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Case Report

The role of myocardial bridge of the left anterior descending artery in a sudden death of a ketamine and cannabis user. Addressing the uncertainties on the cause of death in a forensic pathologist's casework

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ABSTRACT

In this case report, the role of a myocardial bridge of the left anterior descending artery (LAD) and recent use of cannabis in a sudden death of a drug user is discussed, also considering the relevance of histopathological pulmonary findings.

A 37-year-old man with a history of drug abuse was found dead in his house. External and autoptic examination were performed, as well as histologic and toxicologic analyses of tissues, organs and body fluids samples. Autoptic examination revealed signs of previous endovenous drugs administration and a 0.5 cm deep intramyocardial bridging of a 1.5 cm long segment of the left anterior descending (LAD) artery. Histo-pathologic examination revealed the presence of diffuse alveolar hemorrhage and multiple alveolar macrophages containing brownish pigments. Toxicological analysis of post-mortem blood revealed recent use of ketamine (<0.5 ng/ml) and cannabis derivatives (8.8 ng/ml for THC and 2.2 ng/ml for 11-OH-THC) in a subject with a history of previous abuse of cocaine, MDMA, and ketamine, confirmed through hair analysis. The analysis of forensic literature, thoroughly examined in this paper, along with a comprehensive evaluation of all post-mortem data, indicates that cannabis likely contributed to the cause of death, in conjunction with myocardial bridging. This suggests a dual mechanism involving both cardiac and pulmonary factors. This case could add to the limited body of evidence in the literature and provide insights into the potential contributory role of cannabis in sudden deaths.

1. Introduction

In drug-related deaths, multiple variables, such as inter-individual differences in administration, absorption, distribution and metabolism, development of tolerance in long-term users, use of the substance alone or in polyadministration with other drugs, or presence of previous comorbidities, do not allow the identification of a drug lethality's cut-off [1–3]. Apart from toxicological data, histopathological findings can aid the interpretation of toxicological results, but microscopic damage can be nonspecific and not uniquely attributable to drug abuse [4]. The issue becomes even more complex when assessing the contributory role of cannabis, given that the literature is controversial on this topic and is primarily based on case reports or case series [5].

The interpretation of toxicological results may be also complicated by individual pre-existing conditions or diseases, often unknown, or detection of post-mortem blood concentrations of drugs that are lower than those typically observed in fatalities caused by that specific substance. Among pathological conditions, particularly cardiac ones, those falling into a "gray zone" are difficult to interpret when determining the cause of death, as also reported by the Association for European Cardiovascular Pathology's guidelines [6], that categorize cardiac alteration as "certain", "highly probable", or "uncertain", when defining the cause-effect relationship between cardiovascular findings and sudden death. To address these multiple uncertainties, it is imperative to build a strong body of literature evidence, including autopsy, toxicological analysis and histologic examination, as highlighted by Kasuda et al. [7].

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In this case report, the role of cannabis and myocardial bridge of the left anterior descending artery (LAD) in a sudden death of a drug user is discussed, considering the relevance of autoptic, toxicological and histopathological results.

2. Case report

A 37-year-old man (weight 95 kg), known to the Pathological Addiction Services for a history of drug abuse (cannabis derivatives, cocaine, ketamine) was found dead in his house. There were no anamnesis data indicating familial cardiac abnormalities. After scene investigation and external cadaveric inspection, hypothesizing a toxic-related death (Italian Penal Code, art. 586, death or injuries as consequence of other crime), the Public Prosecutor requested an autopsy to assess the role of drugs of abuse as cause of death. A complete autopsy was performed following the Association for European Cardiovascular Pathology's Guidelines for autopsy investigation of sudden cardiac death [6]. During the exam, samples from tissues, organs and biologic fluids where collected to perform histologic and toxicologic analysis.

