PROFESIONAL PAPER

Intrahospital Sudden Cardiac Death (SCD) at Internal Medicine Clinic of University Clinical Hospital Mostar

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Aim: Evaluation of general and specific sudden cardiac death (SCD) mortality and comorbidity which advances to SCD in patients of Internal Clinic of UCH Mostar in period from 2000 to 2009. **Methods and Results:** We analysed 2547 death of patients, part of general population of 26.471 hospitalised over this time period. Registered general mortality was 9,6% , specific SCD mortality was 5,85% ,and hospitalised sudden death occurred in 1548 or 60,78% patients. Factors causing death of SCD patients in observed period were as follows: CHF was present in 368 or 23,8%, death after AIM was 344 or 22,2%, ICV was 212 or 13,7%, Shock cardiogenes was 200 or 12,9%, AHF with oedema pulmonum (145 or 9,4%), cardiac arrest was121 or 7,8%, 89 patients experienced sudden death on the street and were transported to Clinic, electrical instability with malignant arrhythmias VF/VT was found in 22 or 1,4%, dissection of aortae 16 or 1,03%, thrombosis a. mesentericae in 12 or 0,78%, and circumstances and end-presentation of death. Age structure was changing, with increase in numbers of younger patients. The results reflects general trend in SCD in grater Mostar region and whose inhabitants gravitate toward our Clinic. **Conclusion:** There is continuous increase in SCD in our patients and in the numbers and comorbidities, which explain existing burden. Although incidence of SCD increases with age, existed continuous trend of increase in SCD in the young people accelerates over time. **Key words:** Sudden cardiac death, mortality, comorbidity.

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1. INTRODUCTION

Sudden cardiac death (SCD) is syndrome, which is more definite at clinical presentation and no real physiology of disease. WHO defined SCD as an unexpected death due to cardiac causes occurring in a short time period (generally within 1 h of symptom onset) in a person with known or unknown cardiac disease. Most cases of SCD are related to cardiac arrhythmias. Approximately, half of all cardiac deaths can be classified as SCDs. SCD represents the first expression of cardiac disease in many individuals presenting with out hospital cardiac arrest.

SCD accounts for approximately 400.000-500.000 deaths per year in the United States, and the same in Europe. Only 2-15% cams to the Hospital, and half them died in Hospital before demission with high risk of recidivisms among survivors (1,2). More deaths are attributable to SCD than to lung cancer, breast cancer, or AIDS. This represents an incidence of 0.1-0.2% per year in the adult population. SCD is often the first expression of CAD and is responsible for approximately 50% of deaths from CAD. The frequency of SCD in Western industrialized nations is similar to that in the United States. The incidence of SCD in other countries varies as a reflection of the prevalence of coronary artery disease or other high-frequency cardiomyopathies in those populations. The trend toward increasing SCD events in developing nations of the world is thought to reflect a change in dietary and lifestyle habits in these nations. It has been estimated that SCD claims more than 7,000,000 lives per year worldwide.(1) The most common mechanisms causing SCD are malignant arrhythmias: 62% VT, 17% bradycardia, 13% VT type torsades de pointes, a 8% primary VF. In 80% arrhythmias de novo is consequence of the higher ventricular ectopia advancing to ventricular arrhythmias. In the 33% cases of SCD, tachiarrythmias results in R/T phenomenon, and 66% cases is results late potentials. Bradyaarythmias and asistolia are only 10-17% of the nonischemic cardiomyopathias. There are multiple factors at the organ (imbalance of autonomic tone), tissue (re-entry, wave break, and action potential duration alternance), cellular (triggered activity, and automaticity) and subcellular (abnormal activation or deactivation of ion channels) level

involved in generation of VT or VF in different conditions. An anatomical or a functional block in the course of impulse propagation may create a circuit with the wave front circling around it and resulting in VT. Other mechanisms such as wave break and collisions are involved in generating VF from VT. While at the tissue level the above-mentioned re-entry and wave break mechanisms are the most important known mechanisms of VT and VF, at the cellular level increased excitation or decreased repolarisation reserve of cardiomyocytes may result in ectopic activity (automaticity, triggered activity), contributing to VT and VF initiation. At the subcellular level, altered intracellular Ca^{2+} currents, altered intracellular K^{+} currents (especially in ischemia), or mutations resulting in dysfunction of a sodium channel (Na^{+} channelopathy) can increase the likelihood of VT and VF (3).

