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Sudden cardiac death and thymic hyperplasia in adults: myth or reality? A case report

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Abstract

Background Sudden cardiac death is a major public health concern. The incidence of sudden cardiac death in young adults remains unclear and is generally underestimated.

Case presentation This is a case report of a 24-year-old man with a silent pathological history, who suddenly collapsed in a restaurant during dinner with his workmates. Autopsy and ancillary examination revealed focal atrioventricular node fibrosis and thymic hyperplasia.

Conclusions Thymic hyperplasia has been considered a cause of sudden death in the past century. The mode of death was explained through nosographic entities such as status lymphaticus or thymic asthma, which are currently consigned to history books. Nevertheless, recent studies have sought to determine the relationship between thymic hyperplasia and sudden unexpected deaths in adults. Moreover, isolated fibrosis of the atrioventricular node is a rare condition that can remain undiagnosed even after a full autopsy. This report aims to provide a concise review of the existing literature concerning sudden cardiac death and discuss the so-called "thymic death" theory, which is now considered a myth. Was the finding of thymic hyperplasia and atrioventricular node fibrosis a coincidence? Should the myth surrounding "thymic death" be re-examined?

Keywords Sudden cardiac death, Atrioventricular node fibrosis, Heart dissection, Thymic death, Forensic Pathology, Forensic Medicine

Background

Sudden cardiac death (SCD) is an unexpected natural death due to cardiovascular disease within a short time (generally $\leq$ 1 h) after the onset of symptoms in a person without any prior condition that would appear fatal (Zipes and Wellens 1998). A prospective study in Italy reported a cumulative annual incidence of SCD of approximately 1 per 100,000 young individuals (Corrado et al. 2006; Fudge et al. 2014). The most common mechanism of SCD is abrupt ventricular fibrillation due to underlying asymptomatic cardiovascular disease (Harmon et al. 2015). The majority of SCDs in older subjects are due to atherosclerotic coronary artery disease (Basso et al. 2008; Corrado

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and Zorzi 2018). The prevalent causes of SCD in young adults are as follows (Wilhelm et al. 2015).

- Cardiomyopathies accounted for 9% of SCD victims. The most common type is hypertrophic cardiomyopathy, (Holst et al. 2010) followed by the myocardial bridge, (Donohoe et al. 2010) and arrhythmogenic cardiomyopathy (Deasy et al. 2010; Harmon et al. 2011; Winkel et al. 2011).
- Premature coronary atherosclerosis (Green and Sheppard 2016).
- Anomalous origin of the coronary artery from the wrong coronary sinus, a congenital malformation with a silent clinical course (Basso et al. 2000).
- Mitral valve prolapse, with an estimated prevalence of 2–3% in the general population, explains SCD in 8% of the cases (Marra et al. 2016; Thiene 2018).
- Aortopathy in the setting of the bicuspid aortic valve with aortic dissection and rupture accounted for 3% of the SDC cases (Basso et al. 1995).
- Myocarditis can provide an electrical substrate for life-threatening ventricular arrhythmias and SDC (Zorzi et al. 2016).
- Diseases of the conduction system, with abnormal propagation of the electrical impulse, account for 6% of SCD cases in the young population. The most frequent culprit disease is Wolff–Parkinson–White syndrome, with a prevalence of 1:1000 individuals with ECG pre-excitation patterns in the general population (Basso et al. 2001).
- Primary arrhythmogenic disorders (the congenital long-QT syndrome, the Brugada syndrome, and catecholaminergic polymorphic ventricular tachycardia) (Bagnall et al. 2016).

Moreover, many genetic mutations predispose to SDC but the underlying disease pathways have not been completely understood (Bezzina et al. 2015).

Despite a detailed autopsy and histopathological examination of the heart, 6-53% of cases of SCD cases show no morphological heart alterations at autopsy (Papadakis et al. 2009). Probably pathogenetic genetic variants have been observed in up to one-third of unexplained sudden cardiac death (Bagnall et al. 2016).

This report describes a case of sudden cardiac death in a healthy young man who underwent a deep cardiological examination ten days before death. The autopsy revealed thymic hyperplasia, which many authors considered to be a possible cause of SCD during the first half of the nineteenth century (Greenwood and Woods 1927). This report aims to provide a concise review of the existing literature concerning sudden cardiac death and to discuss the so-called "thymic death" theory, which is now considered a myth.

Case presentation

A 24-year-old man who worked as a laborer suddenly collapsed during dinner with workmates. The patient died after several attempts at cardiopulmonary resuscitation. The victim had a healthy lifestyle, played football regularly, and had no history of diseases or alcohol and drug abuse.

Due to the occasional finding of self-measured elevated blood pressure nearly ten days before death, the patient underwent precautionary cardiovascular examinations. Echocardiography revealed normal findings, while the electrocardiogram showed an incomplete right bundle branch block, which was considered non-pathological (Floria et al. 2021) (Fig. 1).

