Original Research

Cardiovascular Disease and Drowning: Autopsy and Laboratory Findings

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Drowning, submersion, cardiovascular disease, post mortem investigation.

Introduction: The purpose of this report is to describe the main autopsy and laboratory findings from a large number of drowning victims in Greece.

Methods. A retrospective analysis was carried out of the consecutive cases of drowning victims autopsied in our department during the period 1997-2004.

Results. A total of 197 submersion cases were referred to the Department. In 168 cases drowning was considered as the cause of death. In 82 cases (49%) significant histopathological findings from the cardiovascular system were present. Alcohol was found in 21 cases (13%) and psychoactive substances in 4 cases (2%). Food was found in the stomach of 45 drowning victims (27%). Men (65%) and elderly people (60 years and older, 74%) made up the majority of drowning victims. In 29 submersion cases the cause of death was other than drowning; in 25 of these cases death was attributed to cardiovascular disease (complication of coronary artery disease, 23 cases; dissecting aortic aneurysm, 1 case; cerebral stroke, 1 case).

Conclusions. The great majority of drowning victims are the elderly and men. Moreover, in a considerable number of submersion cases cardiovascular disease was related to the death, either as a contributing factor, or as the cause of death.

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Drowning is defined as the situation in which an individual experiences a deterioration in respiratory function due to submersion in a liquid medium.[1] Death by drowning is due to the inhalation of the liquid medium (usually water) and may occur immediately or after some time. According to data from the Global Burden of Disease, during 2000 around 449,000 individuals died of drowning worldwide, while drowning also cost 1.3 million years of lost life and disability.[2]

In Greece, drowning is a serious public health problem. According to data from the National Statistical Service, 300-400 people die from drowning annually. More specifically, during the period 1997-2004, 2947 people lost their lives as a consequence of drowning. The majority were men (2103 cases, 74%). The age distribution over the above eight-year period was as follows: 3% of victims (80 cases) were ≤14 years old; 12% (349 cases) were aged 15-29 years; 14% (401 cases) 30-44 years; 17% (478 cases) 45-59 years; 32% (905 cases) 60-74 years; and 22% (634 cases) were over 74 years old.[3]

When a person is pulled from the water dead, or is found alive but dies later, it should not be assumed that the death was due to drowning. In fact, the investigation of these cases is one of the most difficult in forensic medicine. In the following report these cases will be referred to as "submersion cases".

In the present study we recorded the autopsy and laboratory findings from submersion cases that were investigated in our

forensics department. The aim was to identify and classify those factors (pre-existing conditions, effect of substances, etc.) that could have contributed to the occurrence of drowning, with particular emphasis on diseases of the cardiovascular system.

Material and methods

All reports of submersion cases for the period 1997-2004 were collected and studied. The victim's age and sex were recorded, as well as the month in which drowning occurred. The autopsy findings and the results of the toxicological and histopathological examinations were also noted.

The *post mortem* examination of the heart included measurement of the thickness of the walls of the left and right ventricles and the interventricular septum. The dimensions and weight of the heart were recorded. The valves were checked for stenosis and calcification. The ascending aorta was checked for dilatation, thickening, or atheromatosis, and the pulmonary artery was examined mainly for the presence of embolus (and more rarely atheromatosis). Regions of either old or recent myocardial ischaemia were checked for and their location and size were noted. Finally, the coronary arteries were examined (anterior descending and circumflex branches of the left coronary artery; right coronary artery and posterior descending branch) using regular sections every 4-5 mm. Their course was recorded, along with any thickening, atheromatous lesions and stenoses.[5]

For the histopathological examination of the heart, representative sections were taken from the anterior, lateral and posterior left ventricular wall, the interventricular septum, the anterior and posterior right ventricular wall, the anterior papillary muscle, the left and right atrium, as well as multiple sections from the coronary vessels. In addition, sections were taken from any regions with suspected pathological lesions.[4,5]

Alcohol analysis was performed in whole blood samples utilising a gas chromatography head-space method. Psychoactive substances were detected in urine samples using screening techniques and their presence was confirmed by means of gas chromatography-mass spectrometry.[7,8] (It should be further stated that since 1995 our laboratory has participated successfully in the International Collaborative Exercises—the former International Proficiency Testing Programme—of UNDCP for the complete analysis of psychoactive substances in biological fluids).

Results

A total of 197 submersion cases were referred to our department. In 168 cases drowning was considered as the cause of death. All involved seawater. In 167 of these the drowning was considered accidental and only in 1 case was there information supporting the possibility of suicide. In 10 cases (6%) the victim was a foreigner.

