

# ANI ÖLÜM OLGULARINDA KARDİYAK İLETİ SİSTEMİNİN DEĞERLENDİRİLMESİ

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## ÖZET

### Amaç:

Bu çalışmada, ani kardiyak ölüm vakalarında kardiyak ileti sistemi patolojilerinin bölgesel özelliklerinin ortaya konması amaçlandı. Ayrıca bu çalışma, kardiyak ileti sistemi patolojileri konusunda Adana bölgesindeki adli olgularda yapılan ilk çalışmadır.

### Yöntemler:

Yirmi yedi ani-beklenmedik ölüm olgusu ile ölüm nedeni bilinen 4 olgu kontrol grubu olarak çalışmaya alındı. Toplam 31 olguda, sinoatrial nod ve atrioventriküler nod bölgelerinden örnekler alındı. Örnekler histokimyasal boyama yöntemleri ile boyanarak ışık mikroskopunda incelendi.

### Bulgular:

Ani-beklenmedik ölüm olgularının 10'unda sinoatrial nod ve atrioventriküler nodda belirgin patolojik bulgular saptandı. Tüm olguların 3'ünde (%11,1), ileti sistemi anomalilerinin ölüme neden olabileceği düşünüldü.

### Sonuç:

Ani beklenmedik ölüm olgularında kardiyak ileti sistemi değerlendirilmesinin önemli olduğu ve adli patolojide bu konunun göz önünde bulundurulması gerektiği sonucuna vardık.

**Anahtar Kelimeler:** adli patoloji, kardiyak ileti sistemi, ani ölüm, otopsi

# EVALUATION OF CARDIAC CONDUCTION SYSTEM IN SUDDEN DEATH CASES

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## ABSTRACT

### Objective:

In this study, revealing the regional characteristics of the cardiac conduction system pathologies in sudden death cases is aimed. Besides, this is the first study of Adana region forensic cases related to the cardiac conduction system pathologies.

### Methods:

Twenty seven cases of sudden unexpected death and 4 cases with known causes of death as a control group have been included in the study. Samples from sinoatrial node and atrioventricular node were taken in total 31 cases. Samples were evaluated with light microscope after staining through histochemical staining methods.

### Results:

There were significant pathological changes of the sinoatrial node and atrioventricular node in 10 of the cases. And in 3 of all cases (11.1%), the abnormalities of the conduction system were considered to have the possibility for causing death.

### Conclusion:

We have concluded that it is of importance to evaluate the cardiac conduction system in sudden unexpected death cases and this topic should be kept in sight in forensic pathology.

**Key words:** forensic pathology, cardiac conduction system, sudden death, autopsy

## INTRODUCTION

A forensic pathologist is frequently asked to find out the cause of death in cases of sudden unexpected deaths in adults. Approximately 50% of all medico – legal deaths are due to natural causes and 1–5% of all those cases remain as negative autopsies against to all odds (1-6).

Sudden cardiac death is usually defined as the death from cardiac causes without apparent antecedent symptoms or within the first hour after the onset of symptoms. Studies of morbidity and mortality related to cardiac diseases estimate that there are between 300.000 and 400.000 sudden cardiac deaths annually in the United States (7). In this study, sudden death has been accepted as the death occurring within couple of hours (3 - 4 hours) without significant diagnosis of the cause of death. Sudden unexpected death (SUD) due to some other diseases, or toxic substances were eliminated during the study; we have chosen the cases with cardiac pathology and /or cases without significant pathological lesions or the cases considered as negative autopsy. On the other hand, we have planned this preliminary study because of the lack of the cardiac diseases series with autopsy findings at our region Adana and in our country, Turkey.

The statutes of the country make sudden deaths reportable to the legal authorities, usually if the person was previously healthy and review of the medical history

does not elicit the probable cause of death. As a result, forensic medicine specialists or forensic pathologists frequently encounter cases of sudden cardiac death. Approximately 80% of sudden cardiac deaths are caused by atherosclerotic coronary artery disease including all its manifestations (fixed coronary obstructions, coronary spasm, plaque rupture-erosion with coronary thrombosis, acute-healed myocardial infarction and chronic ischemia). Because essential hypertension is a frequent comorbidity of atherosclerotic heart disease, left ventricular hypertrophy may also be present. Any increase in left ventricular mass deteriorates the imbalance between myocardial oxygen supply and demand, thereby increasing the likelihood of ischemia and sudden death (8).