Histologic analyses were performed after sample paraffin fixation

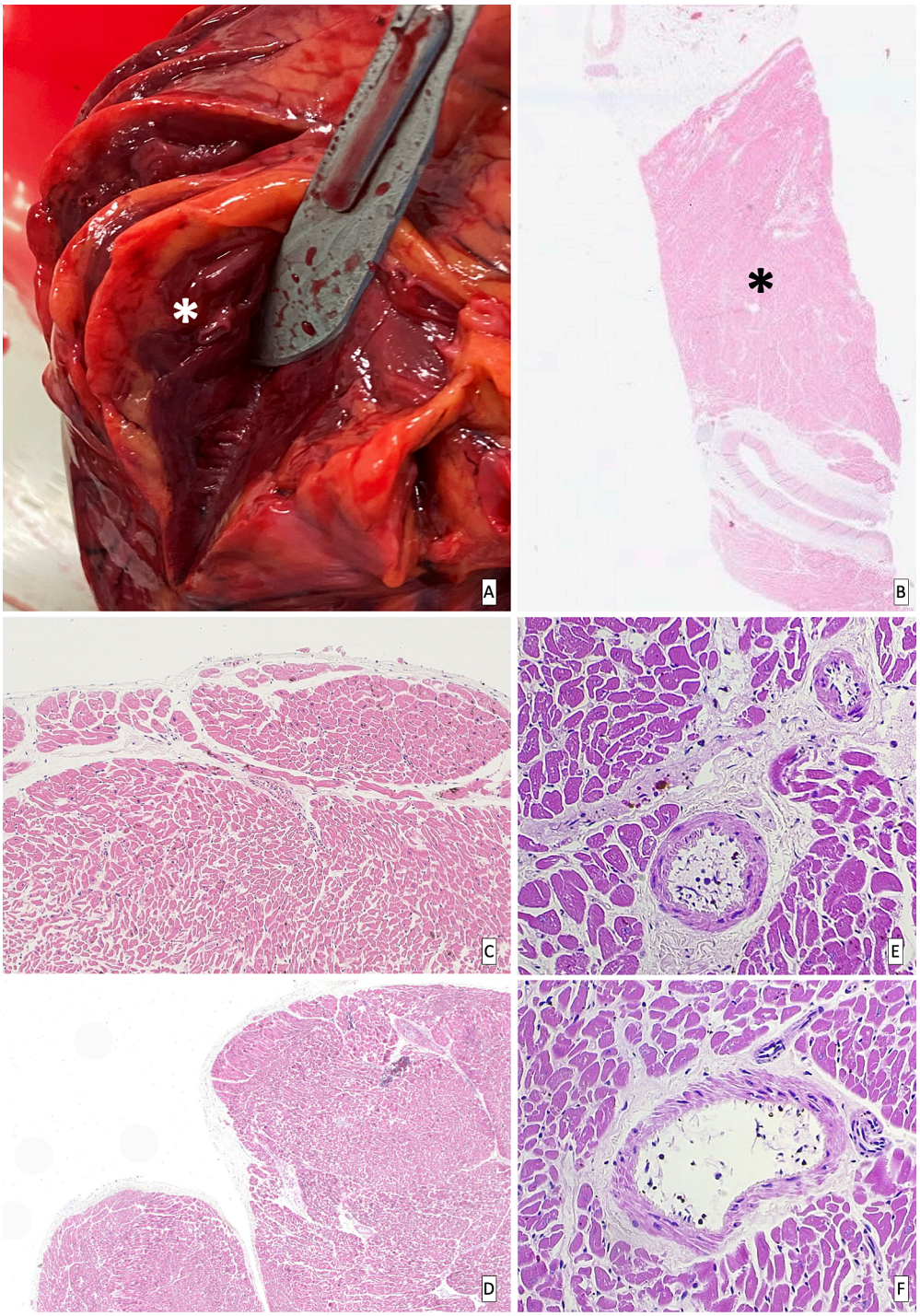

Fig. 1. Myocardial bridge histology. A) Macroscopic picture of the heart and of the LAD's myocardial bridge (0.5 cm deepness); B) histologic picture (hematoxylin-eosine) of the LAD'S myocardial bridge and its distance from the epicardium (5× magnification); C-D) histologic pictures (hematoxylin-eosine) of anterior subendocardial myocardium (C: 10× magnification; D: 5× magnification); E-F) histologic pictures (hematoxylin-eosine) of the myocardium (20× magnification).

and hematoxylin-eosine coloration. Toxicologic analyses were performed on peripheral blood and keratinous matrix (hair), using GC–MS and UPLC-MS/MS, searching for traditional drugs of abuse and New Psychoactive Substances (NPS) [8–10]. Alcohol testing was performed on peripheral blood using GC-FID.

During scene investigation, no drugs or signs of previous consumption of drugs were found. The body was found lying in supine position on the floor of his bedroom, presenting leakage of a red-brown liquid from nasal orifices during head handling.