Most of the drowning victims were men (109/168, 65%, Figure 1). Only 2 cases (1%) involved children under 15 years old. In 8 cases (5%) the victims were aged 15-29 years; in 17 (10%) they were aged 30-44 years; in 17 (10%) 45-59 years; in 53 (32%) 60-74 years; and 71 (42%) were aged 75 years or older (Figure 2). In most cases the event occurred during the summer months: 24 cases (14%) in June, 65 (39%) in July, and 42 cases (25%) in August (Figure 3).

In 82 cases (49%) there were histopathological findings from the cardiovascular system. In 77 of these cases, the victims suffered from coronary artery disease (old myocardial infarction and/or coronary

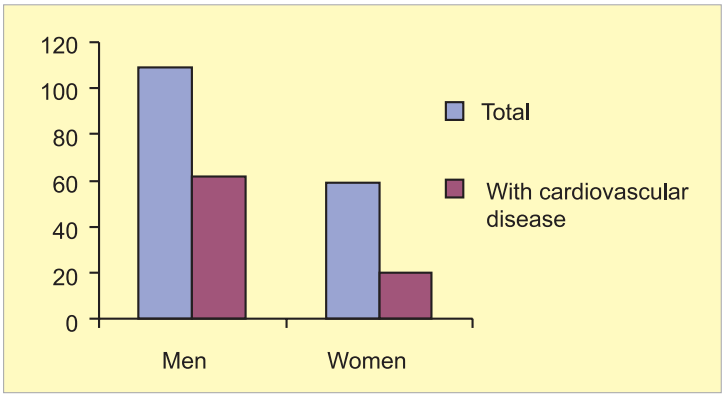

Figure 1. Cardiovascular disease in drowning victims according to sex (total 168).

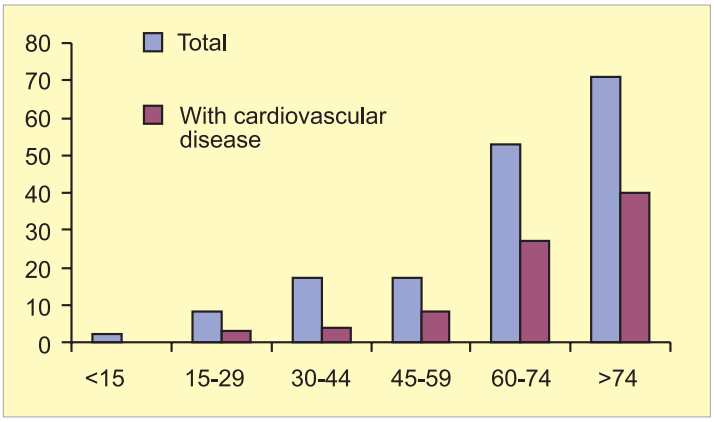

Figure 2. Cardiovascular disease in drowning victims according to age (total 168).

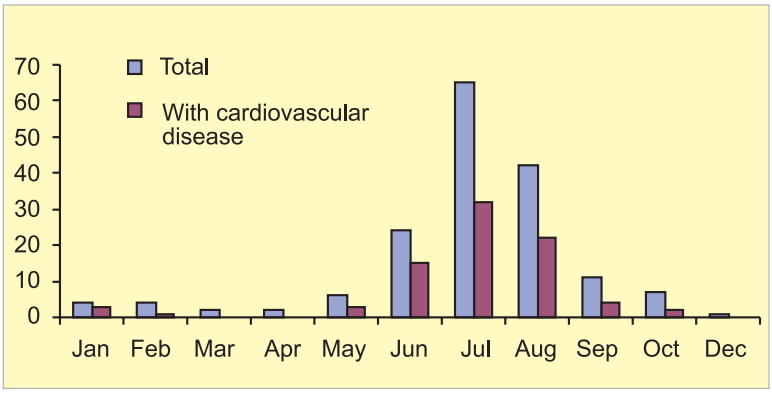

Figure 3. Cardiovascular disease in drowning victims according to the month when drowning occurred (total 168).

artery stenosis >75%), while 8 showed signs of a recent myocardial infarction and just 2 cases had recent coronary vessel thrombosis. In the remaining cases the findings were as follows: aortic valve stenosis in a 66-year-old man, hypoplasia of the left coronary artery in a 37-year-old man, hypertrophic cardiomyopathy in two men aged 16 and 23 years, and Ebstein's disease in a 34-year-old man (Table 1). The incidence of cardiovascular pathology in the drowning victims in relation to sex, age and month of death is shown in Figures 1-3.

Alcohol was identified in 21 cases (13% of the total), with blood concentrations >100 mg/dl in 9 (5%).

