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Morphology, microstructure and biomechanical properties of tendinous cords of heart – a systematic review of cadaveric studies

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Abstract

Objectives: The rupture of tendinous cords (TC), affects the proper functioning of the atrioventricular (AV) valves and requires replacement by the prosthetic chordae. The morphology and ultrastructure of TC are vital to design prosthesis and to prevent the treatment failure.

Methods: The databases like PubMed, Google Scholar, Science direct and Scopus were searched till June 2024. The keywords used were TC, chordae, TC anatomy, TC morphology, TC morphometry, TC histology, TC ultrastructure, TC blood supply, TC development, TC embryology, TC biomechanical properties, strut chordae, false chordae, basal TC, and subvalvular apparatus. Out of 2545 articles collected, 43 were finally included.

Results: Majority of human studies were on the hearts from formalin-embalmed cadavers. There were more number of studies (28 studies) examining TC of left ventricle, than that of the right (8 studies). The number of chordae from anterior papillary muscle were greater in number than that from the posterior muscle in both the ventricles. In the left ventricle, anterior papillary muscle chordae were longer and broader. Maximum chordae inserted on the rough zone of mitral and tricuspid valves. Human chordae were structurally more rigid than the animal chordae. The arrangement of collagen bundles inside TC was orthogonal in human, but random and irregular in animal tissues.

Conclusion: This review outlines the details on the morphology, ultrastructure, and biomechanical properties of TC which would aid formulate appropriate reparative procedures to prevent postoperative complications and recurrence.

Keywords: chordae tendineae; heart ventricles; morphology; papillary muscle; review; tendinous cords

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Introduction

The tendinous cords (TC) or chordae tendineae are strong, filamentous collagenous cords connecting atrioventricular (AV) valve leaflets to papillary muscles (PM) of heart.[1,2] They transmit contraction of PM to AV valve, thereby preventing prolapse of leaflets into atria, during ventricular systole. Proper functioning of TC is vital for effective closure of AV valves.[3] In right ventricle (RV), the anterior and posterior leaflets of tricuspid valve (TV) are attached to corresponding PM via TC. Whereas, septal leaflet often directly attaches to ventricular wall via septal chordae.[4] In left ventricle (LV), both anterior and posterior mitral leaflets are attached to PM via TC. The AV valve receives TC with various branching patterns.[2]

Histologically, chordae are composed of collagen and elastic fibers. They possess high degree of elasticity and endurance to perform efficiently.[3] Comparison of the ultrastructure and mechanical properties of human and porcine TC, provides possible replacement of chordal prosthetics by porcine chordae.[5–7] In both, TC configuration is essentially the bundles of collagen fibers forming the core structure. There are, however, differences in the arrangement of collagen tissue at TC and PM juncture where bundles are more organized in human tissue than that in porcine.[6] The junction of chordae with valve may comprise diminished collagen content in its thin outline and the higher load of cardiac muscle contraction on the left side increases the risk of rupture of left ventricle TC. Rupture of TC leads to displacement

of AV valves, resulting in regurgitation of blood into atria, during ventricular systole.[2] Standard treatment for chordal rupture is substitution with expanded polytetrafluoroethylene (ePTFE) sutures. Here, ruptured chordae are replaced by synthetic ones. Bioprosthetic, which may replace mechanical valves, reduce thromboembolic occurrences. Recurrent mitral regurgitation in chordal replacement patients is reported in the literature. The possible causes of treatment failure are placement of less sutures, increased length and inappropriate thickness of suture materials. Thus, understanding of normal measurements, mechanics and microstructure of TC is essential for improved success rate of chordal repair and replacement procedures.[8]

Various authors have described the morphology of TC in human cadaveric and animal hearts. Cadaveric studies mainly focus on gross appearance of chordae which includes origin, insertion, and branching pattern.[2,9–12] Studies providing morphometric data of TC such as their length and thickness, in both ventricles are restricted as only few have examined these parameters.[3,10,13–16] Based on branching pattern and insertion of TC on to valvular leaflet, several classifications have been proposed, that are useful for academic purpose and their clinical applicability needs to be reviewed further.[2,11,12,15,17,18] Existing literature also describes the histology, biomechanical properties, and developmental aspects of TC.[3,6,13,19–22] Most available studies are on TC of LV than RV, as area where TC merge and fix to the valve tissue is thin and more likely to get disrupted on the left due to higher systolic load, thus maximum reparative procedures are done on mitral valve (MV) apparatus. The data on morphology and morphometry of TC of RV are comparatively less in the literature.[2,9,10,12,17,23,24]

Surgeons need comprehensive knowledge on TC morphology and morphometry, to repair AV valves and subvalvular apparatus. Awareness on variations in position and structure of TC is vital to visualize them intraoperatively and during echocardiography.[2] TC could pose difficulties with transcatheter MV prosthesis placement, expansion, and fixation.[24] Data on length and thickness of TC are crucial for surgeons to decide on measurements of chordal prosthetic sutures.[25] Existing studies on histology and biomechanical properties of TC improve the understanding of their structure and function.[3] This review further outlines the morphology, ultrastructure, and biomechanical properties of TC which would aid in designing prosthesis and preventing treatment failure.