The autoptic examination revealed venous scarrings on the right forearm and multiple recent acupuncture signs, probably resulting from previous endovenous drugs administration. Lungs were increased in weight (right: 740 g; left: 680 g) and appeared with a smooth and regular red–purple surface, with diffuse purple black dyschromic areas. Lung parenchyma appeared of a red–purple color and congested, with an increased consistence. The heart had slightly increased weight (480 g) and regular morphology. Heart examination revealed the presence of a 0.5 cm deep intramyocardial bridging of a 1.5 cm long segment of the LAD (Fig. 1**a, b)**, with no atheromatic plaques of the coronary circulation.

Histo-pathologic examination confirmed the intramyocardial bridging (Fig. 1**b)**. No fibrosis or signs of ischemia were found in the region supplied by the LAD or in other samples taken from different regions of the myocardium **(**Fig. 1**c-f)**. Histology of the lungs showed diffuse alveolar hemorrhage (DAH) (Fig. 2**c-d)**, characterized by diffuse areas of edema and *endo*-alveolar erythrocyte and multiple alveolar macrophages containing brownish pigments (Fig. 2**a-b**).

Results of toxicologic analysis are summarized in Table 1 and demonstrate a recent use of ketamine and cannabis derivatives in a subject with a history of previous abuse of cocaine, MDMA and ketamine.

Table 1
Results of toxicologic analysis.

Matrix	Instrumentation	Results
Peripheral blood	UPLC-MS/MS [8]	Ketamine and norketamine < LLOQ (0.5 ng/mL)
Peripheral blood	GC–MS [10]	Δ-9-THC 8.8 ng/mL 11-OH-THC 2.2 ng/mL
Peripheral blood	GC-FID	Alcohol negative (LLOQ 0.05 g/L)
Keratinous matrix	UHPLC-MS/MS	Cocaine, benzoylecgonine, ecgonine methylester, cocaethylene, MDMA, MDA, ketamine, norketamine [11]

3. Discussion

In the presented case, relevant autopsy findings in evaluating the cause of death included a myocardial bridge of the left anterior descending artery (LAD) and acute diffuse alveolar hemorrhage (DAH). Toxicological analysis of post-mortem blood detected cannabis active metabolites and a very low concentration of ketamine. No other potential cardiac or non-cardiac causes of death were identified. The heart weight in relation to body weight is within the normal range, and no evidence of previous cardiac ischemia has been identified. Similarly, we have no information regarding any previous medical conditions of the