In hearts without significant coronary atherosclerosis, a variety of other cardiac diseases may provide the anatomic basis for sudden death. Most of the practicing forensic medicine specialists or forensic pathologists are comfortable diagnosing the diseases of the heart muscle (hypertensive heart disease, cardiomyopathies, myocarditis) and cardiac valves (mitral valve prolapse, aortic stenosis). Yet examination of the cardiac conduction system is often considered as a last choice in the evaluation of a sudden death case (9,10).

## MATERIAL AND METHOD

Twenty seven SUD cases and 4 cases having known causes of death have been chosen for this study. The autopsies were performed in the Morgue Department of the Council of Forensic Medicine Adana Branch Office. It is legally allowed by the Turkish Criminal Code and Turkish Code of Criminal Procedure to perform research on the routine pathologic samples of the autopsy materials and to publish those without mentioning the names of the deceased or information about his / her identity. A total of 31 cases have been included in this study without gender classification. All of the cases were toxicologically screened for drugs or any other chemical agents and were found negative for toxic substances. The cardiac tissue and coronary artery samples were dissected as described by the College of American Pathologists (11). The Cardiac Conduction System was examined as already described by Cohle et al and Gulino Sam P (2,12). Gross dissection of the conduction system, as SA and AV nodes, was performed after the coronary arteries' examination and the myocardium's evaluation by multiple sections across the short axis of the heart. All of the dissections and examinations were performed before fixation. It is probably only a matter of time before conduction system analysis becomes a standard procedure in cases of apparent sudden cardiac death with a structurally

normal heart.

SA node was dissected by opening the right atrium from the entrance zone of the Vena Cava Superior with the protection of the sulcus terminalis. AV node was demonstrated by dissecting the zone in between the front line of the coronary sinus to the right ventriculi medial papillary muscle, entire Koch triangle and 1.5 cm upper region of the interventricular septum. This block has been taken out including 1 cm sample of both the atrial and ventricular region of the tricuspid valve.

All of the samples were preserved and fixed in 10% buffered formalin solution. Tissue sections, taken as 7 to 8 slide sections, have been prepared both for SA and AV nodes. Myocardium and coronary arteries of the heart were sectioned for routine analyses. All of the samples, with their anterior faces down, were placed in tissue cassettes for processing and embedding. 5 µ sections stained by Harris' hematoxylin and eosin (H+E), connective tissue stain of Masson's trichrome and Verhoeff's elastic Van Gieson and also for amyloidosis: Lieb's Crystal Violet. The slides have been examined by a pathologist

using light microscope.

## RESULTS

There were 31 autopsy cases in this study. They were 27 of SUD and 4 control. Twenty (74%) of the SUD cases were males while the rest 7 (26%) were females (Table 1). The cases were differing in a range of 17 to 78 years of age. Most of the cases were in between the ages of 41 and 50. Female/male ratio was 1/4. Drowning, stabbing, electrocution and intoxication were the causes of death in control cases.

All of the SUD cases were examined in terms of coronary artery, myocardial and conduction tissue histopathologic findings. Fifteen cases were having serious damage of coronary atherosclerosis (75% and over luminal obstruction). Mostly, in 70% of the cases, both descending and circumflex branches of the coronary arteries were affected by the atherosclerosis. In rest of the cases, atherosclerosis was mainly remarkable in descending coronary artery yet combined affection of atherosclerosis was also noticed in both right and left coronaries or in all three of them. Eighteen

cases were found to have hypertrophic changes of the left ventricle. Thirteen cases were found have old infarctions of patchy style in the left ventricle, while 4 of them were having both, old and fresh infarctions. The infarctions were located in the left ventricle at the papillary muscle level. Old, chronic hypoxic changes of the myocytes were also observed at this region.