Materials and Methods

Literature search was carried out till June 2024. Search engine used was Google and databases were PubMed, Google Scholar, Science direct and Scopus. Plan for search was adapted according to abovementioned databases. Search strategy was formulated as per PRISMA guidelines (**Figure 1**). Keywords used were tendinous cords, chordae tendineae, chordae, tendinous cords/chordae tendineae anatomy, tendinous cords/chordae tendineae morphology, tendinous cords/chordae tendineae morphometry, tendinous cords/chordae tendineae histology, tendinous cords/chordae tendineae ultrastructure, tendinous cords/chordae tendineae blood supply, tendinous cords/chordae tendineae development, tendinous cords/chordae tendineae embryology, biomechanical properties, strut chordae, false chordae, basal chordae, basal, and subvalvular apparatus. Initially 2545 articles were found. Articles without full text, unpublished data, case reports, pathological studies, imaging studies, and publications written in language other than English were not considered. We included studies involving only cadaveric and fresh autopsied hearts which comprised data on normal parameters of heart. Since case reports, pathological studies and imaging studies of heart dealt with abnor-

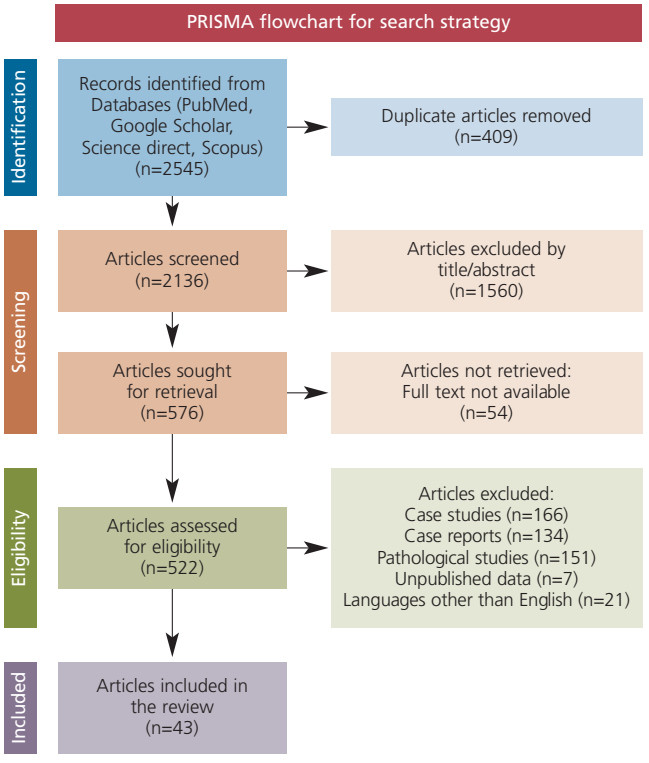

Figure 1. PRISMA flowchart for search strategy.

mal parameters we excluded them. Lastly, 43 studies were included. LV cords were examined in 28 studies, RV in eight studies, four studies examined TC in both ventricles and three did not specify sample source. Based on study material used, 25 studies were human and 14 were animal and four studies compared both. In 25 human studies, 16 were carried out in formalin-embalmed hearts, seven in autopsied specimens and two studies compared both. TC of RV were studied in eight and LV in 17 of human studies. None of the studies observed TC of both ventricles in same sample. Number of studies on each parameter of TC are summarized in **Table 1**. Sample size of the studies included in the review ranged from 8 to 116 in number. A higher sample size is desirable as it is more likely to reflect the population variability, generates ample data for statistical analysis and thus making it appropriate to apply the study outcomes to the general population, however, many factors like availability of specimens, feasibility may limit the sample size. Morphology of TC was systematically reviewed and their features in both ventricles were reexamined. Also, salient ultrastructure and biomechanical properties of TC in human and animals were outlined.

Table 1
Number of studies on various parameters of tendinous cords.