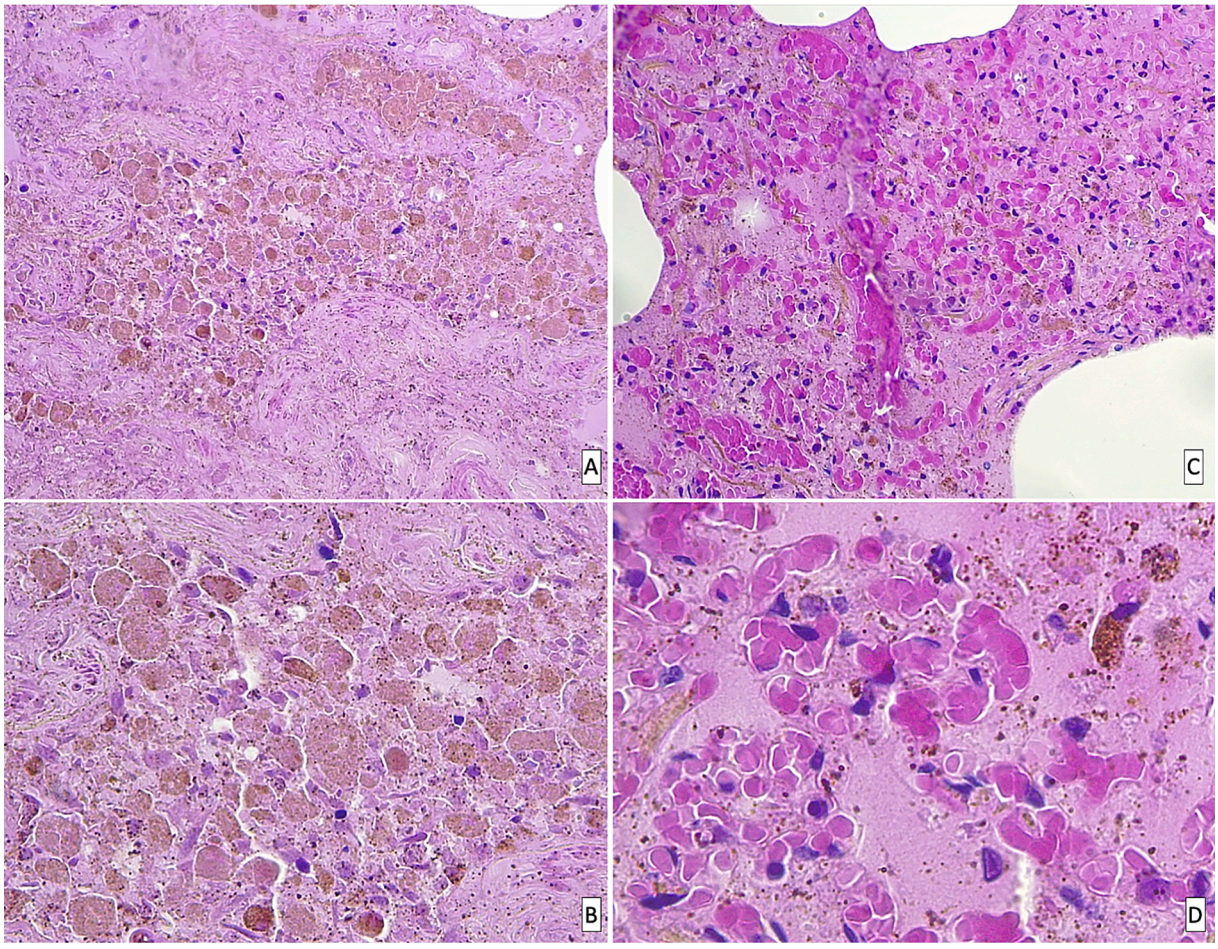

Fig. 2. Lungs tissues histology. A-B) histologic pictures of lungs tissues showing pigmented macrophages (A: 20× magnification; B: 40× magnification); C-D) histologic pictures of the lungs tissue showing alveolar hemorrhage (C: 20× magnification; D: 60× magnification).

subject, who appeared to be in good health.

A myocardial bridge is a congenital anomaly consisting in an intramyocardial segment of an epicardial coronary artery. It is more often located on the LAD, presenting variable prevalences in literature: pathologic studies have found a 25 % prevalence (5 – 86 %), similarly to non-invasive imaging studies using coronary-CT [12]. Ishii et al. [13] reported an autoptic prevalence of LAD bridges of 50 %. Angiographic studies, though, have found a myocardial bridge prevalence of 1.7 % (0.5 – 16 %) [14]. Myocardial bridge can be classified in superficial (depth: 1–2 mm) or deep (depth: > 2 mm) and by length of the involved artery segment. Grassi et al. [15] reported an average depth of 1.23 ± 1.32 mm and an average bridge length of 14.64 ± 9.03 mm. Most of the myocardial bridges have low clinical significance, but deep bridges can induce myocardial ischemia, coronary thrombosis, myocardial infarction, stress cardiomyopathy and sudden cardiac death [15] due to prolonging systolic coronary constriction even after the beginning of the diastolic phase. This is facilitated by increases in adrenergic tone and by myocardial bridge depth [16].

The myocardial bridge found in the present case could be classified as "deep" (maximum depth: 5 mm) potentially inducing cardiovascular complication. The Association for European Cardiovascular Pathology's guidelines define myocardial bridge as an "uncertain" cause of sudden cardiac death, to consider only after the exclusion of other possible diagnosis, thus needing a complete evaluation of every finding of the single case [6].

It also has to be considered that neither macroscopic nor microscopic findings revealed aspects of acute or chronic myocardial ischemia in the areas supplied by the LAD. Therefore, the bridge must be evaluated in the context of severe pathologic pulmonary post-mortem findings that are described after systemic or inhalatory administration of many drugs, through a lung irritative and inflammatory mechanism that, histologically, shows a massive protein-rich alveolar edema and microhemorrhages, as in the reported case [4]. Lung edema can be a cardiac edema, a drug-induced edema, or a combination of both, which together may lead to cardiac death. On the other hand, the lung findings were not associated with any other macroscopic alteration that can induce a mechanical rupture of the alveolus-capillary membrane, such as vasculitic diseases, infections, pulmonary embolism, sarcoidosis, cancer or heart failure [17].