Twenty one cases were reported to have coronary artery and myocardial insufficiency as the cause of death. Also in 7 of those cases, there were conduction system pathologies. In 6 of the cases of all 27, the etiology remained as unknown or the definition was negative autopsy. There were pathologic changes of the conduction tissue in 4 of that 6 cases having unknown etiology. 11 (40.7%) of all cases, pathologic changes were recorded (Table 2).

Most of the SUD cases were determined to have died during resting period, either at their home or at a cafe while having conversation with other people. The man in case 7 died after an exercise, while woman in case 13 died early in the morning when she was on her way for bathroom. The man in

**Table 1:** Age and sex distribution of the SUD cases

Age / Sex	11-20	21-30	31-40	41-50	51-60	Age 60 and over	Total
Male	3	4	2	6	4	1	20
Female	1	-	2	1	2	1	7
Total	4	4	4	7	6	2	27

**Table 2:** Distribution of the cases by age, sex, heart weight, coronary artery and myocardial tissue findings and conduction tissue pathologies.

Case Nr:	Age Sex	Heart Weight	Coronary arteries	Coronary Tissue	Cause of Death	Conduction System Pathology SA nod / AV nod	
1	30/M	440gr	Fully Obs.	*Ch. Hyp. **Old infarction	Cardiovascular Insufficiency	-	-
2	40/M	450gr	50-60% Obs.	*Ch.Hyp.	Cardiovascular Insufficiency	-	-
3	32/F	300gr	10-20% Obs.	Normal	Unidentified	-	-
4	28/M	340gr	70-80% Obs.	***Hyp Changes	Unidentified	-	-
5	53/F	370gr	70-80 % Obs.	*Ch. Hyp.	Cardiovascular Insufficiency	-	-
6	76/M	550gr	Fully Obs.	*Ch. Hyp. **Old infarction	Cardiovascular Insufficiency	-	-
7	17/M	445gr	50% Obs.	***Hyp Changes	Unidentified	+	+
8	57/M	450gr	70-80% Obs.	*Ch. Hyp. **Old infarction	Cardiovascular Insufficiency	-	-
9	48/M	530gr	Fully Obs.	***Hyp Changes Old & New infarction	Cardiovascular Insufficiency	+	-
10	42/M	590gr	40-50% Obs.	*Ch. Hyp. **Old infarction	Cardiovascular Insufficiency	+	-
11	54/M	445gr	Fully Obs.	*Ch. Hyp. **Old & new infarction	Cardiovascular Insufficiency	-	-
12	78/F	340gr	70%Obs.	*Ch. Hyp. **Old infarction	Cardiovascular Insufficiency	-	-
13	18/F	240gr	Normal	Normal	Unidentified	+	+
14	42/M	410gr	Fully Obs.	*Ch. Hyp. *Old & new infarction	Cardiovascular Insufficiency	-	-
15	22/M	350gr	60-70% Obs.	*Ch. Hyp. **Old infarction	Cardiovascular Insufficiency	-	+
16	30/M	420gr	50-60% Obs.	*Ch. Hyp. **Old infarction	Cardiovascular Insufficiency	+	+
17	48/F	360gr	70-80% Obs.	*Ch. Hyp. **Old & new infarction	Cardiovascular Insufficiency	-	-
18	17/M	320gr	40-50% Obs.	*Ch. Hyp.	Cardiovascular Insufficiency	+	-
19	60/M	400gr	70% Obs.	*Ch. Hyp.	Cardiovascular Insufficiency	-	+
20	53/M	615gr	Fully Obs.	***Hyp Changes **Old infarction	Cardiovascular Insufficiency	-	-
21	31/F	320gr	Minimal Change	***Hyp Changes **Old infarction	Cardiovascular Insufficiency	-	-
22	18/M	350gr	60-70%	Patchy Hyp.	Unidentified	-	+
23	48/M	580gr	Fully Obs.	*Ch.Hyp.	Cardiovascular Insufficiency	-	-
24	48/M	740gr	Fully Obs.	*Ch.Hyp **Old infarction	Cardiovascular Insufficiency	-	-
25	54/F	300gr	60-70% Obs.	*Ch.Hyp **Old infarction	Cardiovascular Insufficiency	-	-
26	38/M	390gr	10-20% Obs.	*Ch. Hyp	Unidentified	-	+
27	46/M	375gr	80-90% Obs.	*Ch.Hyp **Old infarction	Cardiovascular Insufficiency	+	-
<b>Control Cases</b>							
1	14/F	260gr	Normal Adipose tissue Infiltration	Grade II	Intoxication	-	-
2	62/M	350gr	50% Obs.	Patchy Hyp.	Electrocution	-	-
3	44/M	330gr	10-20% Obs.	Normal	Stabbing	-	-
4	65/M	370gr	Fully Obs.	*Ch.Hyp	Drowning	-	-

