 or 3 tertiary fibres. Thus, every single papillary muscle is associated with an average of 62 tendinous

chords, but it should be remembered that the exact number of fibre attachments may be an individual variable [1]. The primary chordae tendineae are attached to the free edge of the valve leaflets, the secondary ones to the ventricular surface of the leaflets, and the tertiary cords to the posterior part of the valve annulus. The anatomy, branching patterns, and thickness of individual chords are extremely variable. Tendinous chords together with the papillary muscles contribute to the effective contraction of the ventricles, creating

the so-called “annulo-ventricular continuity”. During the relaxation of the papillary muscles of the left ventricle, the tendinous chords reduce the lumen of the ventricle, and during contraction, they regulate their tension [2]. The mechanical properties of the tendinous chords result from their histological structure, dominated by collagen and elastic fibres, which transmit the contractions of the papillary muscles to the atrioventricular valves [3]. It is suggested that the wavy collagen straightens when the tendinous chords are stretched by the contraction of the papillary muscles, whereas after the muscle is relaxed, the collagen returns to its wavy configuration [4]. Diagnosis of symptoms resulting from tendinous chord dysfunction remains a major challenge due to non-specific symptoms. A better understanding of tendinous chord pathology will be particularly beneficial in improving the treatment of cardio and cardiosurgery patients. Clinicians need to be aware of normal and pathological tendinous chords variants. Tendinous chords anomalies show symptoms with a delay of days, weeks, or even months, unlike papillary muscle damage, which manifests symptoms right away.

Material and methods

This review article was carried out following the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines [5].

Inclusion criteria

In this study, we considered case reports regardless of patients' age or gender. Publications had to be full-text articles available in the English language. Publications had to provide a detailed description of tendinous chord abnormalities of atrioventricular valves and their impact on the patient's clinical presentation. During the review, we focused primarily on identifying the underlying causes of these anomalies. Regardless of treatment regimens, interventions, assessments of cytophysiological outcomes, and publications with lack of described aetiology were excluded from the analysis. We also excluded reports on false chords. Additionally, publications focusing on extensively described cases of ruptured chords were excluded unless the rupture was attributed to previously existing congenital or acquired tendinous chords pathologies. This review did not encompass pathologies related to neoplastic changes originating from the subvalvular apparatus because this is a comprehensive topic beyond the scope of this article.

Search strategy

We conducted a literature search in the PubMed, Web of Science, and Scopus databases. The time frame was not specified for the literature review because, according to our knowledge, this is the first review

article focusing on chordae tendineae pathologies in atrioventricular valves. The following search terms were used: ‘pathology’, ‘abnormalities’, ‘dysfunctions’, ‘fragile’, ‘thin’, ‘fine’, ‘thick’, ‘a few’, ‘redundance’, and ‘fibrotic’ in combination with ‘chordae tendineae’. Article selection involved an initial screening based on abstracts to identify studies potentially relevant to the topic. Then, we utilised article references to identify any articles missed during the database searches.

Data extraction

The included publications were analysed to extract the following information: publication year, primary author, patient's age and gender, underlying disease leading to tendinous chord pathology, description of tendinous chord pathology, the diagnostic method employed for patient diagnosis, and a description of the tendinous chord features (Table 1).

Results

Study classification

Two independent researchers searched the databases according to the predefined search strategy and initially identified 592 potentially eligible studies. Additional review of the bibliographies of the identified publications did not yield any additional studies. After the preliminary screening of titles and abstracts, we excluded studies that were duplicates, and upon applying the inclusion criteria, 110 articles were excluded. Among the remaining 112 studies, 96 were excluded due to a lack of relevant information about the outcomes, or after reading, it was found that they contained information unrelated to the review topic. The flowchart of the analysis is presented in Figure 1.

