Structure of the human tricuspid valve leaflets and its chordae tendineae in unexpected death

A forensic autopsy study of 400 cases

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ABSTRACT

Objective: Congenital variations are known to be potential candidates for mechanical trauma leading to tricuspid valve lesions. For this reason, a detailed examination of heart valves as well as chordae tendineae should carefully be performed to clarify the reason of sudden death with no apparent cause. The aim of this study is to investigate the relationship of valvachordal anatomy in tricuspid valve in sudden deaths, and mainly cardiac disease in connection with its structure as well as leading congenital changes.

Methods: The 400 human hearts were collected between 2000 and 2002 from 400 autopsy cases during a medicolegal autopsy with permission from the Council of Forensic Medicine, Izmir, Turkey. Morphometric and morphological data were obtained in Ege University Faculty of Medicine, Department of Anatomy from each valve namely area, basal width, depth of leaflets, depth of commissure, number of chordae tendineae and their relation to the leaflets. These data were correlated for cardiac and noncardiac death cases.

Results: The results of this anatomical study may explain the increased incidence in wide variations of chordae tendineae in deaths of cardiac origin. In 40 hearts, we found 2 leaflets (20%), in 140 (70%) 3 leaflets and in 20 hearts there were 4 leaflets (10%) in deaths of noncardiac origin. We found 2 leaflets in 36 hearts (18%), 3 leaflets in 130 hearts (65%) and there were 4 leaflets in 34 hearts (17%) in deaths of cardiac origin. Although chordal abnormalities were extremely rare in cardiac death cases, some chordae tendineae retained a normal or near-normal appearance, while others were thickened and shortened in cardiac death cases. Higher ratio of abnormal chordae that were too short and too thick was also significant.

Conclusion: This condition negatively affects the feeding of chordae and leaflets and acts as a culprit of cardiac deaths, since aging starts at an early stage. This situation changes the verdict in legal affairs. For this reason, the valvachordal structure should be carefully examined prospectively in autopsies.

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The classic description of the tricuspid valve is found in the majority of 3 and in specialized works referring to 3 leaflets: anterior, septal and posterior.[1-6] Some authors also make reference to accessory or commissural cusps[3,4,6] and others admit the existence of different numbers of leaflets[6,7] or subdivided leaflets.[8,9] However, literature lacks properly established criteria to distinguish the habitual or supernumerary leaflet from a commissural leaflet. Recently, there has been a great development of heart operations for treatment of congenital heart disease[10-13] and tricuspid

regurgitation of leading blunt chest trauma.[14-20] The possibility of performing surgical interventions on the cardiac valves to treat a variety of diseases involving these structures demand precise knowledge of the valvachordal anatomy of each valve and of their major subdivisions.

Congenital abnormalities of the tricuspid valve, including Ebstein's malformation, dysplasia, found in pulmonary atresia with intact septum and congenitally corrected transposition, are uncommon causes of tricuspid regurgitation.[21] Congenital tricuspid valve anomalies are found as spectrums of disease in which both the leaflets and subvalvar apparatus are often involved.[22] Congenital variations are known to be potential candidates for mechanical trauma leading to tricuspid valve lesions.[16] Over time, progressive regurgitation can lead to right failure and arrhythmias, and death in such people.[10,12,15-20,23] Whether there exists relative connection between death and trauma, which is not the direct cause of death concerns the researches. For this reason, a detailed examination of heart valves as well as chordae tendineae should carefully be performed to clarify the reason of sudden death with no apparent cause. The relevance of valvachordal variations in rheumatic heart disease, reparative procedures, papillary muscle dysfunction, mitral valve prolapse and mitral valve replacement have been discussed in previous studies. All these studies relate anatomical analysis in adults: a specific study of the tricuspid valve in medicolegal autopsies is uncommon. However, valvachordal anatomy in origins of death has not been investigated. We believe that it is important to understand the anatomical characteristics of the right atrioventricular valve and its variation in cardiac surgery and forensic medicine. The present paper is intended to provide structures of right atrioventricular valve and its leaflets, investigate series of deaths of cardiac and noncardiac origin. In addition, the valvachordal anatomy and its variations were studied to determine the most frequent morphologic pattern in unexpected death cases.

