SUDDEN DEATH

GENERAL INFORMATION; DEFINITIONS

Many cases of sudden death, also known as “Sudden Unexplained Unexpected Death” (SUUD), are in fact suspicious deaths that have to be reported to the authorities as subjects of medico-legal investigation. In most situations, complete medico-legal expertise can offer the necessary objective elements for solving these cases considered suspicious by the authorities.

In numerous definitions of SUUD, a special accent is stressed on the notion of unpredictable death of natural or pathological nature.

The sudden death can be defined as a non-violent death that onsets rapidly and unexpectedly, in a moment when the subject is in a state of apparent health (Popov).

Derobert considered sudden death to be a natural death, predictable or not, with a short – less than 15 minutes – agony, occurring in a subject in apparent health, unexpected by the subject or its entourage.

Moraru states that sudden death is a non-violent, often called natural death, that most frequently it onsets suddenly, preceded only by a short agony, in subjects with apparent health or with illnesses in evolutive stages that don’t predict a near end.

CLASSIFICATION

Simonin divided sudden death in:

- Organic sudden death, with evident lesions and rapid lethal evolution due to cardio-vascular, pulmonar, CNS, digestive, suprarenalian or genital disorders;
- Sudden or suspect death with no evident lesions, specific for a certain illness;
- Functional sudden death with a preexisting pathological state that doesn't justify death. It is the case of chronic, lethal illnesses;
- Inhibition sudden death, situation in which there is a disproportion between cause and effect. In these cases death onsets rapidly in a healthy subject, associated with a minor trauma that does not leave external marks (traces) and with no death-generating organic lesions.
- Functional sudden death essentially pathological, characterized by the so-called "white necropsy" (hypertoxic influenza, infectious diseases).

Scripcaru proposes a simple classification, adequate to serve the practical medico-legal purposes. He divides SUUDh in two subgroups:

- with morphological causes
- with functional causes (biochemical, immunological death)

INCIDENCE, RISK FACTORS, ETIOLOGY

Sudden, unpredicted deaths are frequently encountered in current medico-legal practice; they represent a notable percentage of deaths.

Their causes can often remain unknown, due to the uncertainties surrounding the circumstances of death onset and, sometimes, to the lack of autoptic investigation.

Sudden deaths are more frequent in adults and elderly, but lower percentages cover all age groups.

The most important general risk factors are:

- ✓ ***age***
- ✓ ***gender (men are more affected)***
- ✓ ***irrational alimentation***
- ✓ ***sedentariness***
- ✓ ***obesity***
- ✓ ***climate***
- ✓ ***physical effort and consequent fatigue***
- ✓ ***gastric distention after big meals***
- ✓ ***post-emotional stress***
- ✓ ***toxic factors: alcohol, smoking***
- ✓ ***medical or surgical interventions***

The causes of sudden death constitute a vast domain; they cover the entire clinical pathology.

Almost all clinical illnesses can onset and evolve without premonitory signs and can result in sudden death.

Most frequently, these deaths do not raise any diagnostical problem, because the death-generating internal lesion can easily be identified during the autopsy.

A statistical hierarchy has been established concerning the etiology of sudden deaths, as follows (Derobert):

- cardio-vascular etiology (38-83%)
- respiratory etiology (7-18%)
- CNS etiology (6-27%)
- gastro-intestinal etiology (1-14%)
- reno-urinary etiology (0,1-4%)
- other etiologies (1-5%)

Most authors agree that the most frequent cause of sudden death is coronarian atherosclerosis, either within the frame of cardiological disorders or associated with disorders of other organs and systems.

CAUSES OF SUDDEN DEATH

When pathological deaths onsets very rapidly, almost instantaneously, its cause is almost always cardio-vascular.

