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Review / Derleme

The role of eustachian valve and patent foramen ovale on sudden death

[Östakien valf ve patent foramen ovale'nin ani ölüm üzerine etkisi]

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Abstract

Sudden unexpected cardiac death is the leading cause of death in industrialized countries. Patent foramen ovale and eustachian valve are two of cardiac diseases and they may be associated with clinical disorders as embolism, stroke, plathypnea–orthodeoxia syndrome, carcinoid heart disease, atrial flutter and endocarditis.In this study, it is aimed to review the literature for roles of patent foramen ovale and eustachian valve on the causes of sudden deaths.

Keywords:

Patent foramen ovale, eustachian valve, sudden death.

Özet

Ani beklenmedik kardiak ölüm endüstriyel ülkelerdeki başlıca ölüm sebebidir. Patent foramen ovale ve östakian valf, embolizm, strok, platipnö-ortopnö sendromu, kardiak kalp hastalığı, atrial flatter ve endokardit gibi klinik rahatsızlıklara neden olabilen iki kalp hastalığıdır.Bu çalışmada, patent foramen ovale ve östakian valfin ani ölüm sebebi üzerindeki rolünün belirlenmesine yönelik literatür araştırması amaçlanmıştır.

Anahtar Kelimeler:

Patent foramen ovale, östakian valf, ani ölüm.

1. Introduction

Sudden Death is a general term used for many different types of death. Sudden death is non-traumatic, non-violent, unexpected occurrences and it results from cardiac and/or respiratory arrest within maximum 24 hours of previously witnessed normal health [1]. Sudden unexpected cardiac death is the leading cause of death in industrialized countries [2].

A patent foramen ovale (PFO) develops when fibrous adhesions fail to seal the atrial septum after birth, allowing the persistence of a potential shunt between the right and left atria of the heart [3], however, normal higher pressure in the left atrium keeps the foramen ovale functionally closed [4] (figure-1 [5]). This is a common finding in the general population and PFO occurs in about 25% of normal healthy adults [6]. Hagen et al described that the incidence of the PFO was 27.3% in 965 autopsy specimens of human hearts, but it progressively declined with increasing age from 34.3% during the first three decades of life to 25.4% during the 4th through 8th decades and to 20.2% during the 9th and 10th decades [7]. Patent foramen ovale is detected in 10-15% of the population by contrast transthoracic echocardiography. The advantage of the directly visualization of PFO on autopsy studies was shown as the probably cause of this difference between two rates [8].

EV is described by Bartolomeo Eustachio (Italian anatomist) at 1552 [9]. It is an embryological remnant of the right sinus venosus valves that oxygen-rich blood flow in the fetal circulation from the vena cava through the foramen ovale into the left atrium and away from the tricuspid valve [10-13]. After the closure of the foramen ovale, it does not have a specific function (figure-2 [5]). In echocardiographic examinations, the EV may appear as a thin flap originating from the orifice of the inferior vena cava, or it may be totally absent [12]. EV sometimes may appear as a rigid, elongated structure in the inferior portion of the right atrium or as a membranous, undulating, echogenic mass within the right atrial cavity [13]. Strotmann et al described that

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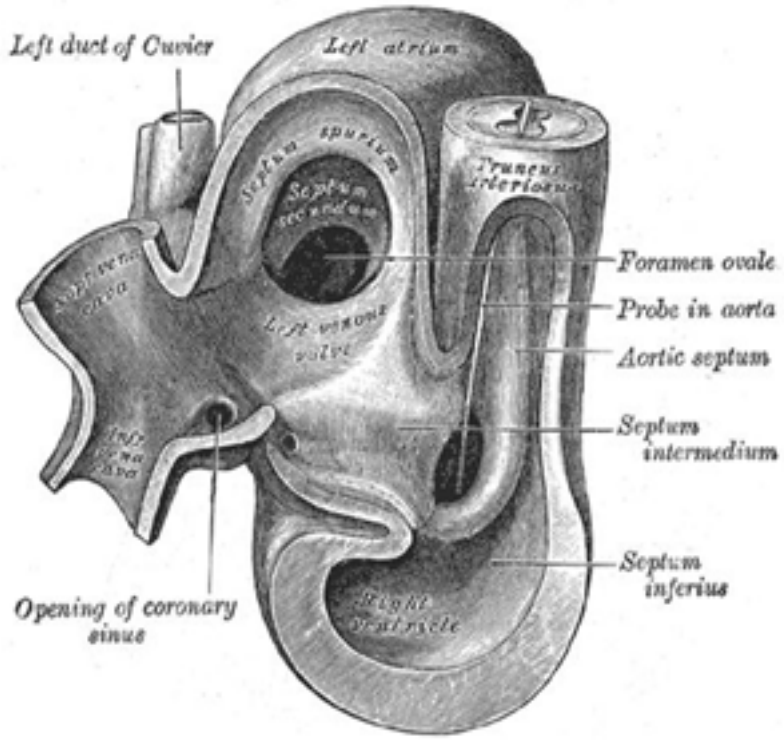
there were EV in approximately 60% (in a group of 120 consecutive necropsies) in adults [14], while Wong et al reported that the persistence of this valve in an adult is uncommon [13]. In echocardiographic study of Limacher et al, EV was identified in 82% of the children with normal heath, in 59% of children with congenital heart disease and in 70% of all children. Their height ranged from 2 mm to 20 mm (mean 5.4±3.6 mm). Although the EV was less likely to be visualed in older children, it was larger in these children when present. According to the paper above, Yater had identified EV in 86% of 120 hearts in an autopsy series at 1929 and had described an average height of 3.6 mm with a range of 1.5 mm to 23 mm [15]. Yavuz et al described an unusual giant eustachian valve with an echocardiographic appearance of a septal structure dividing the right atrium into 2 separate chambers [12]. Schneider et al Persisting EV defined in up to 70% of patients with PFO [16].