Parameters studied	Human and animal studies	Human studies
A	16	12
B	1	1
C	6	1
D	5	2
A+B	5	5
A+C	1	0
C+D	1	0
A+B+C	1	1
A+B+D	1	0
Development	2	0
Blood supply	2	2
Lymphatics	2	1
Total	43	25

A: morphology (includes origin, insertion, branching pattern); **B:** morphometry (includes length, thickness); **C:** histology (includes connective tissue, neurovasculature); **D:** biomechanical properties.

Results

Development

TC are derived from endothelial cells of endocardial cushions.[26] Chordal development is a programmed cellular and hemodynamic event which occurs between 6 to 13 days of development. PM develop as primitive elevations from ventricular wall. These elevations later bifurcate into thin, web-like folds which are attached to AV valve leaflets. These folds are the primordial chordae. Alternate linear ridges and depressions develop on primordial chordal folds. The depressions later perforate to form individual chordae from linear ridges. Some interchordal connecting tissues can persist.[21] Morphological variations of TC are a result of various aberrations occurring in delamination of ventricular musculature.[23] Muscular chordae are formed because of abnormal delamination of endocardium during development, which leads to persistence of myocardial fibers within the endocardium lined cords.[27] Muscular cords are prone to fibrosis and hence their presence become undesirable.[28]

Number of tendinous cords at origin

TC arise either from PM or directly from ventricular wall and insert onto AV valve.[12] In RV, there are anterior PM (APM), posterior PM (PPM) and septal PM (SPM). PM give origin to one or more cords which insert to TV.[1] Average number of chordae arising from APM is 3.88±0.45, PPM is 3.71±0.31 and SPM is 3.15±0.7.[29] Maximum chordae arise from APM.[11,29] In RV, TC originate from SPM or when SPM are absent, they directly originate from RV septal wall. When only one prominent SPM exists (Lancisi's muscle), its small head with single apex attaches to TC which head to septal and anterior TV leaflets.[30–32] The Lancisi's muscle, other nearby SPM and isolated TC that directly originate from septal wall are together termed as medial papillary complex.[31]

LV comprises APM and PPM (**Table 2**). Each PM gives rise to TC which insert to the MV.[1] Average number of chordae present in LV are 17.31±3.28.[13] Number of cords originating from one PM is 9.09± 4.29 (range: 2–18).[2,33] APM gives rise to more chordae than PPM.[12] Most chordae arise from lateral sides of PM belly (73.22%) and apex gives rise to 19.06%.[10] Chen et al.[13] discovered that more chordae originated from APM (11.23±2.24) than PPM (8.23±2.05). In MV replacement surgeries, preservation of PM and TC improves LV function post-operatively. Hence, clear knowledge about number of chordae at origin becomes important.[2]

Table 2
Number of tendinous cords originating from left ventricular papillary muscle.

Studies	Sample size	Number of cords originating from anterior papillary muscle	Number of cords originating from posterior papillary muscle
Victor et al.[23]	100	4–22	2–18
Kavitha et al.[10]	50	7–16 (10.42)	6–17 (9.72)
Mundra et al.[12]	98	12.45±3.16 6–1 (12)	9.67±3.85 3–22 (10)
Ozog et al.[17]	100	11.5±4 (1–14)	13.1±4.1 (2–13)

Number of tendinous cords at insertion

Each TC after emerging often divides into multiple thin secondary branches and attach to AV valve.[1,3] Each leaflet can receive chordae from one or more PM.[34] Chordae can also insert to adjacent PM or ventricular wall.[2,11] In RV, chordae either arise from PM belly or directly from ventricular septal wall and insert to TV. Average number of chordae inserting to TV are 19.25 and most insert on its rough zone.[11,18] Number of marginal chordae is more in posterior, than anterior and septal leaflets. Anterior leaflet has TC from APM (86.19±11.66%) and PPM (13.09±1.74%). Posterior leaflet receives TC from PPM (85.67±11.48%) and SPM (14.33±1.83%). Septal cusp receives cords from SPM (19.0±2.55%) and APM (80.99±10.85%).[29]

In MV, TC attach to anterior/aortic leaflet and posterior/mural leaflet (**Table 3**). Each leaflet can receive chordae from APM and PPM.[34] From a surgeon's perspective, chordae arising from APM insert to left halves of both MV leaflets and chordae arising from PPM insert to right halves.[35] Ozan et al.[33] noted that 9 to 60 chordae inserted to corresponding half of MV. Leaflet coverage ratio of APM chordae (51.58±3.35) is significantly greater than that of PPM chordae (48.42±3.35).[13] There is no gender difference in number of chordae at the valve.[15] In chordal repair procedures, ePTFE are used to construct artificial chordae.[25,36] A single suture material is successively passed from tip of PM to free margin of AV valve, to create multiple pairs of artificial cords.[25] Thus, a wide information about origin and insertion of TC to AV valve is mandatory.