Histopathological changes despite normal macroscopic appearance

Histopathological changes of tendinous chords are most commonly detected spontaneously when biological material is obtained for examination during surgery. The macroscopic appearance of tendinous chords is typically normal. Only advanced changes can lead to the development of pathologies that often spread to the leaflets of the atrioventricular valves. A literature review identified case reports of patients with myxoid changes of tendinous chord fibres [6], mucoid degeneration, and fibroid changes from previous endocarditis [7], as well as heterogeneous deposits of AA-type amyloid [8]. Deposition of pathophysiological substances in tendinous chords can remain asymptomatic for a long time or present with non-specific symptoms. These changes are not isolated to tendinous chords but result from diseases affecting the entire subvalvular apparatus, valves, or papillary muscles of the heart chambers. Myxoid changes in the fibres disrupted the physi-

Table 1. Summary of included publications

Year	Age	Gender	Underlying disease	Chordae tendineae pathology
2022 [1]	63	F	Refractory right heart failure	Shortened chordae tendineae of the tricuspid valve
2022 [2]	22	F	Mitral valve leaflet prolapse secondary to anomalous left coronary artery from the pulmonary artery	Shortened chordae tendineae of the mitral valve
2021 [3]	36	M	Parachute Mitral Valve	Elongated and thickened chordae tendineae of the mitral valve unifocally attached to a single papillary muscle
2020 [4]	9	F	Congenital multiple sclerosis	Shortened and thickened chordae tendineae of the mitral valve
2017 [5]	43	F	Hypertrophic interventricular septum coexisting with anomalous anterolateral papillary muscle	Shortened chordae tendineae of the mitral valve
2016 [6]	56	M	Hypotrophic cardiomyopathy	Absent chordae tendineae of both leaflets of the mitral valve
2016 [7]	64	M	Endomyocardial fibrosis	Fibrotic chordae tendineae of the tricuspid valve
2013 [8]	31	M	Left ventricular non-compaction	Histopathologically irregular fibres, myxoid changes of chordae tendinea of the mitral valve
2010 [9]	35	F	Shone's complex	Thickened chordae tendineae of the mitral valve
2010 [10]	33	M	Mitral arcade	Absent chordae tendineae of the mitral valve
2008 [11]	14	M	Hypoplasia of the posterior leaflet of the mitral valve	Absent chordae tendineae of the posterior leaflet of the mitral valve
2008 [12]	16	M	Congenital abnormalities of the subvalvular apparatus of the mitral valve	Shortened and reduced number of chordae tendineae of the posterior leaflet of the mitral valve
2008 [13]	60	M	Bacterial endocarditis	Histopathologically mucinous degeneration and change of fibrinoid present in chordae tendineae of the mitral valve
2008 [14]	38	M	Systemic amyloidosis	Histopathologically present amyloid deposits in chordae tendineae of the mitral valve
1982 [15]	26	M	Acid maltase deficiency	Thickened chordae tendineae of the mitral valve
1982 [16]	24	M	Bacterial endocarditis	Swollen chordae tendineae of the mitral valve

ological function of tendinous chords, leading to their rupture. This resulted in the posterior-medial leaflet of the mitral valve prolapsing, subsequently causing symptoms of congestive heart failure. Additionally, excessive trabeculation and deep recesses were observed in the left ventricle, which led to left ventricular dilation. The described patient exhibited left ventricular noncompaction. Fibrous changes in the tendinous chords were also found in a patient who had undergone infective endocarditis. Mucoid degeneration of the tendinous chords did not disrupt valve function and was spontaneously discovered. Macroscopically, the tendinous chords did not show active inflammatory changes, and mucoid degenera-

tion was associated with the previous infective endocarditis [7]. Although cardiac amyloidosis is often associated with the pathophysiology of mitral valve function due to amyloid deposition, severe mitral regurgitation in patients with systemic cardiac amyloidosis is rare [9]. Postoperative histopathological findings in the patient revealed heterogeneous amyloid deposits in the right ventricular myocardium, mitral valve, and ruptured chordae. Additionally, immunohistochemical staining revealed a positive component of amyloid A – AA amyloid, with deposits spreading towards the ruptured chords. In patients with cardiac amyloidosis, the accumulation of amyloid deposits in the mitral valve leaflets can lead to decreased elastic-