Table 1. Pathological findings from the cardiovascular system in 82 drowning victims.

Cardiovascular findings	
Complications of coronary artery disease	77
Aortic valve stenosis	1
Left coronary artery hypoplasia	1
Ebstein's disease	1
Hypertrophic cardiomyopathy	2

Psychoactive substances were found in 4 victims (2%).

Food remains were found in the stomachs of 45 victims (27%).

In 29 submersion cases death was not due to drowning. In 23 the person died from complications of coronary artery disease, in 2 from injuries (amputation by a marine vehicle), while the other 4 causes of death were bowel necrosis, dissecting aortic aneurysm, use of toxic substances and poisoning, and subarachnoid cerebrovascular stroke (Figure 4).

Discussion

The main focus of this study was the collection and presentation of data concerning the circumstances and conditions (diseases, use of alcohol and other psychoactive substances, ingestion of food) related to the causes of drowning in Greece. In addition, the identification of population groups with a higher risk of becoming victims of drowning is of special importance, since they should be the main target of any prevention strategy.

Our study showed that an appreciable percentage

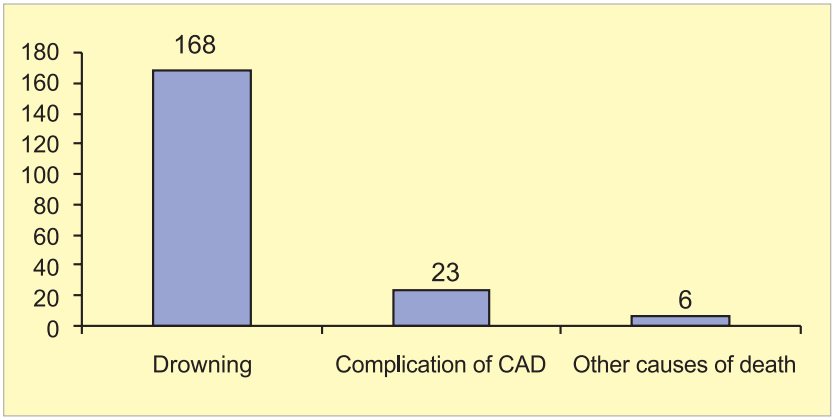

Figure 4. Causes of death in submersion cases (total 197). CAD – coronary artery disease.

of drowning victims (49% of the total) suffered from pre-existing cardiovascular disease, mainly coronary artery disease and its complications. In several cases there was a recent myocardial infarction and in the remainder there were findings showing a large degree of obstruction of the coronary vessels and/or regions of old myocardial ischaemia.

It is, of course, true that physical exercise can contribute significantly to the prevention and better prognosis of myocardial infarction, while swimming, as an endurance sport, has been considered suitable for patients with a history of cardiovascular disease.[9] However, swimming differs from other forms of exercise because of its additional hydrostatic and thermal load. Anyone who is immersed in water with a temperature of 25-29˚C has increased energy conversion, equal to an ergometer load of 75 kW, even without physical activity.[10] Even simple immersion in water induces haemodynamic changes, such as alterations in heart rate, stroke volume and cardiac output, as well as in systolic and diastolic pressure. These changes will be a function of the water temperature, swimming activity level, age, and pre-existing diseases of the cardiovascular system.

During immersion, especially when the body is in a vertical position with the head above water, the central venous pressure increases as a result of flotation pressure.[11,12] In addition, immersion has been shown to cause an increase in the circulating blood volume and a reduction in colloidosmotic pressure (blood dilution) because of fluid transfer from the interstitial to the intravascular space. Subsequently, we see the development of bradycardia and an increase in stroke volume and cardiac output.[11,13] The blood dilution leads to increased diuresis and natriuresis by affecting renal function. At the same time, the increased circulating blood volume stimulates the arterial and cardiopulmonary receptors,[14] while chemical-hormonal mechanisms are also involved in the above haemodynamic changes.[15] Indeed, changes have been found in levels of angiotensin I and II, renin activity, aldosterone, antidiuretic hormone, cortisol, and catecholamines.