Branching pattern and types of tendinous cords

Several branching patterns have been documented in literature so far (**Figure 2**). Most common branching pattern of chordae is fan-shaped (100%) and spiral/dichotomous (34.48%).[2,12] Fan-shaped chordae are frequently found at commissures. This fanning out of chordae at commissures is essential to open and close the leaflets during ventricular diastole and systole respectively.[15,23] Least common patterns are irregular/ web-shaped (15.51%) and unbranched chordae (19.82%).[2] There can be anastomosis between adjacent chordae (52%).[12]

TC can originate from different sites on a PM such as its apex, base, or lateral margins.[10] Chordae which arise from apex of PM are termed as apical pillar chordae. Basal

Table 3
Number of tendinous cords inserting to mitral valve leaflets.

Studies	Sample size	Number of cords inserting to anterior valve leaflet	Number of cords inserting to posterior valve leaflet
Victor et al.[23]	100	14–72	12–80
Mundra et al.[12]	98	28–91 (50)	33–84 (56)
Ozog et al.[17]	100	30.3±7.8 (14–55)	40.6±13 (6–40)
Lam et al.[15]	50	9	14

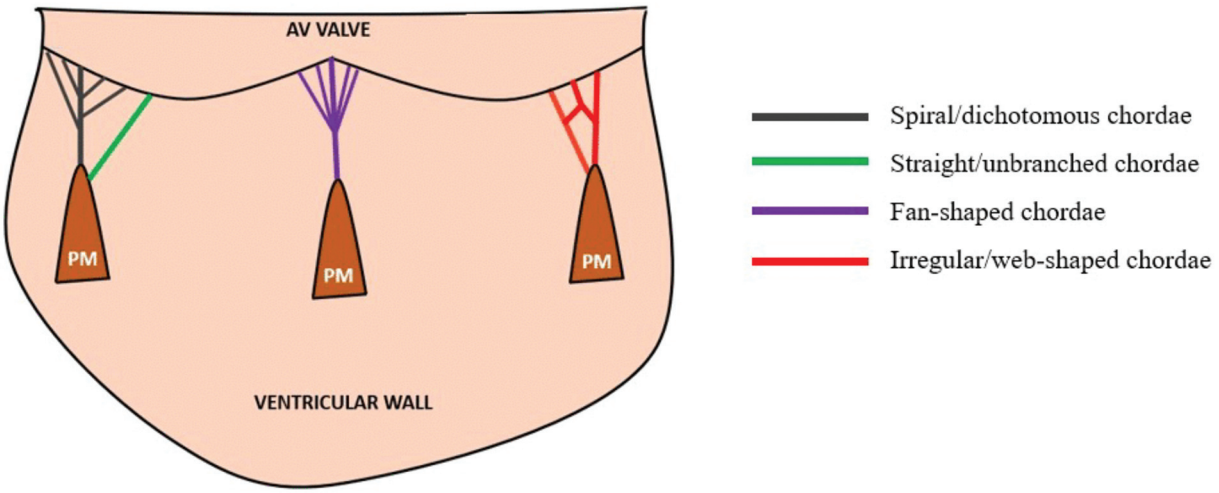

Figure 2. Branching pattern of tendinous cords. **AV:** atrioventricular; **PM:** papillary muscle.

pillar chordae arise from base of PM.[2] On AV valve, chordae can attach to various regions on the leaflet. Ventricular surface of each leaflet has free margin, rough zone, clear zone, and basal zone.[12,17] Clear zone is present between rough and basal zones.[18] Based on their shape and site of insertion onto valve, chordae in RV are mostly divided into five types such as rough zone, fan shaped, free edge, deep and basal. Rough zone chordae are the most observed type and always found in anterior and septal leaflets of TV.[11,18] Fan shaped chordae are found in antero-posterior and postero-septal commissures. Free edge chordae branch before inserting to leaflet margin and form a delta shaped expansion at insertion site.[11]

Gunnal et al.[2] have classified TC of MV into various types based on their attachment to different parts of MV complex. True chordae or cusp-pillar chordae arise from PM and insert into cusp of MV. These are further classified based on their site of insertion onto mitral leaflet as cusp chordae, cleft chordae, and commissural chordae. Commissural chordae play an important role in approximation of two adjacent cusps during ventricular contraction to bring about valve closure. Based on area of insertion and distribution of chordae on the cusps, TC are divided into different types. First order chordae are attached to free margins of valve leaflet and hence they are also called Marginal chordae or Free zone chordae or primary chordae Chordae inserted to free edges of AV valve, prevent marginal prolapse and align their zones of coaptation.[24]

