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Case report

Soccer related sudden deaths in Turkey

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Abstract
Regular physical exercise is recommended by the medical community, because it offers the potential to reduce the incidence of coronary events. On the other hand, vigorous exertion may act as a trigger of acute myocardial infarction and sudden cardiac death in susceptible individuals. Death during sports activities differs among sports disciplines and countries. In Turkey, soccer attracts more spectators than any other sports activity and the attention of the press and media, and is preferred over other sports by many young and middle-aged individuals. As autopsy-based studies are infrequent in literature and there is a lack of data detailing sudden death during physical activity in Turkey, we present a Turkish series of sudden deaths that occurred during soccer games based on data provided by the Morgue Specialization Department of the Council of Forensic Medicine. We identified 15 male cases of soccer-related sudden death aged from 10 to 48 years. Coronary artery disease was identified as the cause of sudden death in 11 cases.

Key words: Recreational, soccer, sudden death, autopsy.

Introduction

Regular physical exercise is recommended by the medical community, because it offers the potential to reduce the incidence of coronary events (Thompson et al., 2003). On the other hand, vigorous exertion may act as a trigger of acute myocardial infarction and sudden cardiac death (SCD) in susceptible individuals (Hull et al., 1994). Sudden death (SD) has been defined as a natural unexpected death occurring instantaneously or up to a maximum of 1 hour after the onset of symptoms (Aguilera et al., 1999).

The main pathology of SCD differs from region to region. In the United States (US) hypertrophic cardiomyopathy (HCM) (36-42%) (Maron et al., 1996b; Van Camp et al., 1995), in Italy arrhythmogenic right ventricular dysplasia (ARVD) (22.4%) (Corrado et al., 1998) and in France coronary artery disease (CAD) (%29) (Fornes and Lecomte, 2003) are the most common causes of death. Although sports-related death is mostly of cardiac origin, trauma, hypo-hyperthermia, dehydration and drug use can also cause it (Oakley, 2001). The most common causes of SCD have been reported as structural abnormalities of the heart in children and adolescents (Franklin et al., 1997; Corrado et al., 1998), HCM below the age of 35 (Maron et al., 1996b; Yanai et al., 2000) and CAD over the age of 35 (Futterman and Myerburg, 1998).

Death during sports activities differs among sports disciplines. In the US rugby (76%) and basketball (68%) (Maron et al., 1996b; Van Camp et al., 1995), in Ireland (Quigley, 2000) and Rhode Island (Ragosta et al., 1984) golf (31.3% and 23.4% respectively), in Italy soccer (44.9%) (Corrado et al., 1998), in Spain soccer (21.3% total and 33.3% of athletes under 35 years) (Suárez-Mier and Aguilera 2002), in France running (42 %, 13/31) (Fornes and Lecomte, 2003), in Israel soccer (39 %) (Yanai et al., 2000) and in Germany swimming (18%) (Turk, 2007) occupy the first place. This is a consequence of the different level of popularity sport disciplines enjoy in different countries: the majority of SD occurs in the sport that is practiced by the most people of that country. In Turkey, soccer attracts more spectators than any other sports activity and the attention of the press and media, and is preferred over other sports by many young and middle-aged individuals.

As autopsy-based studies are infrequent in literature and there is lack of data detailing SD during physical activity in Turkey, we present a Turkish series of SD occurring during the soccer game. This series was examined via a detailed gross and microscopic post mortem examination by forensic examiners. The focus of this study was generally on causes of death during or shortly after practicing soccer and specifically on cardiac pathologies, pathophysiologic mechanisms, optimal screening strategies, and prevention.

Methods

The data of the Morgue Specialization Department of the Council of Forensic Medicine were reviewed in this retrospective study. Approximately 3284 unexpected and enforced suspicious deaths are evaluated annually by this department in Istanbul City.

Deaths that occurred during or shortly after soccer games and were autopsied between the period of January 2000 and December 2005 have been evaluated retrospectively, with respect to the subject's age, sex, incident site, course of incident, all postmortem examination findings and causes of death.