Methods. The materials were collected from 400 sudden death cases (200 of cardiac origins and 200 of noncardiac origin) during a medicolegal autopsy with permission from the Council of Forensic Medicine, Ministry of Justice, Izmir, Turkey. We studied hearts with no macroscopic evidence of rheumatic and congenital lesions. There were 2 groups both of cardiac and noncardiac origins death including 60 hearts from females and 140 hearts from males. The age of the individuals, whose autopsies were performed between 2000 and 2002 in Izmir, Turkey, varied from 17-68 years. In all cases, a complete autopsy, toxicological screening and histological examination were performed. Deaths of unnatural origin or from alcohol and overdose of drugs were excluded from the study.

Each heart was sectioned along its acute margin. The section passed near the anteroposterior commissure of the right atrioventricular valve with an incision from the right atrium to the apex of the right ventricle. After opening, each heart was washed under tap water to remove blood clots. Dissection of the myocardium was carried out from the right atrioventricular fibrous ring to the origin of papillary muscles, preserving the integrity of the valve apparatus as a whole (**Figure 1**). The right atrioventricular valves were determined and valve was removed with their chordae tendineae and papillary muscles. After examination in the fresh state, the valve of the right ventricle was flattened on glass plates. Morphometric and morphological data were obtained from each valve: area, basal width, depth of leaflets, depth of commissure, number of chordae tendineae and their relation to the leaflets. These data were studied and measured under magnifying lens.

The leaflet surface was impressed on aluminum foil and cut to measure its area and set up an isometrical sample. This aluminum foil was placed on a smooth surface without changing the folds made by projection and indentation of the leaflet. The area of the leaflet surface was obtained by orthogonal projection on the paper. Boundaries of the projectors were drawn on the paper, and the areas were calculated using the point counting probes. The number of chordae attached to each leaflet, their distribution and arrangement, morphology at the site of their insertion and its attachment to the leaflet were investigated. The length of chordae from origin to insertion and chordal thickness at its midpoint were measured. Measurements were made with a digital caliper, a flexible metric rule and surgical silk thread. The right atrioventricular valves and chordae tendineae were photographed. Data were recorded into Statistical Package for Social Sciences for Windows.

Results. The annular circumference of the tricuspid valve was a large oval aperture and enough to admit the tips of 4 fingers. It was surrounded by a strong fibrous ring that was covered by the endocardial lining of the heart. That opening was 12.4 ± 1.1 cm in males and 11.8 ± 1.3 cm in females of noncardiac death origin. The annular circumference of the tricuspid valve of cardiac death origin was 11.2 ± 1.2 cm in males and 10.8 ± 1.1 cm in females. When viewed from the atrial aspect of the basal attachment of the tricuspid valve, orifice was roughly triangular with anterior, posterior and septal sides. In this study, the number of leaflets varied from 2-4 in deaths of both cardiac and

noncardiac origins. In 40 hearts, we found 2 leaflets (20%), in 140 (70%) 3 leaflets and in 20 there were 4 leaflets (10%) in deaths of noncardiac origin. We found 2 leaflets in 36 hearts (18%), 3 leaflets in 130 (65%) and there were 4 leaflets in 34 (17%) in deaths of cardiac origin (**Figure 2**). A commissural leaflet was present in 160 hearts (80% of the cases) in deaths of noncardiac origin. The number of commissural leaflets varied from one to 4 and in 78.2% of the cases, there was only one commissural leaflet. Their preferential localization was between the anterior and septal leaflets (42.1%), and between the posterior and septal leaflets (37.9%). Commissural leaflets were observed in 74% of the valves analyzed: in most specimens there was only one commissural leaflet located between the anterior and posterior leaflets in cardiac death cases. The number of commissural leaflets found in a single valve was not related with the number of supernumerary leaflets, that is, frequency of commissural leaflets with 2-3 leaflets was not different from that observed in valves with 4 leaflets. The surface of the leaflets was divided into 3 zones. Passing from free margin to the inserted margin. All leaflets of tricuspid valves displayed rough, clear and basal zones. The rough zone of the tricuspid leaflets was rough and thick on palpation and semiopaque on transillumination. It was rough at the point where chordae tendineae inserted. The clear zone was smooth and translucent, received few chordae tendineae and had a thinner fibrous core. The basal zone was a few millimeters wide and extended from the annulus of the posterior leaflets to the clear zone. Heights of rough and clear zones are summarized in **Table 1**.