1. CARDIO-VASCULAR SPECIFIC CAUSES

Most sudden and unexpected deaths are caused by a lesion of the cardio-vascular system, regardless of the vascular topography (cerebral, thoracic, abdominal etc.). According to Moraru, the most frequent cardio-vascular causes of sudden death are:

a. coronary occlusion
b. high blood pressure
c. bacterian or rheumatic endocardites and miocardites
d. undiagnosed, life compatible, congenital malformations

1.1.The coronary disease

Lately the atherosclerotic coronary disease displays an increasing incidence (frequency) among subjects under 50.

In most cases atherosclerosis represents the vascular cause at the origin of other coronary alterations, especially in ischaemic cardiopaty.

It is well-known that the coronary stenosis due to atheromatosis is a common disorder. Its consequences, results of the diminished blood flow to the myocardium, are dangerous because they can lead to death by various mechanisms.

Coronary failure is the result of the progressive narrowing of the coronary arteries. The walls are thickened as consequence of atherosclerosis or arteriosclerosis.

This change damages of the vascular wall can onset even at young age. The changing of the vascular caliber is more severe than the replacement of cardiac fibber with conjunctive tissue.

The thrombosis of the descending branch of the left coronary artery has the highest lethal risk, followed by the right coronary and the circumflex artery.

The insufficient oxygenation leads to atrophy and systematized fibrosis in the myocardial fibers, with no necrosis.

The hypoxic myocardium is electrically unstable and this state can lead to malignant arrhythmias.

The atheroma can undergo evolutive changes that narrow the initial coronary stenosis and, later on, the myocardial ischemia.

The obstruction of a coronary branch can be the result of massive atheroma (that can ulcerate, split in fragments, mobilize etc.), thrombosis or subintimal haemorrhage.

A frequent complication of atheroma is the subintimal haemorrhage. It usually appears in the atherosclerotic plaques in cases with lethal myocardial infarction.

1.2. The acute myocardial infarction

- is represented by the necrosis of a myocardial area, determined by the arrest or massive reduction of blood flow in one or two coronary (arteries), or of one of their main branches. If 70% of the lumen of a main coronary branch is obstruated, the infarction will usually follow.

Besides the general risk factors, there are also some pathogenic factors specific for the myocardial infarction. The most important one is the coronary occlusion, usually of thrombotic or embolic nature. There is also a particular type of infarction with permeable coronaries; in these cases, the necrosis mechanism is unknown.

A visible myocardial infarction cannot constitute during the onset of sudden death, because it becomes apparent only hours after the coronary occlusion, so in order to be macroscopically identified, 6-8 survival hours after its onset are needed. If death occurs before this interval, the coronary obstruction may be visible, but the infarction can only be confirmed by microscopically or thanato-chemical examinations.

A typical, visible infarction zone is a red-yellowish, homogenous area, of various dimensions, with irregular margins, surrounded by a haemorrhagic collar.

In evolution:

- *the first 15 hours it is pale, with associated oedema;*
- *after 35 hours the center becomes yellowish and the margins hyperemic;*
- *after 3 days the center is brown-yellowish;*
- *after 7 days retraction is visible and after 6 weeks the post-infarction scars is constituted.*

Rhythm and conduction disorders, thromb-embolies, free ventricular wall rupture, interventricular septum rupture and papillary muscle rupture, mostly represent the complications of myocardial infarction that can lead to sudden death.

There are various types of post-infarction conduction disorders, depending on the necrosis location and the coronarian territory.

The etiology of the suffering sinus syndrome (SSS) is the atherosclerotic or rheumatic cardiopaty, with injuries to the sinusal node arteriolae. They will manifest as disorders in generating and conduction the sinusal impulse.

The fatal effects of myocardial infarction can manifest in any moment, once the myocardium is ischaemic.

Atrio-ventricular block that appears in the anterior myocardial infarction is much more severe then in the postero-inferior one; being represented by vagal hypertonia, the latter is reversible.

Pathological studies showed that the complete atrio-ventricular block is the result of necrosis in the Hiss common trunk fascicles_(the right and left branch).