Figure-1. *Foramen ovale from Gray H [7]*



Although patent foramen ovale (PFO) is not a cause of death alone, death can occur when complications accompany it [8, 17]. And EV has not been reported adequately to forensic medicine literature.

In this review, we aimed to invest the pathogenesis of both of PFO and EV, and their roles for cardiac sudden deaths

2. Pathogenesis of patent foramen ovale:

In the recently reports, it is defined that PFO is associated with cryptogenic stroke and migraine. Henrich et al showed at 1986 that migraine is associated with PFO and atrial septal aneurysm in patients with ischemic stroke [18]. Anzola et al described that the prevalence of PFO was 48% in patient with migraine with aura and 20% in patients with migraine without aura, and claimed that the increased risk of stroke found in epidemiologic studies in patients with migraine with aura may be explained by an increased predisposition to paradoxical cerebral embolism [19]. In a prospective cryptogenic stroke study, 46% of patients had a PFO and migraine was present in 27.3% of patients with PFO. Migraine was present in 36% of patients with PFO and stroke, while only 16% of stroke patients without PFO had migraine [20]. Paradoxical embolism through a patent foramen ovale may be responsible for stroke more often than is usually suspected in older and young adults in other studies [21, 22]. In patients affected by this cardiac disease, a possible cause of ischemic stroke may be an increased propensity to paradoxical cerebral embolism during migraine attacks, when there is a condition of platelet hyperaggregation. Moreover, in small uncontrolled series, it has been observed that percutaneous closure of PFO can reduce the risk of ischemic stroke and the frequency of migraine attacks [23], suggesting that migraine may be a symptom of cardio-embolism [24]. Paradoxical embolism may occur in patients with acute pulmonary thrombo-embolism, when PFO coexists with pulmonary hypertension (right-left shunt) [25]. The most common intracardiac defect associated with paradoxical embolism is PFO [26]. Also

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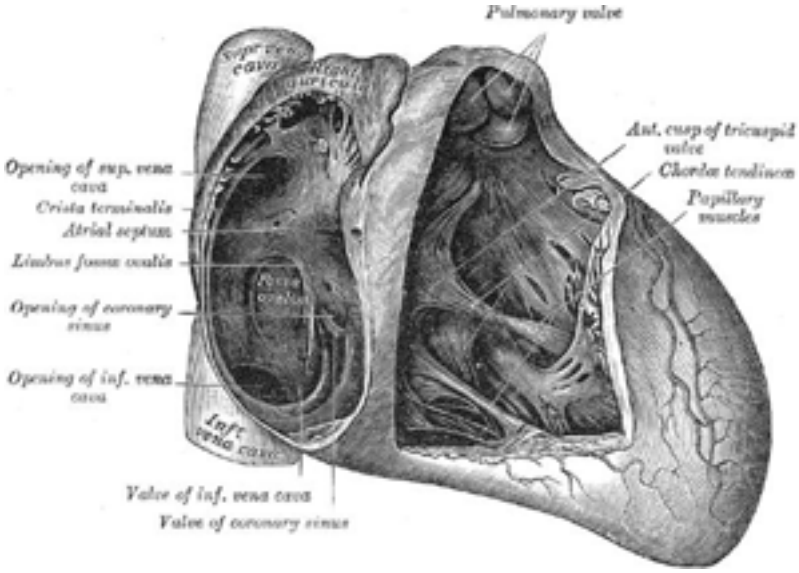
PFO is an independent predictor of major adverse events in patients with pulmonary embolism [27].

Figure-2. *Eustachian valve (=valve of inferior vena cava=) from Gray H [7]*



PFO was reported to be a common cause of plathypnea–orthodeoxia syndrome [28-34]. Plathypnea is defined by dyspnea induced by an upright position and relieved by recumbency. Orthodeoxia is defined as arterial deoxygenation increased by an upright position and improved by recumbency [34, 35]. Platypnea–orthodeoxia syndrome is an uncommon clinical disorder associated with conditions such as pneumonectomy [33, 36], lobectomy [37], pulmonary emphysema [33], liver cirrhosis [33] and ascending aortic aneurysm [38]. PFO can contribute to hypoxemia in patients with obstructive sleep apnea [39].