CAD was established as the cause of SD in cases where at least some of the following lesions existed: more than 75% reduction of the lumen by an atheroma plaque in at least one of the main epicardial coronary arteries, extensive infarction scars, acute myocardial infarction, or coronary thrombosis (Burke et al., 1991; Suárez-Mier and Aguilera 2002). HCM was diagnosed in hearts of high weight and/or extensive areas of disorganization of the myocardial fibers, frequently associated with patches of fibrosis and dysplasia of the intramural arteries (Suárez-Mier and Aguilera 2002; Virmani et al., 1997).

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Table 1. The age and the gender of cases, incident time, site, and information about the course of the incident.

Case no	Age/ Gender	Incident site	Day/ Time	Course of incident
1	10/M	Street	Thu /3.30 PM	Fell down while hitting the ball, death at arrival to hospital.
2	11/M	Street	Mon/5 PM	Died suddenly during the match.
3	11/M	Street	Mon /[a]	Collision with another player, death at arrival to hospital.
4	14/M	Stadium	Sat/5.30 PM	After collision with another player.
5	16/M	Stadium	Sat /5 PM	While resting during the break.
6	17/M	Artificial turf pitch	Sun /8.30 PM	Sudden deterioration during the match, death at arrival to hospital.
7	18/M	Artificial turf pitch	Sun /9 PM	Ball strike to the genital area, death at arrival to hospital.
8	18/M	Artificial turf pitch	Fri/11.10 PM	Nausea and headache during the game, death at arrival to hospital.
9	31/M	Artificial turf pitch	Sun/7 PM	Became ill, left the game, fall in the resting room, death at arrival to hospital.
10	33/M	Artificial turf pitch	Sun /4 PM	Became ill, arrest while leaving the game and died in the hospital.
11	35/M	Artificial turf pitch	Sat /4 PM	Became ill during the match, death at arrival to hospital.
12	35/M	Artificial turf pitch	Sat /11 PM	Fell down during the match, died in the hospital.
13	43/M	Artificial turf pitch	Fri/7.30 PM	Became ill during the match, death at arrival to hospital.
14	46/M	Artificial turf pitch	Thu/7 PM	Became ill during the match, death at arrival to hospital.
15	48/M	Artificial turf pitch	Wed/11 PM	Became ill during the match, death at arrival to hospital.

[a] There is no information about the time of incident in the investigation data that were evaluated.

This study was made possible by the authorization of the Education Commission of the Council of Forensic Medicine.

Results

We identified 15 cases of soccer-related SD between 2000 and 2005. All cases were male aged from 10 to 48 years (25.7±13.5). Thirteen cases were recreational soccer players, and only two of them were players (cases 4 and 5) from amateur soccer clubs. The age and the gender of the cases, incident time, site, and information about the course of the incident are listed in Table 1.

All cases except 1, 5 and 12 experienced symptoms at the onset of death. All deaths were witnessed, but resuscitation attempts failed in all. Eyewitness statements and hospital records revealed that all of the cases died on the incident site or during transport to the emergency unit, except for cases 10 and 12. Case 7 had a history of underlying congenital cardiac disease. The other cases did not have any symptoms or previously known disease and cardiovascular risk factors such as familial hyperlipidemia, hypercholesterolemia, family history of SCD, substance and drug intake.

The blood alcohol concentration was 34 mg/dL only in case 14. All of the other toxicological analyses were negative. In all cases, there was no sign of acute trauma to explain death.

The postmortem examinations revealed a 3x2 cm focal subarachnoidal hemorrhage (SAH) in case 8 and a 10x7 cm SAH in case 11. The SAH diagnosis was corroborated through microscopic identification of extensive extravasated erithrocytes in the subarachnoidal space in case 11 and in some areas of the same in case 8.