The pulmonary embolism (emboly) is the major complication of myocardial infarction and it frequently determines sudden death. The venous thrombosis represents the source of embolus in the inferior vena cava system; rarely thrombi can start from the right ventricle, when the infarction interests the intraventricular septum.

On the interior surface of the infarction, a mural, endocardial thrombus can develop and parts of it can become detached, producing an embolism.

The rupture of the necrotic myocardium takes place between the second and the twelfth day from the infarction debut.

Before scaring is complete, the necrotic area is soft and friable, with potential for fissures or ruptures.

Free ventricular walls rupture mostly affects the free wall of the left ventricle. The determining factor is the extension of the infarction area.

Another mechanical complication of myocardial infarction is the ventricular pseudo-aneurysm.

The pericardial rupture can be sudden, complete, or it can be preceeded be small, repeated bleedings form the haematoma into the pericardial cavity, before the massive rupture.

An acute cardiac aneurysm appears in the third phase; it is an ectasy in the necrotic area.

The apparition of a massive haemo-pericardium, with cardiac tamponade and brutal sudden death, follows this aneurysm rupture.

The myocardial infarction complicated with myocardial rupture can determine sudden death started from a haemo-pericardium followed by cardiac tamponade.

The rupture of the inter-ventricular septum is rare and it determines a high mortality rate because of the severe haemodinamic disorders it determines.

The rupture of a papillary muscle is another complication of the infarction and of the associated necrosis. It only appears in 1% of the cases and it is a severe complication, with a survival rate of only 14%.

Subjects with total pilier muscle rupture never survive – this complication is incompatible with life.

1.3. Myocardial fibrosis

- usually develops after the infarction heals, since myocardial fibers do not multiply.

The functionality of the conduction system can later be influnced by the extensive post-infarction scars.

1.4. Myocardial sclerosis

- can be the result of inflammatory, toxic or associates processes, with the condition that they have a slow evolution. In the initial phase, it interests the myocardial fibers. Sclerosis appears as a secondary reaction to the changes suffered by the cardiac muscle.

A series of anatomical criteria can be emphasized in myocardial sclerosis: left ventricle hypertrophy, increase of heart weight, valvular indurations and calcification, papillary muscles hypotrophy, degenerescence of the atrio-ventricular node etc. Sclerosis can have various locations, shapes and sizes.

Senile myocardial degenerescence is dennied as a pathological entity by some cardiologists, but it is well known that elderly people die suddenly due to cardiac exhaustion.

Autopsy shows normal coronary arteries and no signs of myocardial ischemia in many elderly subjects. In these cases, sudden death is determined rather by the exhaustion of the myocardial function rather than by a coronary disease.

1.5. Myocardites

- can cause sudden death but their incidence is much lower than the degenerative processes.

Myocarditis can appear in some infectious diseases, such as diphtheria and viral infections, including influenza.

The aspect of the myocardium gives the elements that can lead to the suspicion of myocarditis during the autopsy.

It can be dull, with a boiled meat aspect, dry, rose-yellowish, pale as a whole or tigerish in some areas, with flasque consistency so that it barely maintains the fingerprint.

1.6. Cardiomyopaties

- represent another situation with a more powerful intrinsic effect.

An enlarged heart, with specific histo-pathological characteristics, characterizes them. Some of them are due to metabolic disorders; others are "idiopathic".

In cases where the heart is severely enlarged, over 1000 grams, strong, sometimes asymmetrical, vibrations of the ventricular walls can occur, especially in cases with hypertrophic enlargement.

In congestive enlargement, an accentuated dilatation of the cardiac cavities can be observed. All types of myocardiopathies can be associated with sudden death.

One of the most frequent is the ***ethanolic myocardiopathy***. It occurs in chronic alcoholics due to the acetaldehyde excess resulted from ethanol metabolism, with toxic effect on the myocardial fibers enzymes.

A pale myocardium with fibrosis stripes and thickening of the endocardium are elements that suggest this type of disorder.