The 24-h ambulatory blood pressure monitoring did not show any pathological findings. According to the cardiologist's conclusions, there was no need for further examination.

No cutaneous swelling, upper airway edema, or hyperinflation of the lungs with mucus plugging was observed, and quantification of specific immunoglobulins for allergens and macrophage tryptase was negative. Hence, the anaphylactic shock was excluded.

The heart weighed 360 g and had normal morphology. The atrial septum was normal and the mitral and tricuspid valves, papillary muscles, and chordae tendineae were intact. The thicknesses of the walls of the cardiac chambers and chambers were normal. The coronary arteries showed no anomalous origin or atherosclerosis and appeared open throughout the course.

The wall of the right atrium showed a whitish area near the anterior tip of Koch's triangle (Fig. 2).

Dissection of the conduction system was performed after the examination of the coronary arteries and evaluation of the myocardium. The dissection was performed in two steps: (1) a myocardial sample was taken near the lateral edge of the ostium of the superior vena cava along the posterior edge of the terminal crest, which lies at the junction of the smooth posterior and muscular anterior walls of the right atrium (sinus node), and (2) another tissue block was taken near the anterior tip of Koch's triangle, including the apical septal myocardium. The slides were stained with hematoxylin, eosin, and Masson's trichrome. Histological examination revealed extensive fibrotic replacement of the atrioventricular node, the remnant of which was still evident with a tiny lymphocytic infiltrate in the atrial approach to the node. Inflammation

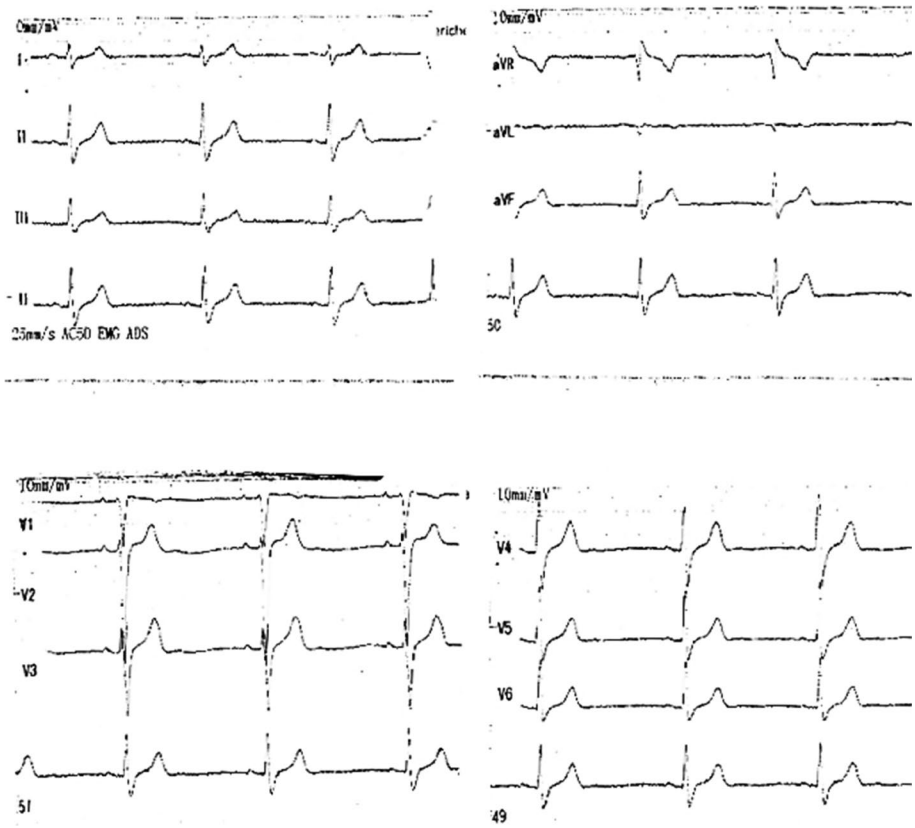

Fig. 1 Electrocardiogram of the victim, performed 10 days before the death

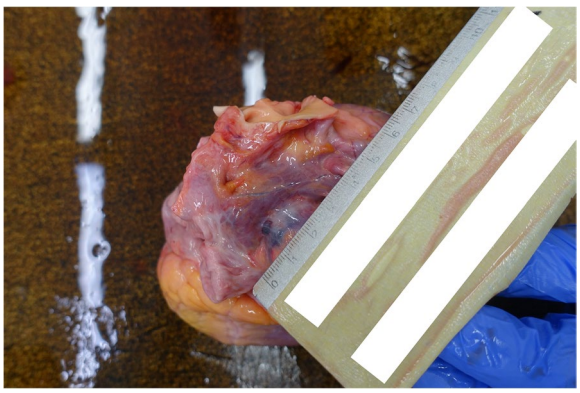

Fig. 2 The whitish area on the right atrium wall, localized near the anterior tip of Koch's triangle