1.1. Causes of intrahospital mortality

Causes of in hospital death are frequently non cardiac, caused by anoxic encephalopathy or respiratory complications in the respiratory device. Only about 10% patient's deaths are caused by arrhythmias recidivism, and 30% are caused by low ejection fraction or cardiogenic shock. Other factors causing negative outcomes are sepsis, asistolia or pulmonary embolism, resuscitation after 5 minutes attacks, neurology deficit in the cerebral creases, carcinoma or Alzheimer disease, presence of two or more chronic diseases, history of cardiac disease (3).

Cardiac arrest (SCA), is manifestation of SCD caused by heart electrical system problem. Olso, MI occurs when one or more of the arteries that supply blood to the heart muscle are blocked. The affected area loses blood supply (ischemia) which results in damage to the heart tissue. An estimated 13 million people worldwide had coronary heart disease (CHD) in the U.S. in 2002. Sudden death is the first manifestation of CHD in 50% of men and 63% of women (1). CHD accounts for at least 80% of sudden cardiac deaths in Western cultures.(2). Provoking factors are:

- Electrolyte disturbances (any reversible metabolic abnormalities should be identified and corrected, particularly hypokalemia and hypomagnesemia which may predispose to ventricular tachyarrhythmias);
- Antiarrhythmic drugs (whenever possible, antiarrhythmic drugs should be discontinued prior to any diagnostic studies);
- Use of an illicit drug such as cocaine can directly cause arrhythmia or produce coronary artery vasospasm and ischemia; a prolonged QT interval which may be acquired (due, for example, to a drug or electrolyte disturbance) or inherited (3).

2. METHODS

From a large cohort of 26.471 hospitalised in 10-years in Internal Clinic we selected 2547 consecutive patients with SCD who were analysed and compared over time period, adjusting our analysis for age, gender, mortality and other risk factors.

3. RESULTS

Majority of SCD patients are in the age group of 65+, then in the group aged 45-65, with group aged <45,being smallest, with the incidence of SCD increasing in that group (Table 1).

SCD was observed in patients presenting with: -CHF (368 or 23,77%), -AIM (344 or 22,22%), I-CV (212 or 13,7%), -cardiogenic shock (200 or 12,92%), - AHF with oedema pulmonum (145 or 9,37%), -cardiac arrest (121 or 7,82%), -patient transported to Clinic and proclaimed DOA (89 or 5,75%), -electrical instability with malignant arrhythmias VF/VT (22 or 1,42%), -dissection of aortae (16 or 1,03%), -thrombosis a. mesentericae (12 or 0,78%) (Table 2 and 3). In hospital SCD in 10-years in Clinical Hospital Mostar-Internal Clinic presented more SCD in women in the 2002. and 2003. In others years the men have a higher incidence of SCD.

By diagnoses over ten years CHF caused 22-32% SCD, AIM was cause of SCD in the 11-29% ICV caused 2% of SCD in 2006 and 22,8% in 2000. Cardiogenic shock caused SCD in 7,5% in 2008. and 18% in 2006.AHF as cause of SCD was 6,5% in 2008 and 15,5% in 2007. Cardiac arrest (SCA) caused death in only 3,5% in 2006 and 10,5% in the 2001. Among proclaimed dead on arrival SCD was 2% in the 2009. And more than 16,4% in 2005.

4. DISCUSSION

According 38 years of follow-up of subjects in the Framingham Heart Study, the annual incidence of SCD increased with age in both men and women. At each age, the incidence of SCD is higher in men than women. In hospital population of Internal Medicine Clinic of Mostar SCD occured more in women in period between 2002-2003 years. The incidence of SCD parallels the incidence of coronary artery disease, with the peak of SCD occurring in people aged 45-75 years. As the prevalence of coronary artery disease increases with age, the incidence of SCD increases with age in men, women, whites, and nonwhites, however, the proportion of SCD from coronary artery disease decreases with age. In the Framingham study, the proportion of coronary artery disease deaths