Microscopy shows myofibrilar hypertrophy alternated with atrophy, vacuole, interposed disk anomalies.

1.7. The hypertensive disease

- is a pathological condition that can lead to sudden death due to the accentuated hypertrophy of the left ventricle.

In hypertension, the myocardium has to perform its activity in conditions of high blood pressure.

This situation leads to a compensatory myocardial hypertrophy, meant to ensure the pumping of high-pressured blood.

Atheromatosis is also frequently associated with hypertension, so the hypertrophied muscular mass becomes ischemic.

1.8. Arterial pathology

- can also be involved in the determinism of sudden death. The most common arterial lesion associated with sudden death is the aneurysm.

The atherosclerotic aortic aneurysm can develop anywhere on the aorta, but most frequently on the abdominal segment.

Usually the aneurysms are large, consequence of the arterial wall deterioration by atherosclerosis. Frequently subjects are elderly males.

The arterial swelling can sometimes reach impressive dimensions; it can be sacular (developing only on one side) or fusiform (cylindrical).

The vascular wall becomes thinner due to the destruction of the elastic fibers, and the intima is ulcerated, often calcified.

Many aneurysms remain intact, silent and are only discovered accidentally, during the autopsy.

Others can rupture, especially when high blood pressure is associated with advanced age.

Sometimes aneurismal ruptures can be surgically repaired, but most of them are lethal, with no possibility of medical aid.

Aneurysms can determine massive haemorrhages, with extension in the retroperitoneal tissues, sometimes peri-renal; in other cases a massive haemo-peritoneum can occur.

The disecting aortic aneurysm **occurs when the aortic blood penetrates inside the arterial wall through a discontinuity. Most of the times the cause is the rupture of the vascular intima near an atheromatous plaque or due to a degenerative disease of the intima.**

As a result, a medionecrosis of the aortic wall will onset; it will lead to a resistance decrease in its middle layer. The blood can erupt in this layer, usually intermediated by an atheromatous plaque.

The blood flow can dissect the aortic wall, extending downwards towards the iliac and femoral branches but mostly upwards, around the aortic arch and in the pericardial sack, leading to haemopericardium and cardiac tamponada.

The syphilitic aneurysm **is relatively rare, due to the effective primary and secondary treatment of syphilis available. It can still be identified during routine autopsies of elderly subjects.**

1.9. Aortic stenosis

- leads to left ventricle hypertrophy.

The hypertrophy can be even more accentuated than in hypertension. Usually the cause is a calcified stenosis of the aortic valve, probably atherosclerosis-related.

It usually affects men over 60 but it can be encountered in younger subjects with congenital aortic bicuspid valve. In these cases sudden death frequently occurs.

1.10. Pokkuri syndrome

- can determine sudden death mostly in young bradicardic subjects.

Its etiology is represented by changes in the structure of vessels that cover areas of origin or conduction of the nervous impulse in the sino-atrial or atrio-ventricular nodes.

Routine autopsy procedures do not identify any significant change, accountable for the sudden death of these subjects.

In the absence of significant clinical and macroscopical autoptic elements to sustain the diagnosis of sudden death, Pokkuri syndrome has some specific markers, such as:

- **subendocardic nodular fibrosis stripes, considered to be of hypoxic nature,**
- **associated with pericapilar and perineuronal diffuse gliosis.**

2. RESPIRATORY SPECIFIC CAUSES

2.1. Pulmonary embolism

- is rather frequent, but it is rarely diagnosed as cause of death.

In almost all cases, the superficial or profound veins of the inferior limbs represent the source of the embolism.

Rarely they can start from haemoroidal, utero-ovarian, iliac, mesenteric thromboses; sometimes they can have cardiac origin.

After any tissular trauma, especially those requesting long immobilization, an accentuated thrombosis develops.

Some emboli do not induce any pulmonary lesions, others determine infarctizations with or without clinical signs, while others block most of the blood vessels and determine death.