In noncardiac death cases, the anterior leaflet interposed between the atrioventricular orifice and the anterior wall of the right ventricle. The anterior leaflet was triangular in 190 cases (95%) and rectangular in 10 cases (5%). The average area of the anterior leaflet was measured as 292.5 mm^2. The heights and widths of leaflets are presented in **Table 3**. The relationship between the width of the leaflet and its number demonstrated that the anterior leaflet was larger in valves with 2 leaflets than those with 3 or 4. The posterior leaflet extended between the anteroposterior and posteroseptal commissures. The posterior leaflet was the smallest (**Table 2**). The average area of the posterior leaflet was measured as 159.3 mm^2 in noncardiac origins. The posterior leaflet appeared as rectangular in 20 cases (10%), square in 20 (10%) and triangular in 160 (80%). The posterior leaflet of 60 hearts had only one scallop, while 84 had 2, 52 had 3, and 4 had 4. The scallops were of equal sizes in 28 leaflets, while the anteroposterior commissural scallop was larger in 52, and the posteroseptal in 16. No relationship was observed in terms of depth, width and number of chordae tendineae in the posterior leaflet when
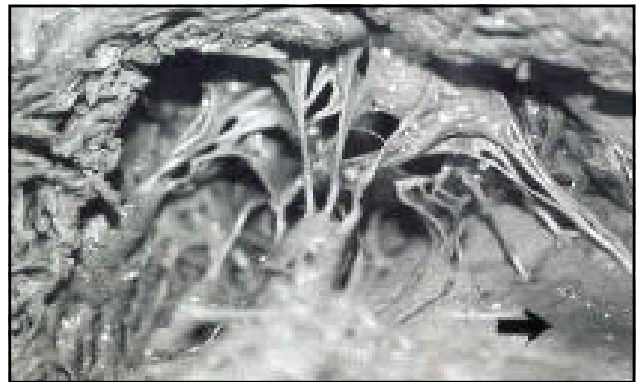

Figure 1 - Chordae tendineae attached to the free edges and ventricular surfaces of the anterior, posterior and septal leaflet.

Figure 2 - Four leaflets with the tricuspid valve.

Table 1 - Heights of rough and clear zones of the anterior, posterior and septal leaflets of the tricuspid valve in noncardiac and cardiac death cases.

Height (cm)	Anterior leaflet	Posterior leaflet	Septal leaflet
Rough zone			
Non-cardiac			
Male	1.3±0.2	1.1±0.2	1.1±0.1
Female	1.1±0.2	1.0±0.3	1.0±0.2
Cardiac			
Male	1.3±0.2	1.1±0.3	1.0±03
Female	1.0±0.2	1.0±0.3	1.1±0.3
Clear zone			
Non-cardiac			
Male	1.3±0.4	1±0.3	0.8±0.1
Female	1.1±0.2	0.9±0.2	0.7±0.2
Cardiac			
Male	1.3±0.3	1.1±0.3	0.8±0.3
Female	1.0±0.2	0.7±0.3	0.6±0.2
Rough and clear			
Non-cardiac			
Male	1	1.1	1.3
Female	1	1.1	1.4
Cardiac			
Male	1	1	1.2
Female	1	1.4	1.8

comparing valves with 2, 3 or 4 leaflets. The septal leaflet extended between the interventricular orifice and the right side of the interventricular septal wall. The average area of the septal leaflet was found to be the largest as 342.3 mm². The septal leaflet appeared rectangular in 32 cases (16%), square in 12 (6%) and triangular in 156 (78%). The heights and widths of leaflets are summarized in **Table 3**. The depth of the septal leaflet was larger in the bicuspid ones than in those with 3 or 4. No difference was observed in septal leaflets when comparing valves with 2, 3 or 4 leaflets as regards with leaflet width and number of chordae tendineae. Septal leaflet with chordal abnormalities was extremely rare.

In cardiac death cases, the anterior leaflet was the largest component of the tricuspid valve (**Table 2**). The anterior leaflet was triangular in 182 cases (91%), rectangular in 11 (5.5%) and square in 7 (3.5%). The sum of the average areas of the anterior leaflet was found to be 221.3 mm². Some chordae tendineae retained a normal or near-normal appearance, while others were thickened and shortened. The sum of the average areas of the posterior leaflet was measured as 220.2 mm² in deaths of cardiac origin. The posterior leaflet appeared as rectangular in 16 cases (8%), square in 14 (7%) and triangular in 170 (85%). The posterior leaflet had scallops all of which were small and had an approximately equal sizes (**Figure 3**). No difference was observed among deaths of cardiac origin. No relationship was observed in terms of depth, width and number of chordae tendineae in the posterior leaflet when comparing valves with 2, 3 or 4 leaflets. In most cases, congenitally dysplastic atrioventricular valves with abnormal chordae, too short, too thick or too long, were observed. The average areas of the septal leaflet was calculated as 212.7 mm². The septal leaflet appeared rectangular in 28 cases (14%), square in 21 (10.5%) and

Table 2 - Heights and widths of the anterior, posterior and septal leaflets of the tricuspid valve in cardiac and noncardiac death cases.