The mean heart weight was 374 ± 102 g (range 150-500 g). The weight of the heart was found to be considerably greater than the average for the same age group in 9 cases [normal mean weight is 200 g for the 10–20s and 300 g for the 20–30s, normal mean weight is 350 ± 30 g in the over 30s (Tabib et al., 1999)].

In 13 cases hypertrophy of myocardium, in 9 cases scarring of myocardium and in 4 cases fibrosis of myocardium were detected. A fresh occlusive thrombus was found in only 2 cases (case 10 and 14). In 7 cases (1, 3, 4, 5, 6, 11 and 12) at least one of the coronary arteries was severely narrowed.

In the light of the findings obtained from all postmortem examinations and the investigation data related to the SD, the cause of death was determined as CAD in 9 cases, CAD and HCM in 1 case, CAD and SAH in 1 case, HCM in 1 case. The cause of death in 3 cases was indeterminate. The causes of death and detailed postmortem findings are presented in Table 2.

Discussion

The reported incidence of sport related SD varies from 0.16/100,000 to 6.66/100,000 per year in the different series (Maron et al., 1998; Maron et al., 1996a; Maron et al., 2001; Ragosta et al., 1984; Quigley, 2000; Tabib et al., 1999). Literature data is limited, however recently, Corrado et al. reported an annual rate of exercise induced SCD in athletes in the period 2001-2004 as 0.43 per 100 000 person-years (Corrado et al., 2006).

Soccer related SD in literature seems to be limited with the 22 cases of Corrado et al. (1998), 14 cases of Yanai et al. (2000), 13 cases of Suarez-Mier and Aguilera (2002), 6 cases of Maron et al. (1996b), 3 cases of Fornes

Table 2. Macroscopic and histopathological evaluation of heart.

Case no	Weight of heart (gr)	Macroscopy of the heart	Histopathology Myocardium				Cause of death
			Hypertrophy	Scar	DMF	Fibrosis	
1	200	Aneurism in both ventricles and apex, thinning and scarring of aneurism wall, scarring of LV, atherosclerosis of the aorta 90% obstructive calcified atheromatous plaques of both coronary arteries.	+	+	-	-	CAD
2	425	Asymmetric LV hypertrophy, permeable coronary arteries.	+	-	+	+	HCM
3	150	70% obstructive atheromatous plaques and calcification foci in the coronary arteries.	+	+	-	-	CAD
4	360	Subepicardial petechia, permeable coronary arteries.	-	-	-	-	Indeterminate
5	460	Extensive scarring of LV and IVS, LV hypertrophy, severely obstructive atheromatous plaques in the coronary arteries**.**	+	+		-	CAD
6	470	Extensive scarring of LV, scarring and thinning of apex wall, 70% obstructive atheromatous plaques in the left circumflex coronary artery	+	+	-	-	CAD
7	460	Extensive scarring of IVS, asymmetric LV hypertrophy, moderately obstructive atheromatous plaques of the left coronary arteries, mildly obstructive atheromatous plaques of the right coronary arteries.	+	+	+	+	CAD+HCM
8	300	Subepicardial petechia, normal ventricular wall thickness, normal circumference of valves, permeable coronary arteries	-	-	-	-	Indeterminate
9	350	Subepicardial petechia, normal ventricular wall thickness, permeable coronary arteries	+	-	-	-	Indeterminate
10	305	Acute thrombosis of LADCA, non-occlusive atherosclerosis of coronary arteries.	+	+	-	+	CAD+thrombosis
11	375	90% obstructive calcified atheromatous plaques in LADCA, 50% obstructive calcified atheromatous plaques in RCA, scarring of LV.	+	+	-	+	CAD+SAH
12	500	Almost complete obstruction of LADCA, extensive atherosclerotic plaques in the left circumflex coronary artery, extensive scarring along the LADCA trace, biventricular hypertrophy.	+	+	-	-	CAD
13	470	Moderately obstructive atheromatous plaques of the coronary arteries, normal ventricular wall thickness	+	-	-	-	CAD
14	430	Scarring of IVS and LV, acute thrombosis of LADCA, non-occlusive atherosclerosis of left coronary arteries.	+	+	-	-	CAD+thrombosis
15	350	Moderately obstructive atheromatous plaques of the LADCA and left circumflex coronary artery.	+	-	-	-	CAD