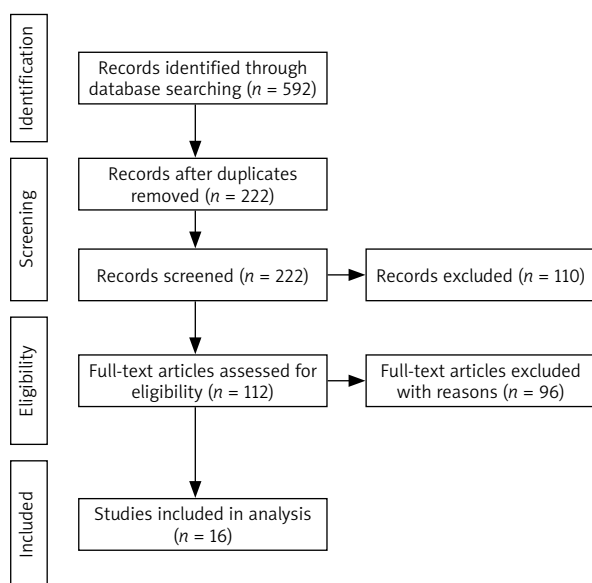


Figure 1. Study flowchart

ity, increased fragility, and weakness, causing chordal stress [10]. However, in this case, despite the amyloid deposits in the mitral valve, its motion was normal, and the valve itself was not thickened, suggesting that the amyloid deposits accumulated in the tendinous chords led to their rupture and secondary impairment of mitral valve function [8].

Pathological elongation of tendinous chords

Pathological elongation of the tendinous chords can result from compensatory changes in acquired heart defects, such as increased ventricular volume [11]. This condition can also be the result of congenital heart defects [12]. This review found an extremely rare and exceptional case of parachute mitral valve associated with abnormally elongated and thickened tendinous chords [13]. They were attached to a single papillary muscle and irregularly interconnected, forming a characteristic mesh. These anomalies resulted in restricted movement of the mitral valve leaflets. The unifocal attachment of the tendinous chords with a unique mesh-like shape caused severe narrowing of the mitral valve, subsequently leading to left atrial enlargement. The only functional communication between the left atrium and the left ventricle was through the mesh-like spaces between the tendinous chords. The significant stenosis indicated the need for mitral valve surgery [14]. Mitral valve valvuloplasty has better long-term prognosis compared to valve replacement, but the distorted chords were too severely affected to choose this procedure [15]. Isolated parachute mitral valve is very rare and can remain asymptomatic throughout life. The patient's only subjective symptom was dyspnoea on exertion persisting for 2 months since hospitalisation. In parachute mitral valve, the tendinous chords are often

poorly developed, resulting in them being short, thick, and adherent, which reduces the mobility of the valve leaflets and decreases the diameter of the mitral orifice [16]. This makes the described case exceptional. Additionally, parachute mitral valve can also cause thickening of the tendinous chords in the atrioventricular valve leaflets, which are attached to the single papillary muscle [13].

Pathologically thickened chordae tendineae

Pathological thickening of the tendinous chords can result from acquired heart defects such as parachute mitral valve [13], congenital multiple sclerosis [17], Shone's complex [18], and acid maltase deficiency [19], as well as acquired conditions like infective endocarditis [20]. In the case of congenital multiple sclerosis, one variant being mitral arcade, the tendinous chords are thick and significantly shortened or absent, leading to mitral valve stenosis or insufficiency [17]. Shone's complex is a rare congenital heart disease characterised by 4 major defects: aortic coarctation, subvalvular aortic stenosis, parachute mitral valve, and supralvalvular mitral ring. Complete Shone's complex includes all 4 abnormalities, while the partial form, which is more common and also represented by one of the described patients, involves 2–3 anomalies [21]. The described patient had a parachute mitral valve characterised by thickened tendinous chords in both mitral valve leaflets, connected to a single papillary muscle, resulting in left ventricular outflow tract obstruction [18]. Acid maltase deficiency, also known as Pompe disease or glycogen storage disease type II, is an autosomal recessive lysosomal storage disorder caused by a pathogenic variant in both copies of the GAA gene, which encodes acid α -glucosidase enzyme. This can result in partial or complete deficiency of acid α -glucosidase [22], leading to the accumulation of glycogen in various tissues, including the papillary muscles and tendinous chords of the mitral valve. This patient had thickened tendinous chords, and the papillary muscles had become hypertrophied [19]. Infective endocarditis is an infection of the endocardium. In the described patient, there was oedema of the tendinous chords and vegetations on the free edges of the anterior mitral leaflet composed of fibrinoid deposits, platelets, and white blood cells. This pathology resulted in fusion and rupture of the tendinous chords, leading to severe mitral valve insufficiency [20]. Mitral valve insufficiency, also described in one of the specified cases, is often the main manifestation of atrioventricular valve apparatus destruction in the course of this pathology [23].