Leaflets	Non-cardiac deaths		Cardiac deaths	
	Male	**Female**	**Male**	**Female**
Anterior				
Height	2.4±0.3	1.9±0.4	2.5±0.4	2.4±0.3
Weight	3.8±0.6	3.4±0.1	4.0±0.3	3.6±0.4
Posterior				
Height	1.8±0.2	1.6±0.3	1.9±0.3	1.7±0.4
Weight	3.1±0.7	2.6±0.3	3.3±0.4	3.1±0.3
Septal				
Height	1.9±0.3	1.7±0.3	1.7±0.3	1.5±0.2
Weight	3.9±0.5	3.9±0.4	3.4±0.6	3.2±0.2

Table 3 - Average length and thickness of chordae tendinea of the tricuspid valve in cardiac and noncardiac death cases.

Chordae/insertion	Non-cardiac deaths		Cardiac deaths	
	Length (mm)	**Thickness (mm)**	**Length (mm)**	**Thickness (mm)**
Fan-shaped				
AP commissure	134.1±4.2	1.1±0.3	114.1±2.1	1.5±0.1
PS commissure	121.2±3.4	0.9±0.2	111.2±3.3	1.2±1.2
AS commissure	60.7±2.3	1.1±0.4	58.5±2.1	1.5±0.2
Posterior leaflet clefts	143.4±4.1	0.9±0.1	133.4±3.2	1.2±1.1
Rough zone				
Anterior leaflet	151.3±3.4	1.2±0.4	121.3±2.2	1.2±0.2
Posterior leaflet	145.6±2.3	0.8±0.2	154.6±2.5	1.8±0.1
Septal leaflet	172±6.5	0.9±0.3	182±2.7	1.1±0.2
Free edge				
Anterior leaflet	154.5±2.4	0.8±0.2	110.5±1.1	1.2±1.2
Posterior leaflet	163.6±3.4	0.8±0.1	133.6±1.2	1.8±1.1
Septal leaflet	142.9±1.5	0.8±0.2	122.9±1.3	1.4±1.2
Deep				
Anterior leaflet	173.5±5.4	1.1±0.4	123.5±1.4	1.2±0.1
Posterior leaflet	147.8±3.4	0.9±0.2	117.8±3.2	1.2±1.2
Septal leaflet	124.6±6.5	0.8±0.1	114.6±2.5	1.5±1.1
Basal				
Anterior leaflet	61.3±2.3	0.9±0.2	41.1±1.1	1.9±0.3
Posterior leaflet	65.2±2	0.9±0.2	26.1±1	1.7±0.1
Septal leaflet	63.4±3.1	1.1±0.5	43.2±1.1	1.3±0.2

AP - anteroposterior, PS - posteroseptal, AS - anteroseptal

Table 4 - Average number of each type of chordae tendinea in cardiac and noncardiac death cases.

Leaflet	Non-cardiac deaths average (range)		Cardiac deaths average (range)	
	Male	**Female**	**Male**	**Female**
Anterior				
Rough zone	5 (2-6)	3 (2-7)	4 (1-5)	2 (1-7)
Free edge	2 (0-2)	2 (0-5)	1 (0-3)	1 (1-4)
Deep	3 (0-5)	2 (0-7)	2 (0-4)	2 (0-2)
Basal	1 (0-3)	1 (1-3)	1 (1-2)	1 (1-2)
Posterior				
Fan-shaped	1 (0-2)	2 (0-3)	1 (0-1)	1 (0-1)
Rough zone	2 (0-4)	2 (1-6)	1 (0-3)	1 (1-2)
Free edge	2 (0-4)	1 (0-3)	1 (0-2)	1 (0-2)
Deep	1 (0-5)	2 (0-4)	1 (0-3)	1 (0-3)
Basal	2 (0-6)	1 (1-3)	1 (0-3)	1 (1-2)
Septal				
Rough zone	4 (2-7)	4 (1-7)	3 (1-4)	1 (1-2)
Free edge	2 (0-4)	2 (0-4)	1 (0-3)	1 (1-2)
Deep	1 (0-5)	2 (0-4)	1 (0-3)	1 (0-2)
Basal	3 (1-60	2 (0-2)	1 (1-3)	1 (1-2)
Commissures				
Anteroposterior	2 (0-1)	2 (0-1)	1 (0-1)	1 (1-2)
Posteroseptal	1 (1-2)	2 (1-2)	1 1	1 1
Anteroseptal	1 (0-1)	1 (0-1)	1 (0-1)	1 (1-3)
Total	**33 (6-63)**	**31 (7-62)**	**22 (5-42)**	**18 (11-39)**