LV: left ventricle, IVS: interventricular septum, LADCA: left anterior descending coronary artery, RCA: right coronary artery, DMF: Disorganization of myocardial fibers.

and Lecomte (2003), 2 cases of Quigley (2000) and, 1 case of Burke et al. (1991). Our study is one of the largest series of soccer related SD with reported 15 cases.

It is reported that sport related SD is more common in men (Fornes and Lecomte, 2003; Suárez-Mier and Aguilera, 2002). In our study, all of the subjects who died during or shortly after the soccer game are men. In our country women's choice of recreational sports seems not to be soccer.

In 8 cases the incident occurred at the weekend and in 2 cases on Friday. The incident time varies between 5 PM and 12 PM in eleven cases. Especially all incidents involving cases over 30 years coincide within this time interval, which is appropriate for doing recreational sports activities. Except for 5 cases, all deaths occurred on the synthetic grass pitch.

The cause of soccer related SD was detailed in a study by Suarez-Mier and Aguilera (2002). The causes of 13 soccer-related deaths were as follows: 4 CAD, 3 ARVD, one left ventricle hypertrophy, one dilated cardiomyopathy, one coronary anomaly, and 3 indeterminate.

CAD is a disease the prevalence of which increases with age and concomitant risk factors, especially family history, hypertension (HT), diabetes mellitus (DM), high cholesterol levels, smoking, and substance use. Although patients with coronary artery disease typically become symptomatic after the age of 40, necropsy studies have demonstrated that atherosclerotic changes in the vessel wall begin early in life (Berenson et al., 1992; McNamara et al., 1971).

In our series, CAD is the most common cause of SCD also in very young athletes in contrast with international literature (Maron et al., 1980; Maron et al., 1996b;

Yanai et al., 2000). Corrado et al. (1994) identified premature CAD as a prominent cause of SCD in young persons including some competitive athletes in the Veneto region of northeastern Italy. In fact, CAD may be responsible for SD during physical exertion in young athletes (Burke et al., 1991; Maron et all., 1996b). In a study of sports related SD, not limited to competitive athletes, CAD was the leading cause of death along with HCM (Burke et al., 1991).

Regular physical activity is one of the recommendations for the prevention of CAD and SD. However, heavy physical exercises are a well-known factor related to SD in patients with coronary atherosclerosis. As reported in many studies, intensive physical activity may lead to myocardial ischemia or infarction, ventricular tachyarrhythmias, and SD (Candinas and Podrig, 1990; Cobb and Weaver, 1986; Hauer et al., 2000; Mead et al., 1976; Sisckovick et al., 1984). As result of medicolegal investigations, the cause of death during or shortly after soccer games was determined as CAD in 11 cases (73%). These data confirm that coronary artery disease is responsible for most cases of SD associated with vigorous sport (Northcote et al., 1986; Virmani et al., 1982). During this kind of exercise in subjects with coronary artery disease, oxygen demand may outstrip the ability of the coronary arteries to supply it. It is also possible that metabolic influences like increased free fatty acid and catecholamine concentrations associated with exercise may precipitate or contribute to an arrhythmia and arterial thrombosis (Hoak et al., 1963; Johnson et al., 1969; Kurien et al., 1971). These changes, together with exercise-induced lactic acidosis, and hyperkalemia may also help to explain the failure of cardiopulmonary resuscitation (Bouhuys et al., 1966; Linton et al., 1984). It is also important to underline that SCD may also be the result of a plaque rupture or an acute arrhythmia triggered in the scarring area.