The fresh (recent) embolus is different from the blood clot; it is drier, whitish, friable and striated (Zahn). If the subject survives a few days, the thrombo-embolic material becomes adherent to the vessel walls.

The obstruction of the respective pulmonary artery or of some of its main branches determines, in most cases, shock and death in a few minutes.

In these cases the embolus is big (5-10 cm long and 1-1.5 cm diameter); this suggests its origin in large veins, like the femoral or the iliac.

The obstructed artery appears more distended and after sectioning the lungs, the thrombo-embolic material can be expressed the obstructed vessel.

2.2. Bronchial asthma

- is an asphyxic syndrome that can sometimes be incriminated as sudden death generator.

It is caused by diffuse bronchiolar obstruction induced by the presence of mucus in thick, coherent layers ("mucus corks"), the thickening of the bronchiolar membrane due to inflammatory oedema, the cellular infiltrate and the bronchial spasm.

In the initial phase there is only hypoxia, but if the respiratory effort persists, as a result of the resistance in the way of the inspired air, the hypoxia can grow dramatically. The bronchial spasm is always associated with a certain degree of hypertension in the pulmonary circulation, worsened by hypoxia and hypercapnia.

The overload on the right ventricle appears while the myocardium is irrigated with hypoxic blood, so the acute right ventricle failure can occur at any moment.

2.3. Spontaneous pneumothorax

- can determine the onset of acute respiratory failure by brutally colabating a lung, especially when the other lung has preexisting lesions; the colabated lung is crushed by the mediastinum.

Spontaneous pneumothorax, especially the suffocating one, with a "valve", induces circulatory and respiratory disorders like asphyxia and vascular collapse that can lead to death.

2.4. Pulmonary tuberculosis

- can be considered one of the rare causes of sudden death. The accentuated erosion of large vessels can lead to massive, fulminant haemoptisis that can leat to death in a matter of minutes.

2.5. Acute bacterial inflammatory

- *diseases (*pneumonia, broncho-pneumonia), although with a low incidence among sudden death causes due to antibiotic therapy, can sometimes present an unfavorable progressive evolution, with lethal ending.

Acute respiratory failure can onset in the evolution of extensive, massive, severe forms and also in forms complicated with massive para-pneumonic pleurisy.

***Viral pneumonias* are more frequently the origin of sudden death, due to the extension of the inflammatory phenomenon in the pulmonary parenchima, where it generates interstitial pneumopaties.**

2.6. Pulmonary hydatid mole

- although rare, can be involved in the etiology of sudden death. The cyst rupture can determine lethal anaphylactic shock.

2.7. Pulmonary cancer

- is also a rare cause of sudden death. Death can occur due to the erosion of a large vessel, massive haemorrhage and haemorrhagic shock.

3. CAUSES SPECIFIC FOR THE CENTRAL NERVOUS SYSTEM

3.1. Cerebral aneurisms

- are a relatively frequent cause of sudden collapse followed by sudden death, in young, middle aged and elderly subjects.

Most frequently aneurysms are located in the Willis Polygon or on the internal carotid artery. Their rupture results in sub-arachnoidian haemorrhage.

The aneurhysmal apex can rupture at any blood pressure increase, caused by even a minimal physical strain or by emotions.

The combination of these factors during sexual relations makes the association with aneurysm rupture pertinent.

Death can sometimes be very rapid and in these cases it is difficult to establish its cause.

The subject can suddenly fall, without any previous clinical manifestation, and die in a matter of seconds.

In such cases it can be presumed that the sudden brain flooding with high-pressured blood affects the cardio-respiratory centers, determining cardio-respiraotry arrest and rapid exitus.

Alcohol is considered to be a risk factor for ruptures because it determines dilatation of the cerebral vessels and increase of blood flow.

The increased blood pressure in subjects under the influence of alcohol is a probable cause for aneurism rupture.

3.2. Sub-arachnoidian haemorrhage

In over 50% of the cases, sub-arachnoidian haemorrhage is the consequence of ruptured aneurisms.