Secondary chordae include cords which insert to ventricular surface of valve leaflet in general. These chordae are attached to rough zone of the valve and hence also termed as rough zone chordae.[2] Rough zone chordae are more common in posterior mitral leaflet (4–14), than anterior (3–10).[9] Among these rough zone chordae few are strong and tough and named as strut chordae, which often insert to cusp with broad aponeurosis.[2] Strut chordae are responsible for normal and non-homogenous movement of anterior mitral leaflet, and their absence can predispose to mitral regurgitation due to abnormal motion of leaflet.[17] Also, strut cords inhibit ballooning and allow leaflet's load to be distributed evenly and responsible for the tunnel-shaped configuration of MV.[15,17,24] Thus, primary or first order chordae are responsible for valve competence and secondary chordae are involved in maintenance of ventricular geometry and function.[37] Atypical rough zone chordae are those which have less than three chords. Lam et al.[15] examined 50 human hearts and found that 17% of rough zone chordae of anterior leaflet and 16% of those of posterior leaflet are atypical. In such regions of valve with atypical chords, adjacent chordae send branches to overcome chordal deficiency.

The chordae which arise from ventricular wall and insert to basal zone of ventricular surface of valve leaflet close to hinge line are termed as basal or tertiary or third order chordae.[15,17] Degandt et al.[5] compared the number of basal chordae of LV in three different species. They found 24.6±4.21 basal chordae in porcine hearts, 19.7±2.90 in ovine hearts, and 18.81±3.54 in human hearts. They also found that basal chordae always inserted to leaflet close to mitral annulus, except in anterior

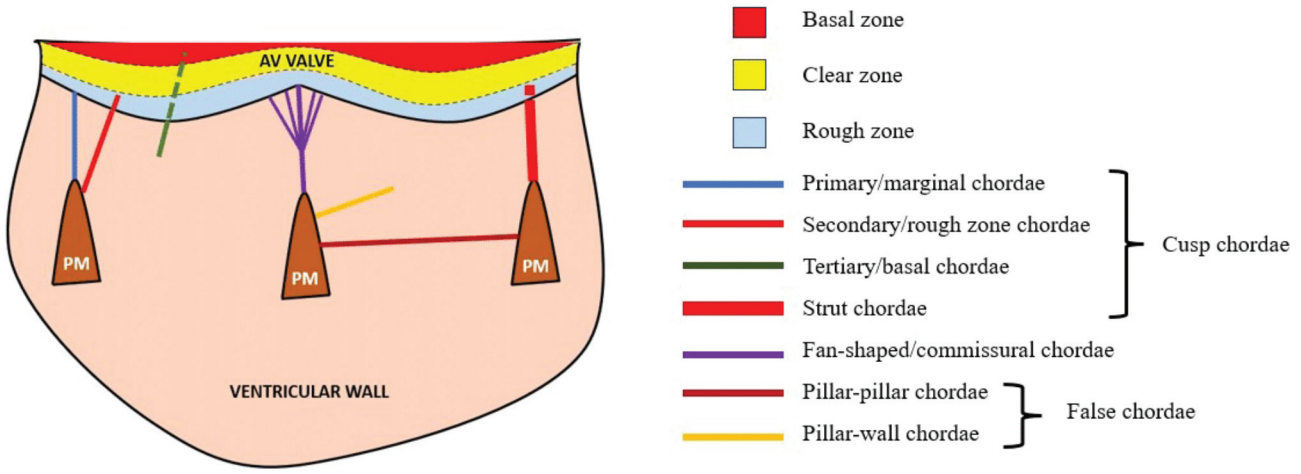

Figure 3. Zones of atrioventricular valve and types of tendinous cords attached. **AV:** atrioventricular; **PM:** papillary muscle.

mitral leaflet where it inserted between smooth and rough zones. Basal cords strengthen the ventricular component of AV junction and aid in maintaining proper ventricular shape.[24] Based on the site of attachment of chordae to leaflet they can also be divided into main, paramedian and paracommissural chordae. Commissural chordae can be anterior or posterior commissural chordae.[16] Nomenclature of cords often used clinically are marginal/primary, rough zone/secondary, basal/tertiary and strut chordae. The zones of AV valve and types of TC attached to them are shown in **Figure 3**.