However, even if pulmonary edema has been observed in a high percentage (82.3 %) of ketamine related deaths [2], ketamine alone cannot be directly linked to death in our case, as there is evidence of extensive metabolism after consumption, with post-mortem concentrations being below the limit of detection (LOD) of the toxicological method used (0.5 ng/mL). Minimum lethal post-mortem blood concentrations of ketamine reported in literature are 107 ng/mL in case of sole ketamine intake and 10 ng/mL in case of intake in association with other drugs [1–3]. Low concentrations found in our case may be related to ketamine pharmacokinetics: after endovenous administration (as revealed, in this case, from venous scarring and acupuncture signs on the limbs), ketamine rapidly distributes itself to the tissues, with a half-life in adults of 2–4 h, and plasmatic concentrations cease to be detectable after a day since the administration [3]. Toxicological evidence indicates that in the present case ketamine was taken several hours before death, and probably within a time frame during which cannabis was also used, as demonstrated by cannabinoids blood concentrations. Nevertheless, the absence of cannabis products at the death scene could indicate that it was removed by others, although there is no judicial evidence to support this hypothesis. Post-mortem THC and 11-OH-THC blood concentrations reflect a recent cannabis intake, as these compounds reach a plasma peak within 3 to 10 min followed by a drastic drop in concentrations within the first 2 h [18], probably taken by inhalation, as indicated by the presence of pigmented alveolar macrophages [4,19]. Cannabis smoking is also associated to respiratory damages, such as airways inflammation, protein-rich pulmonary edema, and increases in alveolar permeability, possibly determining damages to the alveolus-capillary membrane and increasing the risk of alveolar macrophage exudation and DAH [20,21]. The blood concentrations of THC and 11-OH-THC (8.8 and 2.2 ng/mL respectively) overlaps with concentrations normally found in people who consume moderate doses of cannabis [22] or people death for other causeses [23] but have been found also in the few cases of cannabis related deaths reported in literature [5]. A recent review published by Drummer et al [5] collected 13 cases linking recent use of cannabis to sudden death. Most deaths occurred in subjects with coronary artery disease, but in some cases no cardiac abnormalities were found, or no autopsy was performed. Other cases described sudden death or myocardial infarction occurred after cannabis use in patients with a myocardial bridge [24–27], suggesting the possible contribution of cannabis use and cardiac alteration in causing the death.

The absence of fibrosis or signs of previous ischemia indicates a lack of prior episodes of myocardial ischemia. However, the hypothesis that the bridge, in the context of drug use, may have caused an acute reduction in oxygen supply and contributed to the subject's rapid death cannot be excluded. Under this hypothesis, it was not possible to rule out the contributory role of the bridge in the causation of death, also considering that drug addiction can be more dangerous in cases with an (unknown) myocardial bridging.

The analysis of forensic literature and the comprehensive evaluation of all data from the post-mortem examination allowed for the identification of the potential role of the myocardial bridge, in conjunction with the recent use of drugs, as the cause of death. However, given the current state of knowledge, the uncertainty in interpreting both coronary anomalies and toxicological results does not allow for the quantification of the pathogenic role of each contributing factor, which could have significant legal implications.

This case highlights the need for an integrated autoptic and histopathological approach in drug-related deaths, to avoid misclassifications that may result using only investigative findings, comprehensive toxicology, and an external examination in place of a standard autopsy, a procedure suggested by medical examiner or coroner offices in those countries facing a shortage of forensic pathologists [28]. This report could also contribute to the body of evidence in the literature and provide insights into the potential contributory role of uncertain conditions, namely cannabis consumption and myocardial bridge, in sudden or unexpected deaths.

4. Conclusions

This case illustrates that identifying the cause of death in situations involving uncertain anatomical variants and post-mortem blood concentrations of toxic substances that are not clearly lethal can be approached through an integrated data analysis conducted by the forensic pathologist, ideally as part of a sudden death interprofessional team. Reports of similar cases expand the scientific literature on this topic and could be valuable in the future for reducing uncertainty in determining the cause of death in comparable situations.

Ethics Approval and Consent to Participate

The current Italian legislation neither requires the family's consent or ethical approval for a single case, as long as the data are kept strictly anonymous. Because summoning the parents was not possible as it would badly interfere with the grieving process, parents' consent was waived, according to the Italian Authority of Privacy and Data Protection ("Garante della Privacy": GDPR nr 679/2016; 9/2016 and recent law addition number 424/ 19th of July 2018; https://www.garanteprivacy. it).

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Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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