It can be responsible for about one third of all vascular cerebral deaths in subjects over 60.

Once in the sub-arachnoidian space, through the cisterns, a part of the escaped blood mixes with CSF (cerebro-spinal fluid), the other part remains as liquid or partially clotted blood.

After massive haemorrhages most of the blood remains liquid.

In some cases the blood can cross the pia mater and infiltrate the cerebral substance – meningo-cerebral haemorrhage.

If the haemorrhage is situated in the sub-arachnoidian space form the base of the brain, the blood can enter the cerebral ventricles or it can diffuse in the sub-arachnoidian space.

3.3. The intra-cerebral haemorrhage

Sudden bleeding in the cerebral substance is frequent, mostly in elderly or hypertensive subjects.

The etio-pathogenic factors involved in the determinism of intra-cerebral haemorrhages are: high blood pressure, atherosclerosis off the cerebral vessels, malformations of the cerebral vessels, haemorrhagic diseases, tumors.

Most frequently the haemorrhage occurs in the external capsule of one of the hemispheres, due to the rupture of an lenticulo-striated artery, sometimes called "the Charcot vessel" or even "the cerebral haemorrhage artery".

The sudden expansion of the intra-parenchimal haematoma dissects and lacerates the cerebral parenchyma, sometimes associated with ventricular flooding.

It can also compress the internal capsule and destroy a part of it; the result is a hemiparesis.

If the haemorrhage is extensive, a large part of the hemisphere can be destroyed.

Only some of these haemorrhages are lethal and death is usually not instantaneous; it onsets after a certain interval.

3.4. The cerebral infarct and thrombosis

Cerebral infarct is represented by the necrosis of a portion of the cerebral parenchyma as a result of blood flow arrest due to the obstruction of a cerebral blood vessel.

Usually the cerebral infarct is the result of a severe, partial or total, arterial occlusion. The occlusion is mostly of thrombotic origin and rarely embolic.

3.5. The cerebral embolism

- represents the blockage of a cerebral artery by a solid, liquid or gas particle, transported in the systemic circulation.

The embolic arterial occlusion is frequently followed by an embolic, ischemic or haemorrhagic cerebral infarct.

3.6. Epilepsy

- is sometimes an etiological factor of sudden death, especially the overposed seizures or the grand mal, if the apnoea is prolonged and breeding is reinstalled after 3-4 minutes.

Without a detailed anamnesis, it is difficult to diagnose epilepsy as the cause of death during the autopsy.

3.7.Cerebral tumors

- can determine massive cerebral haemorrhage by neoplasic necrosis of the vascular wall.

4. GASTRO-INTESTINAL SPECIFIC CAUSES

4.1. Gastro-duodenal ulcer

- is associated with a feared complication – the superior digestive haemorrhage, its essential clinical manifestation.

If the chronic ulcerative lesion is profound, it can erode a more or less large vessel, either tangential or by transversal sectioning.

It provokes massive haemorrhages with exceptionally rare spontaneous haemostasis.

When the blood loss is of 1500-2000 ml and the haemorrhage onsets rapidly, sudden death can follow.

In the massive digestive haemorrhage, the haemo-dynamic disorders are characteristic to the hypo-volemic shock.

The gastro-duodenal ulcer can frequently complicate with a perforation either in the free peritoneal cavity, determining the generalized peritonitis, or in a limited peritoneal space (lesser omentum), determining a localized peritonitis.

The perforation is the most dangerous complication. Untreated in due time it results in death by peritonitis or septicemia.

4.2. The acute gastric dilatation

- *after a big meal* can determine vagal irritation, associated with a difficult respiratory movements and cardiac contractions.

4.3. Esophageal varicose veins

- can contribute to the onset of sudden death by their rupture followed by massive haemorrhage.

Untreated it can lead to irreversible haemorrhagic shock and sudden death.

