G79 Cardioinhibitory Reflex Cardiac Arrest (CiRCA): Is it Possible Without Contributory Factors?

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After attending this presentation, attendees will learn that a single, short neck trauma can lead rapidly to death stimulating the carotid sinus cardioinhibitory reflex, without any compressive effect on the carotid arterial flow or any mechanical asphyxia.

This presentation will impact the forensic science community by highlighting that, as reported in the literature, there is no evidence proving that carotid stimulation alone can determine death.[1] In fact, in the present case, cardioinhibitory reflex cardiac arrest (CiRCA) resulted in the interaction between mechanical carotid sinus stimulation and underlying pathological and toxicological contributive factors.

It is widely known, in cardiology, that compression of the neck can produce variations in cardiac rhythm with bradycardia, syncope, and, in some cases, circulatory collapse resulting from the activation of an arterial baroreflex.[2,3] Remarkable interindividual variability in cardiac response has been reported.[4]

This physiopathological mechanism has been cited even in forensic setting, as the questioned cause responsible for deaths in which no other clear explanation is found. Many case studies have been recently reviewed[1] highlighting that reflex cardiac arrhythmia by neck stimulation is able to provoke death mainly with the contribution of other preexisting factors, such as drug abuse, cardiac diseases, and physical and/or mental excitation.

A 22-year-old Bangladese male, while spending time with some friends and other guests drinking alcohol in a night club, was dragged into a brawl outside of the establishment. Circumstances were carefully described by five different witnesses, all concordantly reporting that the man was hit by a single very strong punch on the left side of the neck, just below the mandibular angle. After the blow, the young man had a syncope and fell down, unconscious and unresponsive to intensive care.

Clinical records were examined in detail, including electrocardiograms. A history of chronic alcohol abuse, with multiple visits to the emergency for ethanol overdoses, was noted; apart from that, pathological anamnesis was normal.

Autopsy showed a remarkable bruise in the neck subcutaneous tissues and in the upper tract of the left sternocleidomastoid muscle, close to the neurovascular bundle. Further pathological exam ruled out any relevant traumatic brain or neck injury and cardiac disease.

Toxicology on postmortem blood specimens gave negative results for the most common drugs of abuse, except for the detection of ethanol 3.53g/L. Such blood alcohol concentration was similar to *in vivo* toxicological data of the subject, assessed during previous acute ethanol intoxications.

Forensic genetics were carried out screening mutations in three main genes (KCNQ1, KCNH2, SCN5A) involved in the Long QT Syndrome (LQTS). The deceased was a carrier of two different mutations in exon 11 of the KCNH2 gene, codifying for an ion channel named “HERG,” related to the LQTs type 2. One was a silent mutation, but the subject was eterozygous for a missense mutation in A2690C, responsible for aminoacidic substitution (from lysine to threonine) at codone 897 (K897T).

In this reported case, death by CiRCA was highly probable, but it was mediated by contributive factors, like alcohol intoxication and possible arrhythmic diathesis genetically determined. In this case, the high postmortem blood alcohol concentration does not explain death itself. In fact, in the literature,[5] lethal overdoses refer levels above 4.0g/L. Furthermore, tolerance to high alcohol levels by the subject, prone to repeated acute intoxications, must also be considered. More probably death resulted by mechanical activation of cardioinhibitory reflex at the neck with consequent bradycardia and loss of consciousness; then ethanol-related cardioinhibitory effects and brain depression led to final cardiac arrest. Questions remain as to the contribution of the genetic factor. In fact, while the detected missense mutation of KCNH2 gene seems to produce *in vitro* electrophysiological changes in sodium channel permeability,[6-8] its pathological significance *in vivo* is still uncertain.

In conclusion, the reported case confirmed that CiRCA should be a diagnosis by exclusion. It must be designated to cases in which circumstances are directly witnessed, and it should be supported by modern pathological, genetical, and toxicological analyses, to rule out other possible mechanisms of sudden unexplained death.

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CiRCA, Carotid Sinus, Neck Compression

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