triangular in 151 (75.5%). In the majority of cases with septal leaflet abnormally short cords were observed. Leaflets and cords were also thickened.

Chordae tendinea. Five types of chordae were attached to the tricuspid valve: these were fan-shaped, rough zone, basal, free edge and deep chordae. The number was approximately 20 and they were of different lengths and thicknesses. They originated from a papillary muscle either directly from the apex of the muscle or from small nipples, which were usually on their upper third. The chordae were attached to the ventricular end of the valve leaflets and their apices and margins, and then, anchored to the muscular ventricular wall. The majority of the chordae branched soon after their origin, some branched just before insertion and a few did not branch at all. Those inserted into the lateral margins of the leaflets or into the scallops of the posterior leaflet and passed at an oblique angle from their origin to insertion. The average length and width of the chordae are shown in **Table 3**. Each type of chordae tendineae of leaflet is presented in **Table 4**. Fan-shaped chordae had a short stem from which branches radiated to attach the margin of zones. Fan-shaped chordae were present at the anteroposterior commissure in 186 hearts, at the posteroseptal commissure in 200, and at the anteroseptal in 162 noncardiac death cases, while they were present at the anteroposterior commissure in 190 hearts, at the posteroseptal commissure in 210, and at the anteroseptal in 170 in cardiac death cases. At dissection, thickened leaflets with fused commissures were noted along with thickened and shortened chordae (**Figures 4 & 5**). Rough zone chordae was inserted into the rough zone on the ventricular aspect of each leaflet. Each chorda split into 3 cords soon after its origin. One cord was inserted into the upper limit of the rough zone at the line of closure, and another between the other 2. Rough zone chordae were attached to the anterior leaflet in all 200 hearts, to the posterior leaflet in 164, and to the septal leaflet in 196 noncardiac death cases, whereas they were attached to the anterior leaflet in all 200 hearts, to the posterior leaflet in 170, and to the septal leaflet in 190 cardiac death cases. Atypical rough zone chordae, with only cords, were frequent in the tricuspid valve in cardiac death cases. Atypical rough zone chordae originated from the trunk of the papillary muscle (**Figure 6**), those related to the anteroseptal margins of the septal and anterior leaflets were often short. Single, thread-like or often long free edge chordae originated from the apex of the papillary muscle. They branched before insertion, and their fine subdivisions formed a delta-shaped insertion at the free edge. One or more were found attached to 120 anterior, 126 posterior, and 112 septal leaflets in noncardiac deaths. Some chordae tendineae retained a normal or near-normal appearance, while others were thickened and shortened. The near-normal shortened chordae were excised with the attached valve leaflet. Deep chordae branched to various extents and reached the more peripheral rough zone or clear zone. The cord-like branches often had a triangular fold of membranous tissue passing through each other and the leaflet just before their insertion. Such chordae were attached to the anterior leaflet in 152, to the posterior leaflet in 116, and to the septal leaflet in 132 hearts in noncardiac death cases. Deep chordae were attached to the anterior leaflet in 160, to the posterior leaflet in 130, and to the septal leaflet in 140 hearts in cardiac death cases. A congenitally dysplastic atrioventricular valve with abnormal chordae, too short, too thick, too long or ruptured, was found (**Figure 7**). Basal chordae were the round ones or flat ribbons, long and slender or short and muscular. They arose from the smooth or trabeculated ventricular wall and attached to the basal component of a leaflet. They arose directly from the myocardium or from small trabeculae carnae. They inserted a zone approximately 2 mm wide extending into the leaflet from the annular region. Basal chordae were found to be attached to 86 anterior, 100 posterior, and 180 septal leaflets in noncardiac death cases, while they were found to be attached to 90 anterior, 120 posterior, and 170 septal leaflets in cardiac death cases. Chordae tendineae attached to the free edges and ventricular surfaces of the anterior, posterior and septal leaflets. On average, 34 chordae inserted into the tricuspid valve in noncardiac death cases. There was no significant difference between the total numbers of the 2 sexes in noncardiac and cardiac deaths cases. Of the 34 chordae, 12 passed to the anterior leaflet, 7 to the posterior leaflet and 11 to the septal leaflet, and 4 inserted into the commissural areas. On average, 35 chordae inserted into the tricuspid valve in cardiac death cases. There was no significant difference between the total number of the 2 sexes in noncardiac and cardiac deaths cases. Of the 35 chordae, 11 passed to the anterior leaflet, 8 to the posterior leaflet and 10 to the septal leaflet, and 6 were inserted into the commissural areas.