4.4. Acute haemorrhagic pancreatitis

The pathogeny of acute pancreatitis involves two fundamental processes:

- **the intra-pancreatic activation of the proenzymes**
- **the auto-digestion of the pancreatic gland under the action of the activated digestive enzymes. In the end, the auto-digestion determines specific lesions of cyto-steatosis and intra-pancreatic haemorrhagic lesions.**

The necrosis can extend in the neighboring tissues. In severe cases the whole pancreatic tissue is altered.

The intense pain and the state of shock can be followed by rapid death.

4.5. The intestinal occlusion

- is a clinical syndrome with multiple determinants (determinations), characterized by the arrest of the intestinal transit.

The occlusive shock and the intestinal gangrene consequent to the intestinal ischemia can be fulminant and fatal.

4.6. The spontaneous rupture of pathological spleen

- can be encountered in some parasitoses, malaria, leucoses or in case of congestive spleen with the decrease of reticular fibbers.

It can be fatal.

4.7. Non-traumatic perforations of abdominal cavitary organs

- can also determine sudden death.

They can be:

- **perforated appendicitis**
- **Meckel diverticulum perforation**
- **the small intestine perforation in Crohn disease**
- **the colon perforation – tumoral or dyastatic**
- **the perforation of colon diverticulosis**
- **perforation of the gall bladder in acute inflammations.**

All these perforations can lead to frequently lethal peritonitis and septicemia.

4.5. THE URINARY SYSTEM; SPECIFIC CAUSES

4.5.1. Renal sclerosis and chronic nephritis,

- compensated and asymptomatic, are followed by uremia in most of the cases, even in acute forms.

General macroscopic findings include:

- ✓ cerebral swelling and oedema
- ✓ pericarditis
- ✓ uremic pneumonia
- ✓ ulcerations of the gastric mucous membrane.

4.5.2. Difuse acute glomerulonephritis

- is represented by a glomerular inflammatory process that affects almost all glomeruli, bilaterally, associated with proliferative and exudative lesions.

A small percentage of the cases can have a lethal evolution by uremia and cardio-vascular complications.

4.5.3. Acute tubular nephropathies

- are clinically known as acute renal failure.

4.5.4. Suprarenalian apoplexy consequent to severe viral infections can also be causes of sudden death.

4.6. GYNECOLOGICAL CAUSES

Feminine genital disorders sometimes lead to acute abdomen followed by sudden death.

4.6.1. Ectopic ruptured pregnancy

- is the most frequent cause of acute abdomen. It can have tubar, ovarian or abdominal location.

Tubar rupture is the most feared accident in extra-uterine pregnancies and it represents a high emergency situation. If surgical intervention is not prompt, the massive intraperitoneal haemorrhage can be lethal.

4.6.2. The utero-placentar apoplexy (the premature placenta detachment, the Couvelaire disease) is sometimes a high-risk disorder for sudden death.

4.6.3. Placenta praevia

- can be caused by multiple births, genital infectious processes, uterine or para-uterine tumors etc.

Haemorrhages occurring in the third semester of pregnancy or during labor can be extremely severe.

4.6.4. Eclampsia

- is an advanced and severe form of late pregnancy disorder that onsets with an attack.

Its physio-pathological mechanism is vascular, but cerebral oedema can add to it.

The defining clinical element is represented by the tonico-clonic convulsions.

If the coma lasts over 24 hours, the prognostic becomes very reserved and the severe evolution is followed by death.

Autopsy shows severe liver and kidney lesions.

The liver is slightly oversized, pale-yellowish or redish; the capsule is sometimes detached by the escaped blood, other times it has smaller or larger dark-brown spots. On the section surface it is pale-yellowish, with small, irregularly disseminated haemorrhagic spots or irregular, brown-reddish areas (red infarcts).

Kidneys present changes of acute congestive nephritis – easy decapsulation, brown-reddish cortical, brown medullar stripes, due to the congestive vessels; the mucous membrane of the renal pelvis shows reddish spots.