PFO has also been associated with decompression sickness and paradoxical gas embolism in divers [23, 40-43]. Although the term decompression illness is used classically for describing decompression disorders with gas bubbles as the initiator [40], an alternative pathway for venous gas embolism to be transferred to the systemic arterial side consists of an intra-atrial shunt due to an atrial septal defect or a PFO [41].

Also air embolism may occur during neurosurgical operations in the sitting or semi-sitting position [44-49] or tracheal extubation in patients with PFO [50].

Although recent case reports have clearly shown that paradoxical brain fat embolism can occur in the setting of long bone fractures and joint arthroplasties in patients who have PFO [51-53], Colonna et al, suggests that large numbers of fat emboli are able to rapidly traverse the pulmonary circulation (in less than 4 h) in the setting of total hip arthroplasty, even in the absence of a PFO [54].

PFO worsens the prognosis in patients with carcinoid heart disease [55, 56] Carcinoid heart disease is an uncommon and complex form of valvular heart disease that may affect left-sided and right sided valves. Cardiac involvement is a major cause of morbidity and mortality [57].

Paradoxical embolism is an important cause of sudden death and it may occur from paradoxical gas embolism, air embolism and brain fat embolism [58]. The presence of PFO is an important predictor of adverse outcome; according to Goldhaber [59], patients with PFO have a nine-fold higher mortality rate than those without it. Also, Konstantinudes et al demonstrated in a prospective study that echocardiographic detection of PFO signifies a particularly high risk of morbidity and mortality; the death rate increased from 14% to 33%, the ischemic stroke rate increased from 2.2% to 13%, and the risk of peripheral arterial embolism increased from 0% to 15% [27].

Even if it wouldn't be responsible for death, it had to be known by forensic scientists because of the association of PFO with transient global amnesia [18, 60].

3. Pathogenesis of eustachian valve:

EV is named as valvula venae cavae inferioris, caval valve, and sylvian valve too [61]. Strootmann et al discussed the potential role of the EV in the genesis of the stroke [16] and in other some studies, it is defined that, EV or ridge which as one of right atrial endocardial structures were proven to support the atrial flutter reentrant circuit [62-64].

Prominence of EV in adult life is reported to cause a significant right-to-left shunt in the presence of interatrial communication by altering the blood flow pattern [65, 66]. Filamentous strands in right atrium which can span EV with atrial wall are also a remnant of right valve of sinus venosus. When an extensive network of strands is present, it is known as Chiari network,

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which has been associated with paradoxical embolization presumably by directing inferior vena cava flow towards the fossa ovalis area [67]. It was hypothesized that atrial septal aneurysm is associated with large PFO and that prominent EV or right atrial filamentous strands are more frequently found in patients with atrial septal aneurysm. The combination of a large PFO and a structure in right atrium diverting inferior vena cava flow toward the PFO would likely lead to an increased risk of paradoxical embolisation [10].

Nakagawa et al emphasized that, EV behaves as an anatomical line of conduction block, forcing the reentrant impulse, emerging from the inferior isthmus, to travel upwards through a second narrower isthmus between the tricuspid annulus and the coronary sinus ostium (septal isthmus) [63].

EV endocarditis is a syndrome characterized by clinical signs and symptoms of right-sided infective endocarditis in association with infective vegetation on the EV and EV endocarditis usually occurs without associated involvement of any other cardiac valve. In a study conducted by Sawhney et al, EV was found the sole focus of intracardiac infection in 80% of cases [68]. San Roman et al emphasized that EV endocarditis might be more frequent than it was believed [69]. In a case with acute staphylococcal pneumonia, sepsis occurred in spite of the application of appropriate antibiotics and the patient eventually died. At necropsy of his corpse, endocarditis of the EV was found [70]. The common microbial agent responsible to EV endocarditis is Staphylococcus aureus [68, 70-72] However, EV endocarditis due to Escherichia coli [68], Proteus vulgaris [68], Klebsiella pneumonia [13] and Enterobacter cloacae [73] had been described.

Additionally, Bowers et al described an infective endocarditis involving the vestigial eustachian valve in an injection drug user [74] and Nkomo and Miller reported the solitary cyst attached to the EV in a case [75].

4. Conclusion

In developed countries where health controls are applied regularly, the outcome of these disorders may not be fatal but health controls are not usually regular in developing or undeveloped countries, and persons confront to sudden death when they ignore symptoms.

Even if PFO and EV are not a cause of death alone, it can not be ignored that the clinical disorders (i.e. stroke, fat embolism, gas embolism) accompanied to them may be a cause of sudden death.

Finally, the careful examinations applied by medical examiners will elucidate the presence or absence of PFO or EV at medico-legal autopsies. Thus, the real cause can be explained in deaths associated with PFO or EV. It will be important to invest the role of these acts if there are claims the malpractice or trauma.

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