Discussion. The tricuspid valve complex consists of functional units, which include the valve leaflets, chordae tendineae and papillary muscles.[24] The mechanical properties of these functional units depend on the large extent of the link between the muscle and the valve. This link is usually arranged in a branching network of avascular chordae tendineae composed of collagen and elastic fibers, which transmit contractions of the papillary muscle to the valve leaflets.[25,26] Although it has been known for many years that the tricuspid valve has 3 leaflets. Modern anatomy text books suggest that the number of leaflets may be found between the
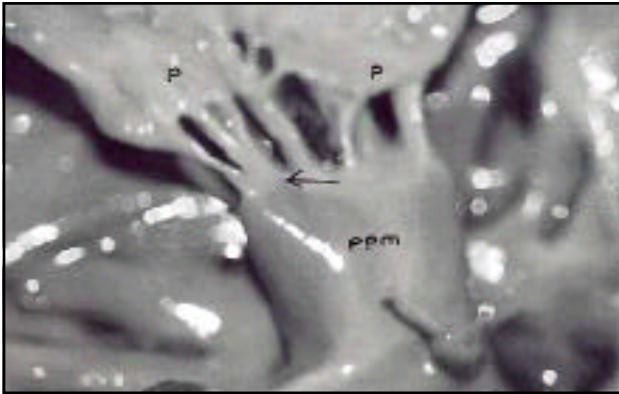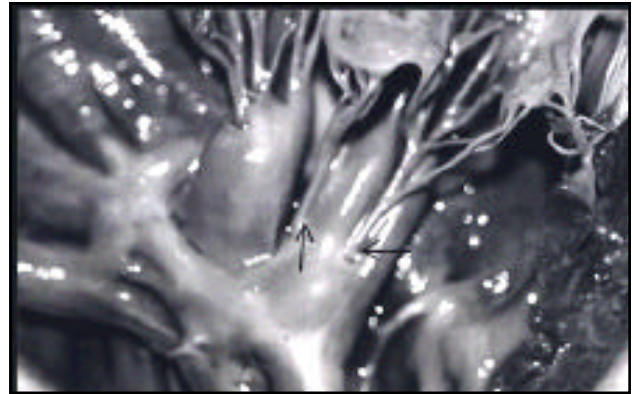
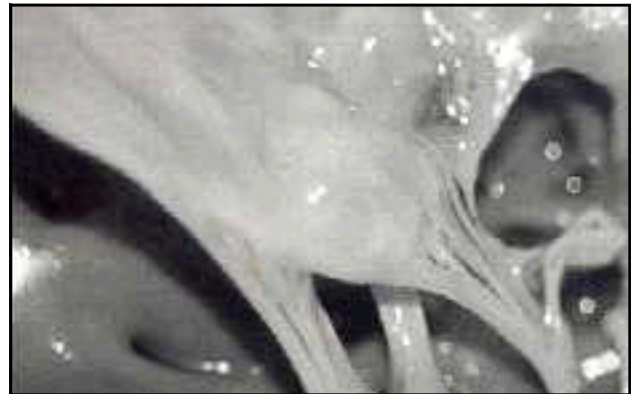**Figure 3**