\*Ch.Hyp : Chronic Hypoxic Changes    \*\* : Old and / or New Infarction    \*\*\*Hyp Changes: Hypoxic Changes

case 18 was having an interesting history, he was taking a shower, and there was a butane gas using water heater in the bathroom for hot water. Since they couldn't hear from him for a long time, his parents became worried for him and when they opened the door, they found him unconscious laying in the bath tub. The case was firstly considered as carbon-monoxide intoxication, however blood toxicological analyses revealed that there was no recordable carboxyhemoglobin. The case was accepted as a SUD.

Four of the control cases had been defined to have died of non-cardiac causes. We found slight fibrosis in 3 of them and slight adiposis in 2. We also found slight fibromuscular dysplastic changes in 2 of the control cases on both SA and AV nodes. In none of the control cases there were significant pathologic changes. However in control case number 2, there was a slight change of fibroadipose tissue and fibromuscular dysplasia of grade 1 which can be considered in normal ranges according to the age.

In consideration of the 27 cases, we found serious fibrotic changes in 26% of both SA and AV nodes (7 cases). Remarkable adipose tissue changes of SA and AV nodes were noted in 29% (8 cases) and 26% (7 cases) respectively. Fibromuscular dysplasia was found in the SA nodes of the 17 cases (62.9%), while it was found in 16 cases' (59.2%) AV nodes. The pathological findings of those nodes have been listed in Table 3.

Second degree fibroadipose changes in both SA and AV nodes as well as serious fibromuscular changes of the nodal arteries were noticed in the case number 7. Since he was at a young age (17/M), those findings were accepted as pathological findings. Second degree of fibrosis, third degree of adiposis and serious fibromuscular dysplasia of the nodal arteries made us consider the case number 10 as pathological although he was a middle aged male (42/M).

Case number 13 was a young female (18/F). She was found to have serious pathological findings of her SA node as third degree fibrosis, second degree adiposis and third degree fibromuscular dysplasia (Figure 1,2,3,4). Serious fibroadipose changes and fibromuscular dysplasia of the SA node of the case number 18, made us consider this case as also a pathological one since he was very young in age (17/M).

## DISCUSSION

Cardiac conduction tissue pathologies are very interesting to discuss for most of the cardiac pathologists as well as forensic pathologists. Some of the authors speculate that dissecting and evaluating the conduction tissue in every SUD autopsy case is not a necessity and neither can be a help to understand and evoke the mechanism and cause of the death sufficiently. However, most of the cardiac pathologists claim vice versa. Those pathologists think and speculate that eva-

luating cardiac conduction tissue will be helpful in understanding and evoking the mechanism and cause of deaths for SUD cases, as well as, increasing the quality of the autopsy standards (2,13-16).

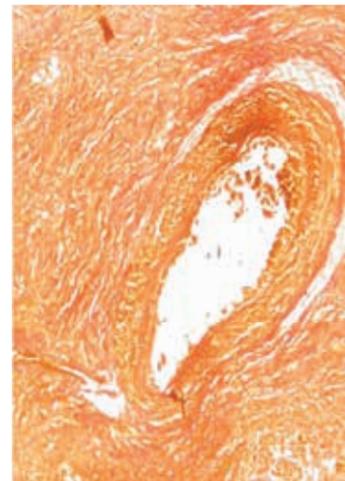
The cardiac conduction tissue is a hard to demonstrate region of the heart, yet, Michaud et al, demonstrated 87% of the AV node of 110 forensic autopsy cases while Ridolfi et al could demonstrate the SA node in 22 cardiac amiloidosis cases of the total 23. It is well known that SA node is easier to demonstrate than the AV node in the literature. We could be able to demonstrate the SA nodes in all cases and control group while AV node was very hard to demonstrate and in some cases only a part of it could be found near by the edge of the HIS component. It is probably because of AV node's being located near by the central fibrous component (14).