INHOSPITAL SCD IN INTERNAL CLINIC MOSTAR										
Years	Death	Adm	H.Mt	SCD	Mt SCD (%)	Male	Female	< 45	45-65	> 65
2000	232	2634	8,81	150	64,66	85	65	4	33	113
2001	270	2423	11,14	171	63,33	85	86	8	40	123
2002	257	2369	10,85	143	55,64	61	82	1	28	114
2003	241	2492	9,67	133	55,19	61	72	2	30	101
2004	275	2587	10,63	162	58,90	84	78	2	27	133
2005	272	2657	10,24	164	60,29	100	64	12	29	123
2006	209	2537	9,24	122	58,37	68	54	2	23	97
2007	253	2575	9,83	144	56,92	72	72	1	21	122
2008	242	2572	9,41	174	71,90	96	78	4	33	137
2009	296	3625	9,17	185	62,50	93	92	5	23	157
W10	2547	26471	9,62	1548	60,78	805	743	41	287	1220

Table 1. Intrahospital SCD in Internal clinic in Mostar; Adm–admision; H.Mt–hospital mortality; Mt SCD – mortality of SCD

Years	Aortic disec.	Carcinogenic shock	ICV	AHF	Cardiac arrest
2000	1(0,67%)	19(12,67%)	21(14,00%)	16(10,67%)	16(10,67%)
2001	0	13(7,60%)	39(22,80%)	18(10,53%)	12(7,02%)
2002	2(1,40%)	23(16,08%)	23(16,08%)	13(9,09%)	15(10,49%)
2003	0	23(17,29%)	14(10,53%)	19(14,29%)	10(7,52%)
2004	1(0,62%)	20(12,35%)	22(13,58%)	13(8,02%)	14(8,64%)
2005	3(1,83%)	24(14,63%)	22(13,41%)	14(8,54%)	6(3,66%)
2006	1(0,82%)	22(18,03%)	11(9,02%)	13(10,66%)	12(9,84%)
2007	1(0,69%)	27(18,75%)	3(2,08%)	19(13,19%)	5(3,47%)
2008	4(2,30%)	15(8,62%)	31(17,82%)	27(15,52%)	14(8,05%)
2009	3(1,62%)	14(7,57%)	26(14,05%)	12(6,49%)	17(9,19%)
W10	16(1,03%)	200(12,92%)	212(13,7%)	145(9,37%)	121(7,82%)

Table 2. Diagnoses for SCD patients

Years	FV	AIM	Thrombosis a. mesent.	Sudden death	CHF
2000	4(2,67%)	28(18,67%)	3(2,00%)	3(2,00%)	39(26,00%)
2001	0	40(23.39%)	2(1,17%)	4(2,34%)	43(25,15%)
2002	3(2,09%)	16(11,19%)	2(1,40%)	6(4,20%)	40(27,97%)
2003	1(0,75%)	23(17,29%)	1(0,75%)	3(2,26%)	39(29,32%)
2004	1(0,62%)	40(24,69%)	1(0,62%)	4(2,47%)	46(28,40%)
2005	1(0,61%)	38(23,17%)	1(0,61%)	17(10,37%)	38(23,17%)
2006	2(1,64%)	24(19,67%)	0	20(16,39%)	17(13,93%)
2007	1(0,69%)	42(29,17%)	0	14(9,72%)	32(22,22%)
2008	2(1,15%)	44(25,29%)	0	12(6,90%)	25(14,37%)
2009	7(3,78%)	49(26,49%)	2(1,08%)	6(3,24%)	49(26,49%)
W10	22(1,42%)	344(22,22%)	12(0,78%)	89(5,75%)	368(23,77%)