Based on the gross appearance, TC are termed as tendinous, muscular, and membranous chordae.[2,12] Majority chordae in a ventricle are tendinous (100%) and next common type is muscular (14.65%). Membranous chords are least common type (6.03%).[2] Mundra et al.[12] found muscular chordae or chordae muscularis in 6.1% of heart specimens. The possibility of conduction of cardiac impulses through muscular cords, predisposes the heart for arrhythmias. Muscular cords are also often encountered in cases of hypertrophic cardiomyopathy and premature ventricular contraction.[27]

False chordae

TC which arises from PM or ventricular wall but not inserting to valve leaflet are termed as false chordae (**Figure 3**). False chordae may connect a PM to another PM, to ventricular wall or pass from one point to other on ventricular wall.[11,17,38] Sometimes a muscular band can connect two adjacent PM.[17] False chordae are classified as interpillar chordae (50%) and pillar wall chordae (6.03%). Interpillar chordae connect two PM and pillar wall chordae connect a PM to ventricular wall.[2] Ozog et al.[17] examined the LV and noted average number of false chordae arising from anterior PM as 3.5±2.2 and from posterior PM as 5.4±2.7. thus, a greater number of false chordae arose from posterior PM. Kosinski et al.[38] studied false chordae of RV and classified them into six groups, based on their pattern of attachment. Type I attach a PM to ventricular wall; type II chordae connect two individual segments of PM; type III connect a PM to interventricular septum; type IV connect various PM. False chordae attaching to ventricular wall around the apex are classified as type V. Type VI chordae connect the septomarginal trabeculae to other structures in RV.

False cords protect the ventricles from over dilatation. They are worth to note as they predispose to thromboembolic and arrhythmogenic events. Literature reports that they significantly influence the electromechanical functions of heart.[38] They are also found in LV outflow tract and significantly impair the transcatheter procedures related to aortic valve. Usually, these false chords cannot be visualized using imaging techniques as they are too thin, but thick false cords may lead to diagnostic errors in echocardiography.[17,38]

Morphometry of tendinous cords

In valve replacement and chordal repair surgeries, length of TC plays a vital role in deciding effective postoperative ventricular functioning and thus the outcome.[10] In LV, TC are 15–22 mm long and 0.45–0.66 mm broad on an average.[3,13] The average length of anterior PM chordae is 12.77±4.03 mm and its average breadth is

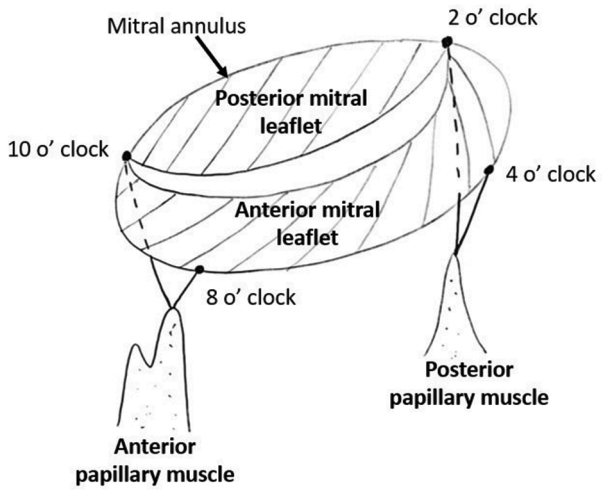

Figure 4. Schematic picture representing measurement of annulo-papillary distance in the mitral valve. The 2-o'clock and 10-o'clock positions are placed at the valvular commissures, and 4-o'clock and 8-o'clock positions are placed at the anterior margin of mitral annulus. For the papillary muscle with two heads, highest point is taken for the measurement.

0.28±0.20 mm. Whereas, on an average posterior PM chordae are 12.33±3.89 cm long and 0.25±0.14 mm broad. Thus, the length and breadth of anterior PM chordae is more than that of posterior PM chordae.[10] Rough zone chordae of anterior mitral leaflet are 1.75±0.25 cm long and 0.84±0.28 mm thick on an average. Similarly, in posterior mitral leaflet, rough zone chordae measure about 1.40±0.08 cm in length and 0.65±0.24 mm in thickness. Length of anterolateral and posteromedial commissural chordae is 1.2±0.31 cm and 1.4±0.40 cm, respectively. Posteromedial commissural chordae are thicker (1.0±0.30 mm) than anterolateral commissural chordae (0.70±0.20 mm).[15]

Kumar et al.[14] compared the length of TC in LV, in both cadaveric and autopsied heart specimens. Chordae attached to anterior mitral leaflet of both specimens were nearly same in length from 1.6 to 1.8 cm. In posterior and commissural mitral leaflets, a slight difference of 2–3 mm was observed, where chordae were longer in autopsied hearts. TC are thinnest near their insertion to MV leaflets. Hence chordal ruptures are most encountered at that site.[24]