4.6.5. The ruptured piosalpinx

- can lead to diffuse generalized peritonitis, which can sometimes be lethal due to toxico-septic shock.

4.6.6. The ruptured or torsioned ovarian cyst

- can reproduce the clinical symptoms of the ruptured ectopic pregnancy or of the ruptured corpus luteum, with massive intra-peritoneal haemorrhage.

The ovarian cyst torsion compresses its blood vessels and blocks the return circulation, without modifying the blood flow.

The result is intra-cystic haemorrhages, infection, necrosis and rupture, with acute siffuse peritonitis.

4.6.7. The amniotic liquid embolism

- consists of the transportation of amniotic elements (epidermal cells, vernix caseosa, lanugo, trophoblastic cells), through a discontinuity in the maternal circulation, followed by vascular distress, shock or acute fibrinolisis.

This type of incident is more frequent in subjects with multiple births, when strong uterine contractions determine the dissolution of the amniotic membranes.

SUDDEN INFANT DEATH SYNDROME (SIDS)

Sudden death in infants (SIDS) is relatively frequent, with variable incidence and etiologies. It occupies the first place among the causes of perinatal deaths in countries with relatively low infant death rates.

SIDS can be defined as the sudden death occurred in an apparently healthy suckling.

It is unexpected, considering the child's medical history and the autopsy does not always reveal a satisfactory cause of death.

Most of the times, the cause of death is established by association with the autoptic findings and the microscopical examination of samples collected during the autopsy.

The history of SIDS cases is usually similar – an apparently healthy baby or with minor symptoms, set in his cot in the evening, is found dead in the morning.

The main characteristics of infant sudden death emerge from the following findings:

- ***The generally accepted age range for SIDS is from 2 weeks to 2 years, with a peak between 2 and 3 months;***
- ***There is no significant difference between genders;***
- ***SIDS incidence is high among twins, identical or not;***
- ***There is a clear seasonal variation in the temperate areas, with higher incidences in the cold, windy nights;***
- ***Low economic standards seem to associate with higher incidences.***

The etiology of SIDS:

- the cause of death is usually determined during the autopsy. Histo-pathological examination usually confirms the macroscopical diagnosis or it offers the death cause in cases in which the autopsy is irrelevant in this respect.

Respiratory pathology occupies the first place among the causes of death, especially viral and bacterial infections with supra-acute, haemorrhagic evolution.

One of the most frequent causes of death in SIDS is the interstitial pneumonia. Macroscopically it is characterised by isolated or confluent centres; in severe forms, diffuse processes cover large pulmonary areas on entire surface of the lungs.

In the affected areas the pulmonary parenchima is dense, hypo- or an-aerated, meat-like reddish or slightly greyish. The dense areas alternate with emphysematous or atelectatic areas, secondary to the bronchial lesions.

Microscopy shows the thickening of the alveolar walls with consequent lumeneal decrease.

Another respiratory cause is the broncho-pneumonia. Even though its incidence was decreased by the use of antibiotics, it can still be encountered during the autopsies, documented by the presence of white-yellowish pus clots on the lungs sections.

A series of cardio-vascular disorders are involved in SIDS etio-pathogeny:

- congenital cardio-myopaties
- idiopathic cardiac hypertrophy
- supra-ventricular paroxistic tachycardia etc.

During the warm months there is an increase in the incidence of digestive disorders, represented by acute dyspepsia, diarrhoea and severe hydro-electrolitical alterations that will finally result in acute cardio-respiratory failure and death.

Intra-thoracic haemorrhagic petechiae are found in approximately 70% of SIDS autopsies. They are situated on the pleura, epicardium or thymus.

This finding can lead to the wrong interpretation that the death is due to mechanical asphyxia, but petechiae can also be caused by the breathing efforts when the air passages are obstructed due to a glotic spasm or the hypotonic collapse of the pharynx; so they can be a consequence of an agonic phenomenon and not a marker for mechanical asphyxia.