We found that 77.7% (21) of all 27 cases, the causes of death were due to coronary atherosclerosis, ischemia of the myocytes and old or fresh infarctions while in 40.7% (11) of the cases there were significant pathologic changes of the conduction tissue, however only in 11.1% (3) of the deaths were correlated directly with the conduction system. The cause of death for 3 cases remained as unknown. 21 cases were having left ventricle pathologies. Cardiac conduction system pathology was found in 1 case which was having also an old infarction. Although many of the similar studies show parallel results with our study, Song et al detected serious pathologies of the conduc-

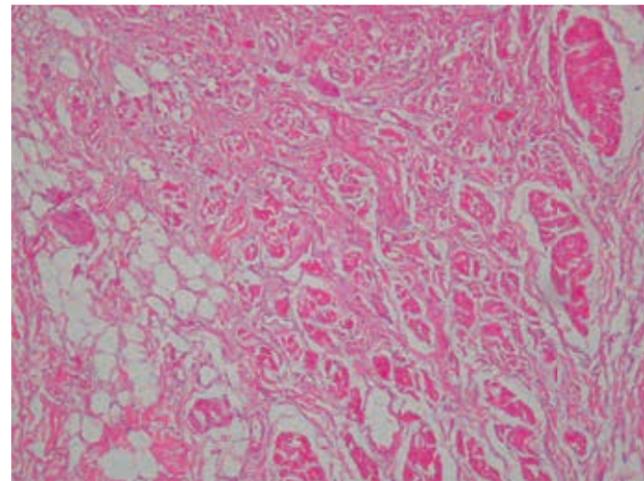
tion system such as fibrosis in their 150 forensic autopsy cases (16). In 110 of their cases, they showed the lesions in SA and AV nodes respectively in 20.9% and 8% of the cases. On the other hand; Charlton and Williams found same pathologies in SA and AV nodes with 23% while in Cohle et al's serial, 2.9% of all 381 SUD cases, cardiac conduction tissue pathologies were the main cause of death (2,13). The difference in between those series

can be explained by the difference of the countries, socio-cultural characteristics, life conditions, environment, nutrition and genetic variations. Our previous studies on various cardiac pathologies had also revealed differences within non Mediterranean countries while almost exactly similar results had been obtained within Mediterranean countries such as Italy, France and Greece (2,13,14,17-27).

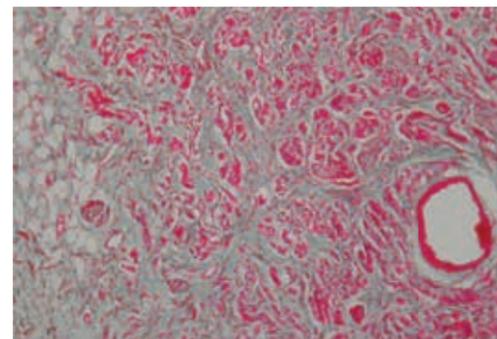
The arteries of the SA and AV nodes may also contain pathological changes and those may also cause SUD due to the ischemia of those areas. Fibromuscular dysplasia is the one of the major pathology for those arteries. We found fibromuscular dysplasia in 2 of the SA node arteries (7.4%) and 4 of the AV node arteries (14.8%). Our results showed similarities with Cohle et al, and Burke et al's studies while they were slightly higher than Charlton and Williams' studies



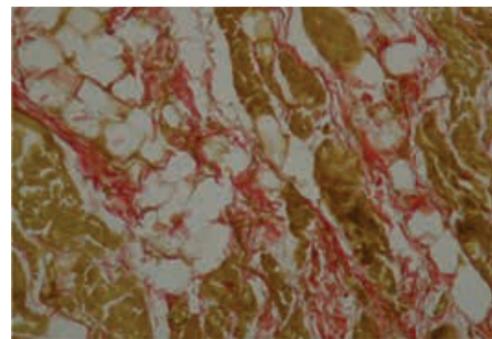
**Figure 1:** Elastic Van Gieson, showing the fibrodisplastic changes of the SA node artery