Chordae attaching to anterior mitral cusp and MV commissures are lengthier in male, when compared to female but their average thickness is same (0.05 cm). Average length of chordae attaching to anterolateral and posteromedial commissures of MV in male are 1.4 cm and 1.7 cm respectively, whereas in female are 1 cm and 1.3 cm respectively. The average length of TC arising from anterolateral and posteromedial PM to the apex of anterior mitral leaflet in male are 2.1 cm and 2.2 cm respectively, whereas in female are 1.6 cm and 1.8 cm respectively.[16]

Annulo-papillary distance

Annulo-papillary distance is measured between the tip of PM to the annulus of AV valve of corresponding ventricle as shown in **Figure 4**.[14,39] In MV replacement and repair surgeries, annulo-papillary continuity is vital. Hence, a normal annulo-papillary distance should be maintained during repair for a proper resuspension. In LV, the distance from tip of anterior PM to left trigone (10-o'clock position) and to the point between anterior and middle scallops of mural leaflet (8-o'clock position) is 23.5±3.7 mm and 23.2±3.6 mm, respectively. Distance from the tip of posterior PM to right trigone (2-o'clock position) and to the point between middle and posterior scallops of mural leaflet (4-o'clock position) is 23.5±4.0 mm and 23.5±3.9 mm, respectively. Annulo-papillary distances of mitral apparatus are alike in various locations such as 2-, 4-, 8- and 10-o'clock positions. Mitral annular diameter correlates with annulo-papillary distance.[39] Kumar et al.[14] observed that the annulo-papillary distance in LV of both cadaveric and autopsied hearts was equal (2 cm). Kavitha et al.[40] examined 50 human cadaveric hearts and found that the mean vertical annulo-papillary distance was 16.56 mm.

Microstructure and histochemical properties

The human chordae are made of collagen and elastic fibers.[3,10] Scanning electron microscopic and light microscopic examination of human TC shows undulated planar wavy pattern of collagen fibrils arranged in longitudinal bundles along their entire circumference which forms the dense core.[3] These wavy crimp like collagen fibrils are intertwined with each other like a helix, which are heterogenous throughout the length of a chordae.[41] These collagen fibrils are of types I and III.[3,22] In human heart, the bundles of collagen fibrils are wavy when relaxed and become straight when stretched which helps in sustaining the peak stress developed during ventricular contractions.[20] Core connective tissue is loose in proximal parts of chordae, dense regular in middle parts and dense irregular in distal ends where they insert on AV valve.[42] The collagen fibril core is covered by endocardium which is simple squamous epithelium which is

continuous with other regions of heart. A dense layer of elastic fibers is present underneath the endocardium. Immunofluorescence studies show presence of thin irregular and longitudinal tenascin fibers in inner fibroblast coat of elastic fiber layer. Collagen fibrils are wavy in younger population but becomes less wavy or straight in adults.[20] Tenascin concentration in the elastic matrix surrounding collagen core increases with aging.[22] Wavy collagen fibrils elongate and reduce in cross sectional area as age advances in human which can lead to stretching and eventual rupture of chordae.[3]

Gupta et al. studied the histology of chordae in goat hearts and found that in a 32 days old goat embryo, the central core of TC was made up of myocardial cells with reticular fibers surrounded by single layer of endothelial cells. Chordae were mainly collagenous in mid and late prenatal periods. They also found few fibroblast, reticular and elastic fibers, and endothelial cells surrounding collagen core. Histochemical reaction exhibited the activity for existence of polysaccharides, lipids, DNA, and alkaline phosphatase in TC which increased with advancing age.[43] DNA and collagen content was more in anterior and posterior marginal chordae when compared to other types of chordae. Thus, microstructure of TC varies based on their type.[44]

The junctional area between PM and TC is histologically different in human and porcine hearts. In human hearts, collagen bundles are more organized forming a meshwork with fibers disposed in orthogonal angles whereas, in porcine hearts they are arranged randomly.[6] In avian hearts, papillary myotendinous junctions resemble skeletal myotendinous junctions in their protein composition, but their ultrastructure more closely resembles sites of thin filament-membrane association in smooth muscle. They exhibit minimal folding of the junctional membrane, resembling smooth muscle more than the highly folded junctions in humans. Avian papillary myotendinous junctions lack certain proteins found in human cardiac fasciae adherentes, such as alpha-actinin and zeugmatin.[45] Various current therapies available for repair of chordal failure, face issues of recurrence of disease and poor therapy outcome.[8] Thus, extensive data on microstructure of TC is imperative.