Fibrotic tendinous chords

Fibrotic tendinous chords are composed of dense, compact collagen and elastin fibres, which alter their structure leading to anomalies within them [24]. Matrix metalloproteinase triggers a signalling cascade

initiating fibrosis of the cardiac muscle and subvalvular apparatus, including the tendinous chords, ultimately contributing to their rupture. However, it cannot be directly determined whether increased expression or reactivity of matrix metalloproteinase 1, a prototype matrix metalloproteinase that degrades fibrillar collagens type I, II, and III, is associated with chordae fibrosis [25]. A literature review highlighted a case description of a patient with fibrotic tendinous chords. The underlying disease that directly contributed to this type of anomaly was endomyocardial fibrosis, a progressive form of restrictive cardiomyopathy characterised by haemodynamic changes primarily caused by endocardial thickening and fibrosis [26]. Endomyocardial fibrosis is characterised by inflammation involving all layers of the heart, present in both early and late stages of the disease. The fibrosis primarily affects the distal parts of the ventricles and atrioventricular valves, leading to the described valve insufficiency. The patient, presented with right-sided heart failure and recurrent pulmonary embolism. They had a markedly dilated and thrombus-filled right atrium, and the right ventricle exhibited significantly impaired contractile function, necessitating hospitalisation. The patient ultimately succumbed to treatment-refractory right-sided heart failure [24].

Pathologically shortened tendinous chords

Pathologically shortened tendinous chords can result from refractory right heart failure [27], Bland-White-Garland syndrome [28], hypertrophic interventricular septum [29], and mitral arcade [17]. Refractory right heart failure refers to the persistence of symptoms related to fluid retention despite optimal pharmacological treatment [30]. In the described case, pericardial effusion led to impaired venous return, masking severe tricuspid valve insufficiency, which became apparent after drainage. The shortened tendinous chords were an organic abnormality associated with congenital structural tricuspid valve insufficiency, which remained asymptomatic due to the presence of pericardial effusion [27]. Bland-White-Garland syndrome, indicating an anomalous left coronary artery originating from the pulmonary artery, is a rare and potentially life-threatening congenital anomaly of the coronary artery [28]. It is divided into 2 types: infantile and, much less commonly, adult, represented by one of the described patients [31]. In most cases, the mechanism of collateral circulation is insufficient, ultimately leading to ischaemic dysfunction of the papillary muscle and vulnerable, shortened tendinous chords, which can lead to their rupture [28]. Hypertrophic interventricular septum is one type of hypertrophic cardiomyopathy [32], characterised by hypertrophy and disarray of cardiomyocytes, as well as interstitial and inflammatory fibrosis [29]. In the specific case, an acute angle of the hy-

pertrophied interventricular septum protruding into the left ventricular outflow tract, an anomalous anterolateral papillary muscle, and shortened tendinous chords contributed to the left ventricular outflow tract gradient, as shown by the immediate entry of the catheter into the aorta during left ventricular catheterisation [32].

Absent tendinous chords

A literature review identified case reports of patients with absent tendinous chords, which were associated with the presence of mitral arcade [17, 33], hypertrophic cardiomyopathy [34], hypoplasia of the posterior mitral valve leaflet [35], and abnormalities of the mitral valve subvalvular apparatus [36]. Mitral arcade, which is one variant of congenital diffuse sclerosis, affects the structure of the papillary muscles and tendinous chords. It is characterised by elongated papillary muscles connected to the anterior leaflet of the mitral valve by a fibrous tissue bridge that resembles an arcade. Mitral arcade affects the structure of the patient's mitral valve, leading to described valve insufficiency or stenosis [37]. Hypertrophic cardiomyopathy is a complex and common genetic disease inherited in an autosomal dominant manner [38]. It is characterised by hypertrophy of cardiomyocytes [34], and its pathophysiology is determined by a complex interaction involving the mitral valve, papillary muscles, tendinous chords, and interventricular septum. Hypertrophy of the papillary muscle, shortening or absence of tendinous chords, and elongation of the mitral valve leaflets result in reduced mobility of the mitral valve leaflets [38], which can contribute to mitral valve stenosis or insufficiency [34, 38]. Hypoplasia of the posterior mitral valve leaflet is a rare congenital heart defect, typically manifesting in infancy and childhood as severe mitral valve insufficiency, either isolated or associated with other cardiac abnormalities [39]. In this pathology, the posterior leaflet of the mitral valve is nearly completely absent, represented only by fibrous tissue markers that closely adhere to the posterior annulus, with a complete absence of chordae inserting into the hypoplastic leaflet, leading to mitral valve insufficiency [35]. Another disorder associated with the absence of tendinous chords is congenital abnormality of the subvalvular apparatus, characterised by the abnormal attachment of the papillary muscle directly to the mitral valve leaflets, which can result in entrapment of the anterior and posterior mitral valve leaflets and ultimately lead to mitral valve insufficiency [36].