In our study, old infarction scars and severe atherosclerotic narrowing of the coronary arteries were detected in 9 cases, the cause of death of which was attributed to CAD, pointing towards the presence of underlying coronary atherosclerosis and past myocardial infarctions. Acute thrombosis obstructing LADCA (left anterior descending coronary artery) in two cases (cases 10 and 14), congenital HCM in one case (case 7), and SAH in one case (case 11) are concomitant to myocardial scars in these cases. In two CAD cases without old infarction scars the diagnosis was based on obstructive atheromatous plaques observed in the coronary arteries (Table 2). Despite the predominant role of non-atherosclerotic CAD in SCD in the under 15s in literature (Maron et al., 1986; Tabib et al., 1999), it is notable that the youngest victims of the sample examined, two boys aged 10 and 11 years (cases 1 and 3), presented with intensive macroscopic and microscopic features of CAD with lack of previous symptoms and known risk factors. The frequency of premature CAD is surprising in our series, but any alarming symptoms suggestive of CAD and/or coronary risk factors including HT, hypercholesterolemia, familial hyperlipidemia and family history of coronary events have not been recognized in these cases. Unfortunately, in Turkey most people do not seek healthcare unless they have medical complaints. Thus, the families of the cases might not have been aware of any pre-existing illness. Available forensic investigation data on the CAD cases was not conclusive in terms of smoking habits; however, national studies report that the rate of smokers below 18 years varies between 13.1% and 50.5% (Çelik et al., 2000; Kutlu, 2006; Ögel et al., 2000). Recent studies point towards an increase in CAD frequency and mortality due to smoking, unbalanced nutrition, sedentary life style, uncontrolled HT, diabetes and many other factors (Yildirim et al., 2007).

In HCM, strenuous physical activity may act as a trigger mechanism for generating potentially lethal ventricular tachyarrhythmias, given the underlying electrophysiologically unstable myocardial substrate composed of replacement fibrosis and disorganized cardiac muscle cells (Maron, 2001). CAD and HCM were the cause of death in case 7 with a history of congenital HCM, a heart weight of 460 gr diagnosed with severe interstitial fibrosis, extensive areas of disorganization of the myocardial fibers, old myocardial infarction scars in the interventricular septum and moderate narrowing/obstructive atherosclerosis in the coronary arteries. The diagnosis of HCM in case 2 was based on the severe intensive interstitial and perivascular fibrosis detected by means of a histopathological evaluation of the heart that weighed 425 gr. found (observed) in only two of our study cases.

HCM particularly common in young age was lower in our report compared with previously published articles (Maron et al., 1996b; Yanai et al., 2000). This may be due to the fact, that cases with known cardiac abnormalities were not referred to legal authorities and the dead body is usually examined by a general practitioner and certified, if the cause of death is not suspicious. This is why we do not know the prevalence of soccer related SD in our country.

ARVD, known as one of the common causes of SCD during a sports activity, has not been found in our study. The reason may be regional differences in the incidence of right ventricular cardiomyopathy (Corrado et al., 1998; de la Grandmaison, 2006; Mittleman et al., 1993; Turk, 2007).

Doping is a relevant problem even among adolescents, and all doping agents may be the triggering cause of SCD during sports. Myocardial infarction (Dickhuth et al., 1989), sudden arrhythmic death and stroke (Frankle et al., 1988) have been described in young steroid abusers. Toxicological analyses on hair may be useful to rule out doping drug intake prior SD such as anabolic steroids in addition to urine and blood samples.

In the absence of structural abnormality and cardiovascular disease, SD during sports activities may follow a blunt blow to the chest resulting in commotio cordis. Projected objects, such as a ball or body contact between players, forcefully and suddenly impacting against the chest wall during the phase of ventricular repolarization, may induce lethal ventricular fibrillation (Link et al., 1998; Maron et al., 1995). Since the collision history with another player was recorded but the chest trauma was not documented reliably, the cause of death in case 4, although a cardiac arrhythmia due to commotio cordis seems to be the most likely cause of death, is a matter for speculation and was reported as indeterminate.