These changes appear to be affected by the water temperature. In fact, at a temperature of 32˚C there has been shown to be an increase in stroke volume and cardiac output, a small decrease in heart rate, systolic and diastolic pressure, and a reduction in renin, cortisol and aldosterone.[16] In addition, apart from the above changes, another study reported a reduction in levels of antidiuretic hormone and angiotensin I and II.[17] At 20˚C similar changes were seen, apart from in aldosterone levels, which remained unchanged. At even lower temperatures (14˚C), there was an increase in heart rate, systolic and diastolic pressure, a reduction in renin, and an increase in aldosterone, dopamine and noradrenaline. It has been suggested that, in relatively warm water, the physiological changes are caused by chemical control mechanisms, whereas in cold water they are due to increased activation of the sympathetic branch of the autonomic nervous system.[16] Peripheral resistances have been shown to decrease in relatively warm water (34.5˚C) by 20-40%,[18] whereas in cold water they increase.[19]

The changes in levels of diuretic and antidiuretic hormones also seem to be related with age, since they have been shown to be less exaggerated in the elderly.[17] In elderly individuals, immersion in water induces an increase in stroke volume without a corresponding increase in heart rate, resulting in an increase in blood pressure.[20] This phenomenon is most probably due to a decrease in the adaptability of the autonomic nervous system with advancing age.[21]

The changes caused in response to the increased circulating blood volume during immersion in water might also be expected to be different in chronically hypertensive patients, where the relevant control mechanisms do not function adequately. A study by Coruzzi et al showed that individuals whose hypertension is refractory to a low sodium diet show non-satisfactory diuresis and natriuresis during immersion in water.[22]

Swimming, rather than simple immersion, can affect the changes described in the paragraphs above. Indeed, swimming has been shown to cause an increase in renin and in concentrations of aldosterone and catecholamines, through activation of the sympathetic system. The increase in renin activity is smaller than that seen in other forms of exercise, such as running, because of the effect of immersion in water.[23] The intensity and the type of swimming, as well as previous experience of sporting activities, are also factors that can affect the haemodynamic and chemical changes. For example, it has been shown that during swimming with flippers, blood pressure and heart rate increase less than during free swimming.[24] People who take regular exercise (not necessarily swimming) have been shown to have a smaller increase in noradrenaline levels and renin activity than those who have not previously engaged in sporting activities.[25]

As can be seen from the above paragraphs, during immersion in water, with or without swimming, significant haemodynamic changes take place, whose nature and extent, however, cannot be predicted precisely because of the effect of various parameters. It is likely that the elderly and those with heart disease are particularly vulnerable to these kinds of haemodynamic changes, with the result that symptoms and conditions may appear, such as angina, pulmonary oedema, or even acute cardiac failure. During swimming, anginal symptoms of myocardial ischaemia are likely to be delayed or even absent.[26]

Arrhythmias, such as ventricular fibrillation, are another cause of death during swimming.[27] Cardiac arrest as a result of arrhythmia is probably underestimated as a main cause of loss of consciousness in elderly people with ischaemic myocardial disease.[26] The risk of hypercoagulability induced during swimming and other forms of exercise due to activation factors has also been discussed.[28,29]

Finally, cold may also exarcebate a pre-existing pathological condition. Of course, the majority of the drownings in Greece occur from May to October, when the sea water is quite warm; however, even slight discomfort may trigger a cardiovascular event, especially if the intervening cold adaptation is short.[30]

It is clear, therefore, that swimming may benefit some patients with cardiovascular disease but could prove fatal to some others. It has been proposed that these patients, and particularly those with infarctions, should undergo special testing (e.g. Holter monitoring during swimming) so that those at increased risk may be identified.[26,31]

Apart from structural anomalies, pre-existing functional disturbances, of the conduction system for example, could also potentially contribute to causing drowning; their existence may only be discovered by an investigation of the victim's medical history.[32]

At the molecular level, the search for genetic mutations that seem to be related with the occurrence of dangerous arrhythmias during swimming has been discussed in some recent studies, with particular emphasis on the genes that are responsible for familial long QT syndrome. This syndrome is due to the abnormal function of the sodium-potassium channels (channelopathy). The genes connected with long QT syndrome are KCNH1 (LQT1), KCNH2 (HERG, LQT2), SCN5A (LQT3), ANKB (Ankyrin-B, LQT4), KCNE1 (minK, LQT5) and KCNE2 (MiRP1, LQT6).[33,34] Although the occurrence of dangerous arrhythmias and/or sudden death during swimming has been attributed mainly to LQT1,[35] a *post mortem* study by Lunetta et al showed the presence of the KCNH2 (HERG, LQT2) gene in one of 164 drowning victims, while the LQT1 gene was not present in any.[36]

The CPVT1 gene also leads to channelopathy and arises from the mutation of the gene for the cardiac ryanodine receptor (RyR)-2. Clinically, it is manifested by catecholaminergic polymorphic ventricular tachycardia (hence CPVT) and it is also associated with causing sudden death after emotional overload and/or physical exercise, including swimming.[34]

A molecular search for the above genes in drowning victims, especially in young people who have no other risk factors, should be a part of the investigation into the circumstances and manner of death.[36-38] The molecular examination is of particular importance in individuals whose close family includes a victim of drowning (whether fatal or not), mainly for preventive reasons.[39] Nevertheless, in view of the low occurrence rate of these genes in drowning victims, as well as the variety of mutations that can lead to channelopathy, molecular examination has not become part of the regular rou-

tine. It could perhaps prove useful in the future, in selected cases of younger people who die from drowning and who do not have known risk factors in their medical history or specific findings from toxicological and histological examinations.