Discussion

Tendinous chords are an important part of the subvalvular apparatus of atrioventricular valves. Depending on their arrangement and morphology, they can affect the clinical presentation of patients, and their

detailed structure is an essential part of cardiac surgical procedures [40]. To the best of our knowledge, this is the first review article discussing the involvement of tendinous chords in congenital and acquired heart valve abnormalities. The appearance of tendinous chords can be macroscopically distorted or involve structures visualised during histopathological examinations. Cellular changes can occur secondarily to endomyocardial fibrosis [24], infective endocarditis [1], or systemic amyloidosis [19]. Macroscopic changes include thickening, elongation, shortening, fibrosis, or complete absence of the chordae. The aetiopathogenesis of these conditions is presented in Table 1. This article is not without limitations. The available research materials are based on case reports, which may introduce potential publication errors in the results section. Case reports focus on the course and clinical presentation of patients, addressing the entire apparatus of atrioventricular valves rather than specifically focusing on tendinous chords. Furthermore, there are no randomised controlled trials describing the effectiveness of surgical treatment for patients with isolated or concurrent heart valve defects involving tendinous chords.

Conclusions

Patients with tendinous chords pathologies may be asymptomatic for years if normal valve function is maintained, but some patients may develop secondary severe valvular disease and cause congestive heart failure. Diagnosis was usually based on transthoracic/transoesophageal echocardiography; then patients underwent surgery to replace the valve with a suitable prosthesis and to correct the associated heart defects. In each described case, the operation and convalescence were successful.

Funding

No external funding.

Ethical approval

Not applicable.

Conflict of interest

The authors declare no conflict of interest.