Figure 6

Figure 4

Figure 7

Figure 5

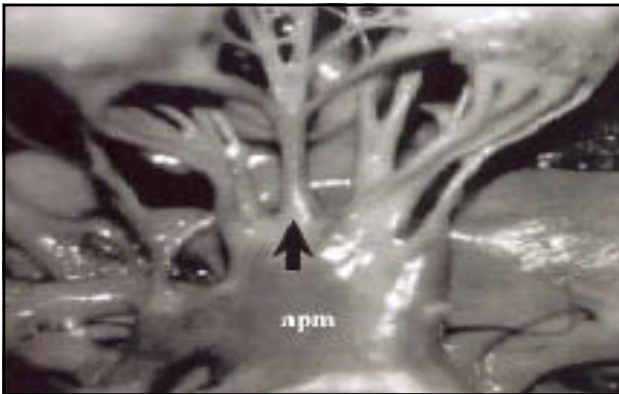**Figure 3 -** The posterior leaflet was divided into 2 scallops (p) by clefts into which short and thick fan shaped chordae (arrow) inserted. PPM -posterior papillary muscle

Figure 4 - Short and thick fan-shaped chordae were atypical.

Figure 5 - Short and thick fan-shaped chordae attached to the leaflet. Short and thick fan-shaped chordae apex of the apm. apm - anterior papillary muscle

Figure 6 - Atypical rough zone chordae originating from the trunk of the papillary muscle.

Figure 7 - Short deep chordae (arrow) attached to the upper limit of the rough zone of the septal leaflet.

main leaflets. Based on morphological and morphometrical criteria, tricuspid valve can be formed by 2-6 habitual/supernumerary leaflets besides commissural leaflets.[1-6] The chordae tendineae, associated with the tricuspid valve, originated from a major anterior papillary muscle, a minor posterior papillary muscle and the septal wall. The anterior and septal leaflets were the major components of this valve and received their major support from chordae tendineae originating from the anterior papillary muscle and the septal wall.

Variations in size of habitual leaflets resulting from the presence of supernumerary leaflets occur mainly at the lateral half of the wall of the right ventricle. Morphometrical criteria such as area of leaflet, the basal width and the ratio of commissure depth to leaflet depth can be utilized for characterization of the tricuspid valve leaflets. The great variation in leaflet appearances is probably due to embryologic development. Division of the common atrioventricular canal into the right and left channels starts in the embryo of 40 days. These channels, which are surrounded by mesenchymal subendothelial tissue called endocardial cushions that contribute to the future atrioventricular valve, grow and straighten to approximate the margins of these endocardial cushions.[23] Recently, using scanning electron microscopic and immunohistochemical techniques have been described as developing equally from endocardial cushions and the myocardium. The inferior leaflet is formed from the lateral and inferior wall of the myocardial gully and the anterosuperior leaflet from the supraventricular crest, which develops from the intraventricular part of the muscular outlet septum. This difference could explain the anatomical differences between the anterior and posterior regions.[4]

In particular, minor anatomical differences in tricuspid valve anatomy are frequent.[3] The tricuspid valve is not always in the same shape and configuration. Minor degrees of variations have no more effect than changing function of the heart.[27,28] Controversies still exist on the importance of these variations and put the human beings at risk of unexpected deaths.[29]

The left atrioventricular valve and its papillary muscle and chordae tendineae have been largely studied.[2,25,30-34] Victor and Nayak[9] pointed out that the internal morphology of the 2 cardiac chambers was extremely complicated and remarkably variable. In particular, the observation of chordae tendineae of the right ventricle showed endless variability in their number, shape and location.[8,24,35] The number, size, location and morphology of the right papillary muscle and their correlation to age and race were studied in an attempt to provide a classification.[27] A normal right ventricular cavity has coarse trabeculations, with diffuse and variable

chordal attachment to papillary muscles within the body of the ventricle and to the interventricular septum. The cavity of the right ventricle is triangular in shape and contains an identifiable muscular moderator band. It is guarded at its inflow by a tricuspid valve of variable anatomy that is separated from the outflow vessels by an infundibular muscular band.[5]

With aging, the tricuspid valve, like other tissues, changes; this changes consist mainly of local fibrous thickening of both the papillary muscle and chordae. The right ventricular cavity becomes smaller as we grow older, and consequently, less area becomes available in the left ventricle to accommodate the tricuspid valve leaflets and chordae. The leaflets then contact one another abnormally during ventricular systole and focal fibrous thickening results. As the right ventricular cavity diminishes with age, so does the tricuspid annulus. The latter change further increases the abnormal contact of the tricuspid leaflets with one another and furthers the leaflet scarring. This scarring from aging, however, is not enough to cause tricuspid dysfunction. Sato and Shimada[36] demonstrated that Tenascin and collagenous matrix components showed a notably higher accumulating of elastic fiber concentration in the age group of 80s and 90s than in that of 20s. Extracellular matrix components may be expressed during aging and by altered stress. Sato and Shimada[36] also demonstrated that young people have ring-like structures but older people do not. The ring-like structures may be reducing mechanical tightening, twisting and slackening of the chordae tendineae. dos Santos et al[18] reported ruptured chordae tendineae of the posterior leaflet of the tricuspid valve as a cause of tricuspid regurgitation following blunt chest trauma. Scorretti[19] reported a case of traumatic rupture of anterior papillary muscle. Becker et al[11] defined tricuspid valve dysplasia as faulty development of chordae and papillary muscle, and thickening or focal agenesis of valvular tissue in congenitally malformed tricuspid valves.[37,38]