In any case, our study showed that only a small percentage of drowning victims were young people (6% of the victims were aged <30 years), while the great majority (74%) were over 60 years old. This age distribution is very different from that found in other countries. In several studies children and young people have been shown to be the high risk groups for drowning.[40,41] In contrast, in our study children made up only 1% of the victims.

The majority of drowning victims were men, which agrees with the findings of studies in other countries.[41-43] According to data from the Global Burden of Disease, men have a higher mortality from drowning at all ages and in all regions.[2] This is probably due to the tendency of men to overestimate their capabilities and to consume more alcohol in comparison with women.[44]

Another factor that has been associated with causing drowning is the consumption of alcoholic drinks. Alcohol may have a negative effect on judgment and/or motor function. Moreover, even mild alcohol-derived hypothermia can result in an impaired cardiovascular response or cause stimulation of parasympathetic tone. Several studies reported that 30-80% of drowning cases were alcohol related.[45-49] Mackie et al presented a quite low alcohol involvement (14%) in drowning cases in Australia.[41] However, in our study alcohol detection was even lower, with only 10% of the drowning victims being positive for alcohol.

The use of psychoactive drugs, and particularly inhibitors of the central nervous system, may also contribute to causing drowning.[45,50,51] In our sample psychoactive drugs were found in only a small percentage of victims (2%).

In Greece there is a common conviction that swimming after a heavy meal can lead to a drowning accident. The consumption of food before entering the water may indeed prove dangerous.[52] However, remains of food in the stomach were found in only 27% of the victims in our study, which probably means that Greeks tend to obey the "don't swim after eating" rule.

It is important to note that the investigation of submersion cases is one of the most difficult tasks in forensic medicine. In these cases, drowning as the cause of death should not be taken as a given. All findings from the autopsy, the histopathological and toxicological examinations, and when feasible, the scene of the accident and the deceased's medical history, should be carefully recorded and evaluated. The strongest pathognomonic finding for drowning on autopsy is frothy fluid in the oral cavity and respiratory tracts. Unfortunately, it is rarely seen because it is expelled by attempts at cardiopulmonary resuscitation or when the body is moved for transportation to the morgue. Heavy, swollen lungs, which fill the chest cavity and cover the precordial space, are also a significant *post mortem* finding considered to be pathognomonic for drowning. Characteristically, on cut section, they have a brick red appearance, with large quantities of oedema fluid flowing from the cut surfaces.[53] In cases with the above findings, death is attributed to drowning, even if there are other pathological lesions or injuries. In fact, according to the rules of the International Classification of Diseases (ICD-10), pathological conditions that lead to drowning through the victim's falling into water or incapacity while submerged are considered to be contributory factors to death, but not causes.[52] For example, if a recent myocardial infarction is found on autopsy, together with pathognomonic findings for drowning from the respiratory system, death will be attributed to drowning and the recent myocardial infarction will be considered as a contributory factor.

In older studies it was maintained that 10-15% of drowning victims do not inhale water, but die from laryngospasm or arrest caused by vasovagal stimulation.[54,55] Recent studies, however, have shown that the incidence of "dry lung" drowning cases is less than 2%, and in those cases another cause of death should be sought.[56,57] In 29 cases of our sample we found no pathognomonic findings for drowning. In all these cases there were other findings to which death could be attributed—such as acute myocardial infarction, coronary vessel thrombosis, dissecting aortic aneurysm—and thus there was no particular problem from the forensic point of view. A particular problem *would* have arisen in the case of a negative autopsy (forensic investigation with no findings). In such cases, the examiner must be wary of attributing the death to drowning. It is probably preferable to characterise the cause as undetermined and to list the possible causes of death in the report.[57]

Conclusions

The findings of this study, of course, are of an indicative nature, since organised epidemiological studies would be necessary to draw clear conclusions about the predisposing factors that can cause drowning and the groups that are at high risk. It is, however, the first study

in Greece to report the findings from a complete forensic investigation of cases of drowning and, more generally, submersion. Our study showed that a considerable number of drowning victims in our sample had heart disease and were elderly. In consequence, prevention strategies should be oriented accordingly. Cardiologists should be informed of the matter and should provide relevant guidance.

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