**Figure 2:** H+E, showing the SA node and fibro-adipose involvement



**Figure 3:** Masson's Trichrome, showing the SA node and fibro-adipose involvement, and also notice the artery.



**Figure 4:** Elastic Van Gieson, showing the AV node and fibro-adipose involvement.

**Table 3:** SA and AV node findings with H+E, Masson's Trichrome, Elastic Von and Crystal Violet staining methods in pathologic cases.

Case nr/ age-sex	Fibrosis SA / AV	Fatty changes SA / AV	Fibromuscular dysplasia SA / AV	Inflamation SA / AV	Bleeding SA / AV	Amiloidosis SA / AV
7/17M	++ / +	++ / +	++ / ++	- / -	- / -	- / -
10/42M	++ / ++	+++ / ++	+++ / +++	- / +	- / -	- / -
13/18F	+++ / ++	++ / +	+++ / +	+/-	+ / -	- / -
15/22M	- / ++	- / ++	- / ++	+ / -	- / -	- / -
16/30M	++ / ++	++ / +	+ / ++	+ / -	- / -	- / -
18/17M	+++ / +	+++ / -	++ / ++	+ / +	- / -	- / -
19/60M	++ / ++	++ / +++	++ / ++	++ / +	+ / -	- / -
22/18M	- / ++	- / ++	- / ++	- / +	+ / +	- / -
26/38M	+ / ++	+ / ++	- / +++	++ / -	+ / +	- / -
27/46M	+++ / -	++ / -	++ / ++	++ / +	+ / -	- / -
CONTROL CASES						
1 / 14F	- / -	- / -	- / -	- / -	- / -	- / -
2 / 62M	+ / +	+ / +	+ / ++	- / -	- / -	- / -
3 / 44M	++ / -	+ / -	+ / -	- / -	- / -	- / -
4 / 65M	++ / ++	+ / +	++ / ++	- / -	- / -	- / -

**Grade 0 (-):** No significant fibrosis, no adipose tissue infiltration, no significant luminal obliteration of the arteries, no mono nuclear cellular accumulation.

**Grade I (+):** Irregular widening of the myofibrils or small amounts of fibrosis, adipose tissue infiltration less than 25%, random mono nuclear cellular accumulation.

**Grade II (++):** Small amounts of irregular fibrous tissues and irregular myofibrils, 25-50% of adipose tissue infiltration, 25-50% luminal obliteration of the arteries, accumulations of mono nuclear cellular infiltration.

**Grade III (+++):** Wide fibrous tissue proliferation, adipose tissue infiltration more than 50%, luminal obliteration of the arteries over 75%

[2,13,28]. We think that it may be a result of the differences between the regional dietary habits as well as the higher average age in our study [2,12-14,28]. Mononuclear cellular infiltration (MNCI) is also a significant pathological change in the nodes. We found MNCI in 66.6% of all our cases. This is relatively higher than Charlton and Williams' study, which we again consider the environmental factors for the higher existence of MNCI, while it's similar to some other studies [12,13,18,29]. Hypoxic changes of the myocardium should also be evaluated during the evaluation of the conduction system pathologies. On the other hand, although it is easy to understand and explain the cause of death and mechanism, it is efficient to evaluate also the conduction system. Acute and chronic coronary artery diseases affect the conduction system tissues, yet the ratio of what is affected is not very well described and still the discussions go on. Some of the studies show that, conduction system tissue may not show any pathologic involvement in acute myocardial ischemia cases. These authors explain this situation with the resistance of the conduction tissue against ischemic necrosis as a result of its increased glycogen deposit and also its decreased oxygen use. We found myocardial infarction in four of the SA nodes and two of the AV nodes of all cases. These findings are similar with the other studies. We suggest that this is an important finding to understand and reveal the conduction system effects on the early and late myocardial infarctions [4,13,30-34].