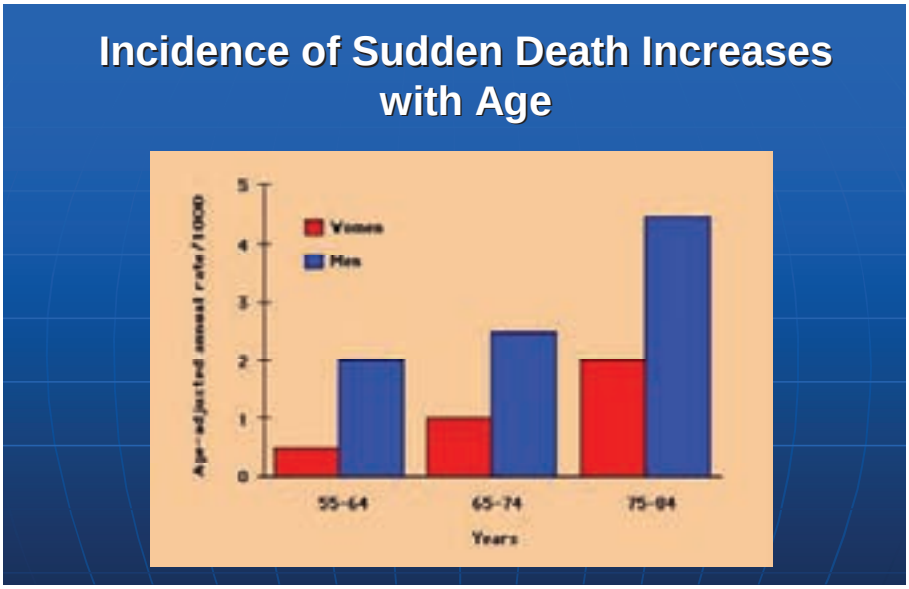
Table 3 Diagnoses for SCD patients- continuation

that were sudden was 62% in men aged 45-54 years, but this percentage fell to 58% in men aged 55-64 years and to 42% in men aged 65-74 years. According to Kuller et al, 31% of deaths are sudden in people aged 20-29 years (1,4,5,6).

It is known that CHF predict increased sudden death and overall mortality. During a 38 years follow-up of subjects in the Framingham Heart Study, the presence of CHF significantly increased sudden death and overall mortality in both men and women (P <0.001).

In people diagnosed with CHF, sudden cardiac death occurs at 6-9 times the rate of the general population (4).

Acute myocardial infarction is associated with an approximate 15% risk of VF within the first 24 to 48 hours, with the incidence falling to only 3 percent over the next several days; in our population in Mostar AIM participated only in 22,22%.



Graph 1. Incidence of sudden death increases with age

When VF is provoked by an AMI, symptoms of the infarction are present for minutes to hours before sudden death occurs; over 80 percent of VF episodes occur within the first 6 hours;

the chance of a successful resuscitation declines by about 7–10% each minute. The actual relationship between defibrillation success and time is a nonlinear one, with the best chance for success probably occurring in the first 3–4 minutes. 5% estimated SCA outhospital survival (4,7). Even in the best emergency medical service/early defibrillation programs it is difficult to have high survival times due to many SCA events not being witnessed and the difficulty of reaching victims within 6-8 minutes. 40% SCAs not witnessed or occur in sleep (1), and 80% SCAs occur at home (8). People who have had a heart attack and have LV dysfunction (less than or equal to 40%) have a sudden death rate that's similar to a CHF population. People who've had a heart attack have a sudden death rate that's 4-6 times that of the general population (2).

Prevention of SCD in general population need to be multidisciplinary program of action and need to encompass correcting ischemia with revascularization and beta-blockers; preventing plaque rupture with statins, ACE inhibitor and Aspirin; stabilizing autonomic balance with beta-blockers and ACE inhibitors; improving pump function with ACE inhibitors and beta-blockers; prevention of arrhythmias with beta-blocker and Amiodarone; terminating arrhythmias with ICDs and AEDs; preventing ventricular remodeling and collagen formation with aldosterone receptor blockade. Al that we help in achieving better prognosis for those patients.(9,10)

5. CONCLUSIONS

Continuous increase in SCD among patients in Mostar is caused by increase in numbers and severity of comorbidity factors and contributes to disproportional increase in deaths. This trend

only partially overlaps with trends in other countries. Modern life stile probably causes increase of incidence of SCD with increase in age, but there is accelerating upward trend of SCD among younger population.

Abbreviations

Sudden cardiac death (SCD), acute myocardial infarction (AIM), chronic heart failure (CHF), stroke (ICV), acute heart failure (AHF), World Health organisation (WHO), Canton Hercegovačko Neretvanski (HNK), Canton Zapadno Hercegovački (ZHK), United States of America (USA), tachicardia ventricularis (VT), fibrilatio ventricularis (VF), angina pectoris (AP), myocardial infarction (IM), coronary heart disease (CAD), right ventricul (RV), block right fasciculus Hissi (BDGH), sudden cardiac arrest (SCA).

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