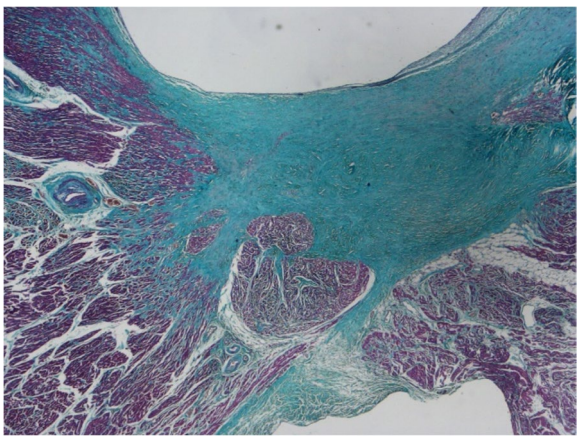

Fig. 3 Sample of the atrioventricular node stained with Masson's Trichrome method (× 40 magnification)

was not observed in any of the ventricular myocardium sections examined (Figs. 3 and 4).

The other organs did not exhibit any macroscopic or histological alterations except for the thymus, which occupied most of the anterior mediastinum. The authors found a weight of 52 g and dimensions of 13 cm along the transverse axis. Histological examination of the thymus revealed physiological aspects of the gland (Fig. 5).

Toxicological examination results were negative for cocaine, cannabinoids, opioids, benzodiazepines, barbiturates, amphetamines, methamphetamines, and

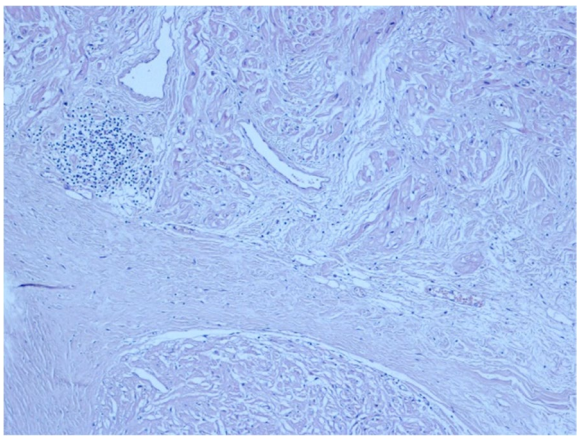

Fig. 4 Sample of atrioventricular node stained with hematoxylin-eosin (× 100 magnification).

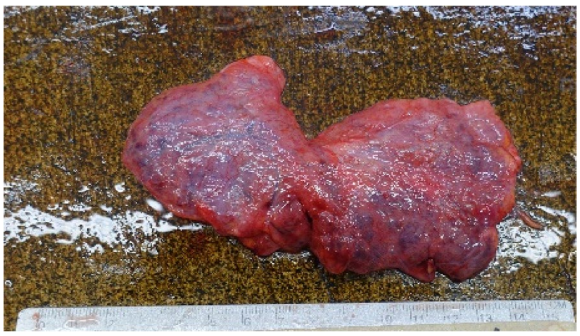

Fig. 5 The 13-cm-long thymus after dissection

tricyclic antidepressants. The blood alcohol concentration was 0.53 g/L.

According to autopsy findings and ancillary examination results, the cause of death was ruled to be arrhythmia resulting from atrioventricular node fibrosis.

Conclusions

Owing to the absence of any other macroscopic, histological, or toxicological findings, the authors assumed that the cause of death in the young man was a cardiac conduction disturbance arising from atrioventricular node fibrosis.

Many cases of SCD in young people with noncontributory medical histories normally represent a matter of concern in forensic pathology. All SCD autopsies should have a structured examination to specifically address the major causes of extra-cardiac and cardiac sudden death (Byard 2010).

In the present case, a careful cardiac examination was performed, adhering to the recommendations on Medico-Legal Autopsy Rules produced by the Committee of Ministers of the Council of Europe (Brinkmann 1999). The histological examination of the heart conduction system revealed atrioventricular node fibrosis with lymphocytes of the interventricular septum.

Atrioventricular node fibrosis can be congenital or acquired and is considered an exceptional condition (Cohle and Lie 1991). Congenital fibrosis causes a congenital heart block, whereas acquired fibrosis develops with age (McCormack and Barth 1985; Olson and Lindsley 1987). Although some degree of fibrosis is normally observed in nearly all individuals, this process is accelerated in some individuals and causes focal fibrous interruptions of the bifurcating bundle and proximal bundle branches (Lev disease), 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 (an intermediate form of idiopathic fibrosis) (Suárez-Peñaranda et al. 2006). These examples of primary fibrosis of the conducting system in the absence of structural heart disease are associated with conduction abnormalities, symptomatic atrioventricular blocks, and sudden cardiac death (Virmani 2001).