Microstructure and neurovascular components

Discrepancies exist among studies on vascularity of human TC and the literature lacks clarity. TC are traditionally referred to as avascular structures.[1,3] Few reports have described presence of blood vessels in them.[44,46,47] Ritchie et al.[44] examined porcine chordae in LV by performing special staining and immunohistochemistry. They observed that the blood vessels from PM, ascended longitudinally and in circumferential manner, encircling the chordae and supplied MV leaflets. Vascularization was observed more often in strut chordae of anterior mitral leaflet. Human fetuses and calves possess blood vessels in free margins of AV valve which anastomose with vessels in PM via chordae.[47] Blood vessels ranging from arterioles to capillaries are observed in human and porcine chordae.[46] In human chordae, lymphatic capillaries are also found within superficial layers of endocardial lining, especially near their origin from PM. There are no lymphatics in deep connective tissue layers of chordae.[48] Ichikawa et al.[49] examined TC in canine hearts and found the presence of lymphatic capillaries in junctional area between TC and PM, under electron microscope.

AV valves contain plexuses of nerves which are acetylcholine esterase positive, and are denser at their free margins where TC attach. These dense nerve plexuses extend into PM via TC attached to them. Such extension of nerve fibers into chordae is detected in porcine and canine hearts. AV valve and TC of rodents like mice, lack acetylcholine esterase positive nerve fibers.[50] In human, only few chordae contain nerve plexuses in them which are adrenergic or cholinergic in nature. The pattern of distribution of nerve fibers in chordae is however similar among human and porcine hearts. Most nerve fibers are located around blood vessels. Presence of nerve fibers in chordae suggests that AV valvular function is under neuronal control, through regulation of TC and PM.[46]

Biomechanical properties

In LV, posterior PM chordae are stiffer than anterior PM chordae due to increased collagen core ratio and dense collagen fibrils.[13] Thicker chordae are more extensible and less stiff when compared to thinner chordae. Thus, strut chordae are most extensible in nature. Marginal chordae are least extensible as they are the thinnest. Thin chordae have lower average fibril diameter, but greater average fibril density when compared to thicker chordae. Hence, they have a greater number of fibril-to-fibril interactions, and therefore a greater modulus.[51] Smaller chordae are less extensible and stiffer when compared with larger chordae. This inverse relationship between size and

Table 4
Comparison of human and animal tendinous cords.

Features	Human chordae	Animal chordae
Collagen bundles	Arranged orthogonally	Randomly arranged (porcine)
Stiffness	Stiffer	Less stiff (avian chordae)
Arrangement of blood vessels	Longitudinal	Longitudinal and circumferential (porcine)
Nerve fibres	Present in few chordae	Present in most chordae (porcine, canine, rodent)

stiffness of chordae maintains even surface of the valve leaflets during closure.[19] Chordae of younger population are more extensible when compared to adults, as collagen fibrils are wavier.[20] Human chordae are significantly stiffer than corresponding ovine chordae.[52] Paskaleva et al.[53] examined the porcine hearts and found that MV chordae were stiffer than TV chordae. They also noted that chordae attaching to septal leaflets were more extensible. It is necessary to know about the biomechanical properties of chordae to formulate different modalities of therapy for chordal repair and to improve treatment outcomes.[8] The features of human and animal TC are summarized in Table 4.

Conclusion

Most true chordae originate from apex of PM and insert onto rough zone of AV valve leaflet. Fan-shaped chordae are often present in commissures of the AV valves. In LV, APM chordae are longer and thicker than PPM chordae. The males have longer chordae compared to the females but there is no difference in TC thickness between two genders. Histologically, human and porcine TC are similar but collagen bundles in human chordae are more regularly arranged leading to higher tensile strength. LV chordae are stiffer than chordae of RV in both human and porcine hearts.

Compared to the related articles in literature, this review has provided the detailed overview of number, morphological types, neurovascular components, development of TC and its biomechanical properties in human and other different species. However, imaging studies and studies involving pathological hearts or symptomatic patients were not included. Meta-analysis could not be done due to limited data about morphology and morphometry of TC in both ventricles.

In future, data on morphometry of chordae from a large sample would help to formulate appropriate reparative procedures. Studies need to further explore the morphology of RV chordae and their insertion to TV, to compare the features of TC in RV and LV. It would aid in prevention of postoperative complications and recurrence. Understanding the ultrastructure and biomechanical properties of human and animal chordae would be useful to produce biological prosthesis for chordal replacement procedures.

Conflict of Interest

No potential conflict of interest relevant to this article was reported.

Author Contributions

SV: protocol development, literature search, search strategy, manuscript editing; RA: protocol development, literature search, search strategy, manuscript writing.

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