McElhinney et al[39] reported a distinct entity in which tricuspid valvar regurgitation resulted from failure of coaptation due to short chordae tendineae tethering the septal leaflet. Asymmetric chordae tendineae of the septal leaflet is a distinct cause of tricuspid regurgitation that can be recognized with autopsy. Although rare, the importance of recognizing these variations lie in their being readily amenable to investigation. They found that some chordae tendineae retained a normal or near-normal appearance, while others were thickened and shortened. In the anatomical investigation, a central regurgitant jet of moderate or severe degree was directed towards the atrial septum through poorly coapting tricuspid valvar leaflets, which did not approximate due to tethering of the septal leaflet by

abnormally short cords.[35] The leaflets and cords were also thickened. In their study Oosthoek et al[40] reported on mitral valves with focalized attachment of cords. They demonstrated in autopsy specimens asymmetric mitral valves with 2 papillary muscles were present, and one of the muscles was elongated, located higher in the left ventricle with its tip reaching to the annulus. Some authors have proposed that some minor changes of the tricuspid valve becomes incompetent under adverse hemodynamic circumstances.[41-44] There is limited research on physical, ontogenetic and forensic studies of valvular structure in sudden death. As a result of studying literature, it has been noted that *there is no exist studies investigating the heart anatomically in deaths of cardiac and noncardiac origins.* In this respect, it can be said that the findings of the present study are quite significant and the data from other researchers which will be used for discussion are rather limited. Other studies presented without basing the data on the tricuspid valves and chordae tendineae on the cause of death may therefore lead to misleading comments.

Our findings indicated that the average area of the septal leaflet (342.3 mm²) was slightly larger than the anterior leaflet (292.5 mm²) and posterior leaflet (159.3 mm²) in deaths of noncardiac origin. Comparing the data of deaths of cardiac and noncardiac origin, anterior leaflets would be considered to be the largest. The sum of average areas of the anterior leaflet (221.3 mm²) and posterior leaflet (220.2 mm²) happened to be higher than those of the septal leaflet (212.7 mm²) in deaths of cardiac origin. In cardiac death cases, anterior leaflets were frequent in all valves and the width of the base was greater in valves with 2 leaflets than in those with 3 or 4.

The results of this anatomical study may explain the increased incidence in wide variations of chordae tendineae in deaths of cardiac origin. Although chordal abnormalities were extremely rare in cardiac death cases, some chordae tendineae retained a normal or near-normal appearance, while others were thickened and shortened in cardiac death cases. Higher ratio of the abnormal chordae that were too short and too thick is also significant. Short, thick or differing from normal can be easily ruptured with chest trauma. This may exhibit individual differences. This condition negatively affects the feeding of the chordae and leaflets, and acts as a culprit of cardiac deaths, since aging starts at an early stage. Types of sudden deaths and traumatic lesions may be associated with minor abnormalities of valvachordal anatomy. Autopsy-investigation requires a good knowledge of the valvachordal anatomy. In our future studies, our aim will be to increase the number 4 cases varying in terms of age, and to investigate the valvachordal changes, which may cause sudden deaths.

Although there are numerous origins of natural deaths, the cardiovascular origin comprises the majority, a conclusion in which the present results confirm those of the previous investigations. We may conclude that autopsies of deaths are as important as their origins. A damage to valves or papillary muscles may occur after a trauma, and ischemia may further lead to even death, negatively affecting valve functions and creating losses in the functional capacity of the body. Congenital variations are known to be potential candidates for mechanical trauma and trauma leading to chorda-papillary muscle lesions. Whether a relative connection between death and trauma which is not the direct cause of death exists the concerns of the researchers. For this reason, a detailed examination of heart valves, as well as valvachordal structure, should be carefully performed to clarify the reason of sudden death with no apparent cause.

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