Fibrous interruption may also follow myocarditis of the conduction tissues (Blanck and Akhtar 1993). In rare cases, myocarditis may be localized to the area of the atrioventricular node, resulting in progressive atrioventricular nodal block and SCD (Hackel 1986). Primary fibrosis of the conducting system in the absence of structural heart disease, primary fibrosis of the conducting system is commonly associated with conduction abnormalities, symptomatic atrioventricular block, and less commonly, sudden cardiac death. Histologically, there is diffuse scarring of the atrioventricular node and bundle branches, often with sporadic lymphocytes, sometimes resulting in atrial-axis discontinuity (Virmani 2001). In the present case, lymphocytes in the interventricular septum may support the hypothesis of post-inflammatory origin. Regarding myocarditis of the conduction system, bundle branches are most commonly affected, usually by contiguous spread from the ventricular septum, although the sinus and atrioventricular nodes may also be involved (Gulino 2003).

Autopsy also revealed that the thymus weighed 52 g and was 13 cm in length. Typically, the thymus weighs 12–15 g at birth, 30–40 g during puberty, and 10–15 g at 60 years of age. Histologically, the thymus did not show any alterations or signs of thymoma or other thymic diseases. Hence, an increase in the weight and size of the histologically preserved thymus defines a true thymic hyperplasia (Khan MA and Anjum F., 2021). Such evidence prompted the authors to discuss the theory of the

so-called "thymic death", which was expressed at the end of the nineteenth century by several authors.

In 1830, German physician Kopp stated that a hyperplastic thymus might lead to tracheal obstruction and cause sudden death in infants (Thursfield 1929). In 1889, Paltauf described "thymic death" as a systemic disorder with enlargement of all lymphoid tissues, which might lead to sudden death by cardiovascular collapse (Greenwood and Woods 1927). The theory of "status lymphatic-chlorotic," or "thymicus-lymphaticus," was popular for over 30 years and sustained by over 800 publications: during the early twentieth century the registration of death causes became progressively mandatory all over the world, and sudden infant deaths were often blamed on "status lymphaticus." (Dally 1997). In 1942, an Italian contribution to thymus studies was given by Pende, who theorized the syndrome also known as "constitutional hyperthymic syndrome" or "Pende's syndrome." This condition is characterized by an increased volume and activity of the thymus since birth, macrosomia, and scarce genital development during puberty (Sellina and Negro 1942). In 1927, Greenwood sustained that, as the thymus is involved within a few days of illness, it is large only if the patient died suddenly (Greenwood and Woods 1927). Finally, in 1931, The Lancet published an editorial entitled 'The End of Status Lymphaticus,' which supported Greenwood's theories (Young and Turnbull 2005). Hence, the pathological entities of Kopp's asthma, the so-called "thymic death, and the status thymicus-lymphaticus are now entities consigned to history books. In 2000, Rackham included the "thymic myth" into "historical medical mistakes." According to Rackham, the cadavers studied by anatomists to define the "normal" thymus size were those of the poor people, who generally died for highly stressful chronic illnesses (e.g., tuberculosis, infective diseases) and malnutrition which cause stress-related thymic involution: so they simply underestimated the size and weight of "normal" thymus gland. Consequently, infants and young adults who died suddenly were erroneously believed to have thymic enlargement (Rackham 2000; Cowan et al. 2020).

Nevertheless, nowadays the "thymic death" theory is reviving thanks to several studies. In 1980, Kendall et al. found an association between cardiovascular disease-related deaths and higher thymic weight (Kendall et al. 1980). Burke and Declich in 1996 described patients with atrioventricular node tumors, hyperplasia of the islets of Langerhans, multicystic ovaries, adrenal heterotopia, clear cell adenomatosis of the right kidney, and thymic hyperplasia, suggesting a unique underlying genetic defect (Burke et al. 1993; Declich et al. 1996). Finally, in 2017, Zou et al. reviewed adult postmortems from 1984 to 2014 and found that thymic hyperplasia significantly increased the risk of sudden unexpected death in young adults in both males and females, predominantly through cardiovascular disease (Zou et al. 2017). However, these observations remain unexplained and further research is required to clarify this issue.

The present case permitted the authors to perform groundwork for increased attention to the thymus and heart in future cases of sudden death. Whether and how the relationship between sudden death and thymus hypertrophy exists is a matter of further investigation.

Abbreviation

SCD Sudden cardiac death

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SN and MB contributed to the writing—original draft. MB and DF contributed to the writing—review and editing. MMC and AM contributed to the methodology and investigation. BS supervise this work. All authors read and approved the final manuscript.

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