References

1. Roberts WC, Cohen LS. Left ventricular papillary muscles. Description of the normal and a survey of conditions causing them to be abnormal. *Circulation*. 1972 Jul; 46(1): 138-154.
2. Athanasiou T, Chow A, Rao C, Aziz O, Siannis F, Ali A, Darzi A, Wells F. Preservation of the mitral valve apparatus: evidence synthesis and critical reappraisal of surgical techniques. *Eur J Cardiothorac Surg*. 2008 Mar; 33(3): 391-401.
3. de Vlaming A, Sauls K, Hajdu Z, Visconti RP, Mehesz AN, Levine RA, Slaugenhaupt SA, Hagège A, Chester AH, Markwald RR, Norris Russell A. Atrioventricular valve development: new perspectives on an old theme. *Differentiation*. 2012 Jul; 84(1): 103-116.
4. Goodwin RL, Kheradvar A, Norris RA, Price RL, Potts JD. Collagen fibrillogenesis in the mitral valve: it's a matter of compliance. *J Cardiovasc Dev Dis*. 2021 Aug; 8(8): 98.
5. Page MJ, Moher D, Bossuyt PM, Boutron I, Hoffmann TC, Mulrow CD, Shamseer L, Tetzlaff JM, Akl EA, Brennan SE, Chou R, Glanville J, Grimshaw JM, Hróbjartsson A, Lalu MM, Li T, Loder EW, Mayo-Wilson E, McDonald S, McGuinness LA, Stewart LA, Thomas J, Tricco AC, Welch VA, Whiting P, McKenzie JE. PRISMA 2020 explanation and elaboration: updated guidance and exemplars for reporting systematic reviews. *BMJ*. 2021 Mar; 372: n160.
6. Igarashi T, Takase S, Satokawa H, Wakamatsu H, Kurosawa H, Yokoyama H. Left ventricular noncompaction complicated by mitral valve prolapse: report of a case. *Surg Today*. 2013 Jul; 43(7): 818-820.
7. Takaya T, Takeuchi Y, Okamoto M, Hata K, Kijima Y, Nakajima K, Kita T, Ito M, Nakajima H, Takaoka R, Nomura T, Iwahashi K, Kawashima S, Seo T. Mitral regurgitation resulting from the consecutive multiple perforations by infective endocarditis mimicking the isolated anterior mitral cleft. *J Cardiol*. 2008 Oct; 52(2): 159-162.
8. Nishi H, Mitsuno M, Ryomoto M, Miyamoto Y. Severe mitral regurgitation due to cardiac amyloidosis—a rare reason for ruptured chordae. *Interact Cardiovasc Thorac Surg*. 2008 Dec; 7(6): 1199-1200.
9. Masugata H, Mizushige K, Senda S, Kinoshita A, Nozaki S, Matsuo H, Kohno M. Physical properties of the mitral valve tissue assessed by tissue sound speed in cardiac amyloidosis: relationship to the severity of mitral regurgitation. *Ultrasound Med Biol*. 2000 Sep; 26(7): 1191-1198.
10. Ammar KA, Khandheria BK, Bajwa T, Port SC, Allaqa-band S, Jain R, Neitzel G, Tajik AJ. Cardiac amyloidosis presenting as severe mitral regurgitation. *JACC Cardiovasc Imaging*. 2016 Aug; 9(8): 1003-1006.
11. Laurence DW, Johnson EL, Hsu MC, Baumwart R, Mir A, Burkhart HM, Holzapfel GA, Wu Y, Lee CH. A pilot in silico modeling-based study of the pathological effects on the biomechanical function of tricuspid valves. *Int J Numer Method Biomed Eng*. 2020 Jul; 36(7): e3346.
12. Ikeno Y, Yokawa K, Henmi S, Nakai H, Yamanaka K, Inoue T, Tanaka H, Tanaka H, Hirata K, Okita Y. A successful report of mitral valve repair for parachute-like mitral valve in adult. *Gen Thorac Cardiovasc Surg*. 2020 Mar; 68(3): 287-289.
13. Duan QJ, Duan CT, Dong AQ, Cheng HF. Parachute mitral valve associated with reticular chordae tendineae in an adult: case report. *J Cardiothorac Surg*. 2021 Apr; 16(1): 72.
14. Yuan SM. Parachute mitral valve: Morphology and surgical management. *Turk Gogus Kalp Damar Cerrahisi Derg*. 2020 Jan; 28(1): 219-226.
15. Lin XF, Xu Z, Zheng ZH, Wang T, Dai XF. Early clinical outcomes of thoracoscopic mitral valvuloplasty: the first 90 cases. *Heart Surg Forum*. 2022 Sep; 25(5): E692-E697.
16. Mohan JC, Shukla M, Mohan V, Sethi A. Parachute mitral valve and Pacman deformity of the ventricular septum in a middle-aged male. *Indian Heart J*. 2016 Sep; 68 Suppl 2 (Suppl 2): S126-S130.
17. Babu NS, Vimala LR, Varghese L, George OK. An arcade in the heart: multimodality imaging. *Ann Pediatr Cardiol*. 2020 Jan-Mar; 13(1): 95-97.