Although it is not a routine procedure in autopsy, a detailed cardio-vascular system evaluation entailing diagnostic differences in the conduction system of the heart may be necessary related to cases in which no major pathological cause could account for the SD. Where old or pre-mortem electrocardiograms do not exist or conduction system abnormalities are not detected, postmortem diagnosis by DNA analysis of long QT syndrome, short QT syndrome and Brugada syndrome may point out the cause of death.

Focal SAH (case 8) and subepicardial petechia (cases 4, 8 and 9) were evaluated as secondary to asphyxia and as non-death-related secondary non-specific lesions and the cause of death reported as indeterminate. The relative risk of cardiac arrest was greater during exercise than at rest for all levels of habitual physical activity (Siscovick et al., 1984). In our study, cardiac arrest occurred during the break only in one case of death of cardiac origin.

In our country, amateur and recreational athletes do not undergo annual health check-ups, contrary to professional athletes. Our study reveals the profile of soccer related SD by means of detailed postmortem analyses contributing to the discussion and the study database aiming to prevent such deaths.

In line with the recommendations of Maron et al. (1996c), the family history (SD and cardiac illness), detailed history and anamnesis (murmur, systemic HT, fatigue, syncope, dyspnea or chest pain during exercise) of individuals doing sports should be taken. They should undergo physical examination (murmur, femoral pulses, characteristic features of Marfan syndrome, blood pressure measurement) and routine blood and urine analysis should be conducted including whole blood picture, electrolytes, liver enzyme values, 12 lead-ECG (HCM, coronary anomalies, long QT syndrome, Wolff-Parkinson-White syndrome, Brugada syndrome). The findings of the above should be evaluated by cardiology and sports medicine specialists to decrease soccer related SD incidents (Maron et al., 1996c). In their recently published report Corrado et al. showed in a preparticipation screening program with young athletes that was initiated in 1979 that the 4.19/100 000 SCD prevalance detected for the period 1979-1981 decreased to 0.437/100 000 per year between 2001-2004 (Corrado et al., 2006). These results are promising in terms of the applicability of similar programs.

It is also recommended that sports activities should be undertaken cautiously, particularly in the presence of risk factors. In our study, with the exception of one case with HCM, no previous symptoms were determined.

Education of recreational soccer players should aim to raise awareness for warning symptoms such as chest pain, palpitations or syncope, mostly occurring during physical exercise, and improve lifestyle to prevent coronary artery disease. On the other hand, the availability of first aid sets/devices against possible risks on an artificial turf pitch preferably used for recreational soccer games in Turkey can be life saving in some cases.

Conclusion

As the number of individuals doing recreational sports for a healthy lifestyle increases, epidemiologic data on prevalence and causes of death gain importance. Thus, screening strategies can be reviewed and individuals doing sports infrequently and lacking fitness will avoid overexertion.

Individuals should select a sport suitable for their age and general physical condition. Medical screening is important for all people interested in sport, not only for athletes, as a powerful means of prevention.

We hope that reports such as this will increase the awareness of both the public and the medical profession of the dangers of exhausting exercise in unsuitable subjects and reduce the number of these tragic deaths.

Limitation of the study

One of the limitations of this study is a potential underestimation of the number of deaths, because many cases were not referred to legal authorities. Conduction system sampling, DNA analysis for syndromes associated with sports-related SD and toxicological screening of doping agents are not routine procedures in the autopsy protocol and have not been performed in this retrospective study either.

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Key points

- This study is one of the largest series of soccer related SD with reported 15 cases.
- In our series, CAD is the most common cause of SCD also in very young athletes in contrast with international literature.
- In autopsy, detailed cardio-vascular system evaluation and toxicological analysis including doping agents are essential to determine precise cause of exercise induced SD.
- Medical screening is important for all people interested in sport, not only for athletes, as a powerful means of prevention.

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