Histological findings of the conduction system can be demonstrated in

patients with heart blocks however we can't comment on this finding due to the lack of our data on the clinical conditions and anamneses of the cases [21,35-38].

Conduction system fibrosis of types II and III may result from immune-mediated destruction of the conduction tissues in infants of mothers with connective tissue diseases such as systemic lupus erythematosus or Sjögren syndrome [39]. Such infants may die suddenly from reentrant tachyarrhythmias under circumstances simulating sudden infant death syndrome. Although routine examination of the conduction system is probably not warranted in cases of apparent sudden infant death syndrome, such an examination should be considered in the death case of an infant whose mother was having an immune-mediated connective tissue disease.

Acquired with age, the summit of the muscular ventricular septum becomes progressively fibrotic. Although some degrees of fibrosis are normally seen in nearly all individuals, this normal process is accelerated in some individuals and may be associated with focal fibrous interruptions of the bifurcating bundle and proximal bundle branches (Lev disease). Other patterns of fibrosis are also detected, including diffuse fibrosis of the mid and distal portions of the bundle branches (Lenegre disease) and fibrosis of the proximal left and distal right bundle branches (intermediate form of idiopathic fibrosis) [38,40].

Lie et al points out that cardiac conduction tissue involvement is

not very often in amyloidosis cases. Smith et al found out that SA node is the most frequently affected one in cardiac conduction tissue involvement cases of amyloidosis. Riboldi et al studied on 23 amyloidosis cases. We have not found any amyloidosis case in our serial, yet still, amyloidosis should always be taken into consideration [2,41].

Cardiac tumors are very occasional and we don't have any cardiac tumoral case [2,13].

In the control cases, we noticed that the fibro-adipose changes of the nodes and fibrodysplastic changes of the nodal arteries and also some other pathological changes were slightly less frequent when compared to our SUD cases. We think that the higher and more serious pathological changes of the cardiac conduction tissue in SUD study cases then the control cases may lead us to conclude that these findings may be considered as the cause of death pathologies in some cases of our SUD serial such as similar ones [2].

SUDs are also often during stress and/or physical activities. In elongated QT values of the ECG or Brugada syndrome cases, the pathology is not morphologically functional. Yet, still in some cardiac rhythm disturbance cases, some may detect cardiac conduction tissue pathologies histologically. We believe that the relationship between cardiac conduction tissue morphological pathologies and cardiac rhythm disturbances will only be demonstrated clearly by clinicopathologic evaluations within large

serial studies [14,21,42-44].

We must learn and understand the conduction system anatomy and its pathology better. The lack of experience in the examination techniques is the real reason for this reluctance of missing the conduction system pathologies. It is not surprising because many anatomic pathology and forensic medicine residents complete their training without learning about the cardiac conduction system. This lack of training is a result of the ongoing decline of the autopsy practice as a teaching tool worldwide. Careful case selection for conduction system analysis together with a sensible approach for dissection and histologic sampling will result in an increased yield of diagnostically specific, potentially lethal lesions found through only a minimal increase in the expenditure of time or money [9,10].

It is well seen that cardiac conduction tissue pathologies are very important for understanding the causes and mechanisms of the SUD cases. In cases number 7, 13, 16, 18, we found out serious cardiac conduction tissue pathologies. We correlated the cause and mechanism of those young age deaths with the conduction tissue pathologies in our study. In various studies of the literature, it has been noticed that conduction tissue pathologies are often in the young age SUD cases. Thus, it is very important and critical to understand and also evaluate the mechanism and the cause of young age SUD cases [2,13,16,26,27,46].

In conclusion, we must and will study the cardiac conduction tissue with larger SUD series, and try to understand the pathologies-mechanisms of deaths in especially young SUD cases in our region and in our country. We think that our findings in this model study are important for demonstration of the young SUD cases and their relation with the conduction tissue pathologies. Therefore, we conclude that examination of the cardiac conduction system can be a very useful adjunct to the examination of the heart in cases of sudden cardiac death especially in our region. Careful case selection, proper technique, and mindfulness for nonspecific findings or normal variants may increase the likelihood of identifying abnormalities that may be the morphologic basis of sudden cardiac death.

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