18. Purvis JA, Kennedy V, McNeill AJ. Don't stop at two. *Int J Cardiovasc Imaging*. 2010 Apr; 26(4): 369-371.
19. Akamatsu A, Nomoto R, Nagao H, Murakami H, Nonaka I, Tara M, Kato M. Adult form acid maltase deficiency: a case report. *Jpn J Med*. 1982 Jul; 21(3): 203-209.
20. van Leeuwen K, Fast JH, Deppenbroek JH, Skotnicki SH. Abnormal echoes in the left ventricular outflow tract caused by ruptured chordae tendineae of the mitral valve. *Chest*. 1982 Jan; 81(1): 103-105.
21. Sinfield S, Ranasinghe S, Wang S, Mendoza F, Khoynzhad A. Shone's complex and aortic dissection: case report and review of a rare, underdiagnosed congenital heart disease. *J Cardiothorac Surg*. 2022 Feb; 17(1): 21.
22. Taverna S, Cammarata G, Colomba P, Sciarrino S, Zizzo C, Francofonte D, Zora M, Scalia S, Brando C, Lo Curto A, Marsana EM, Olivieri R, Vitale S, Duro G. Pompe disease: pathogenesis, molecular genetics and diagnosis. *Aging (Albany NY)*. 2020 Aug; 12(15): 15856-15874.
23. Rajani R, Klein JL. Infective endocarditis: a contemporary update. *Clin Med (Lond)*. 2020 Jan; 20(1): 31-35.
24. Shiraishi Y, Kohno T, Fujii-Nishimura Y, Shimoda M, Ikeda Y, Nakajima K, Nishiyama T, Nishiyama N, Murata M, Maekawa Y, Sano M, Fukuda K. Endomyocardial fibrosis: missing tricuspid valve and Fontan-like circulation. *Heart Vessels*. 2016 Sep; 31(9): 1579-1582.
25. Lin TH, Yang SF, Chiu CC. Matrix metalloproteinase-1 mitral expression and -1607 1G/2G gene promoter polymorphism in mitral chordae tendineae rupture. *Transl Res*. 2013; 161(5): 406-413.
26. Seth S, Thatai D, Sharma S, Chopra P, Talwar KK. Clinico-pathological evaluation of restrictive cardiomyopathy (endomyocardial fibrosis and idiopathic restrictive cardiomyopathy) in India. *Eur J Heart Fail*. 2004 Oct; 6(6): 723-729.
27. Higuchi M, Mitomi K, Chiba Y. Shortened chordae tendineae of the tricuspid valve with right ventricular dysfunction caused by acute myocarditis lead to cardiogenic shock during pericardial drainage: a case report. *Oxf Med Case Reports*. 2022 Sep; 2022(9): omac097.
28. Wang X, Xia X, Huang W, Li X, Liu Y. Anomalous origin of the left coronary artery from the pulmonary artery as a rare cause of mitral valve prolapse: a case report. *BMC Cardiovasc Disord*. 2022 Jul; 22(1): 304.
29. Wang Y, Ye L, Yin L, Zeng J. Hypertrophic angulation deformity of the basal interventricular septum combined with abnormality of the papillary muscle and chordae tendineae. *Cardiovasc J Afr*. 2017 Jan; 28(1): e1-e3.
30. Wilcox CS, Testani JM, Pitt B. Pathophysiology of diuretic resistance and its implications for the management of chronic heart failure. *Hypertension*. 2020 Oct; 76(4): 1045-1054.
31. Peña E, Nguyen ET, Merchant N, Dennie C. ALCAPA syndrome: not just a pediatric disease. *Radiographics*. 2009 Mar-Apr; 29(2): 553-565.
32. Maron BJ, Sherrid MV, Haas TS, Lindberg J, Kitner C, Lesser JR. Novel hypertrophic cardiomyopathy phenotype: segmental hypertrophy isolated to the posterobasal left ventricular free wall. *Am J Cardiol*. 2010 Sep; 106(5): 750-752.
33. Federici D, Palmerini E, Lisi M, Centola L, Chiavarelli M, Mondillo S. Congenital mitral disease: anomalous mitral arcade in a young man. *Ann Thorac Surg*. 2010 Feb; 89(2): 629-631.
34. Mohan JC, Shukla M, Mohan V, Sethi A. Spectrum of congenital mitral valve abnormalities associated with solitary undifferentiated papillary muscle in adults. *Indian Heart J*. 2016 Sep-Oct; 68(5): 639-645.
35. Caciolli S, Gelsomino S, Fradella G, Bevilacqua S, Favilli S, Gensini GF. Severe hypoplasia of the posterior mitral leaflet. *Ann Thorac Surg*. 2008 Dec; 86(6): 1978-1979.
36. Kudo M, Yozu R, Aeba R, Kokaji K, Kimura N, Iwanaga S. A case report of surgical correction for congenital mitral regurgitation with subvalvular apparatus abnormality. *Gen Thorac Cardiovasc Surg*. 2008 Jan; 56(1): 36-38.
37. Singh R, Wang M. A rare encounter of mitral arcade with anomalous papillary muscles. *Cureus*. 2022 Jan; 14(1): e21253.
38. Cavalcante JL, Barboza JS, Lever HM. Diversity of mitral valve abnormalities in obstructive hypertrophic cardiomyopathy. *Prog Cardiovasc Dis*. 2012 May-Jun; 54(6): 517-522.
39. Bacich D, Braggion G, Faggian G. Hypoplasia of the posterior mitral leaflet: a rare cause of mitral regurgitation in adulthood. *Echocardiography*. 2017 Jun; 34(6): 949-950.
40. Jang DH, Yoo JS. Chordae tendineae approximation technique for severe tricuspid regurgitation with severe leaflet tethering using a totally endoscopic beating-heart strategy: a case report. *J Chest Surg*. 2023 Jan; 56(1): 56-58.

Address for correspondence:

Zuzanna Szewczyk
Students' Scientific Association
Department of Human Anatomy
Faculty of Medicine
Medical University of Silesia
Katowice, Poland
E-mail: s86246@365.sum.edu.pl

Received: 14.01.2024

Accepted: 14.05.2024

Online publication: 30.06.2024