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Case Report

Fatal Cardiac Tamponade After Central Venous Catheterization: Autopsy Evidence of Guidewire-Induced Right Ventricular Perforation

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Abstract

Cardiac tamponade due to iatrogenic cardiac perforation is a rare but potentially fatal complication of central venous catheterization, with relevant clinical and forensic implications. Its onset may be sudden, and the exact mechanism may remain unclear without post-mortem examination. We report two autopsy-based cases of fatal hemopericardium following central venous catheter placement. The first involved a 65-year-old man undergoing guidewire-assisted replacement of a tunneled hemodialysis catheter in the right internal jugular vein. During the procedure, sudden hypotension and loss of consciousness occurred, followed by recurrent cardiac arrest, pericardial drainage, emergency sternotomy, and death. The second involved a 94-year-old woman who developed cardiac arrest immediately after placement of a central venous catheter in the right subclavian vein for prolonged intravenous therapy. In both cases, autopsy revealed hemopericardium and a full-thickness perforation of the right ventricular apex. The findings were most consistent with an acute mechanical injury, with transient excessive guidewire advancement during catheter insertion considered the most plausible mechanism. These cases highlight that an apparently appropriate final catheter position does not necessarily exclude an insertion-phase cardiac injury.

Keywords: central venous catheter; cardiac tamponade; hemopericardium; right ventricular perforation; guidewire; forensic pathology

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1. Introduction

Central venous catheters (CVCs) are commonly used for administration of fluids, drugs, total parenteral nutrition, chemotherapy, hemodialysis, and for monitoring. Their placement is associated with mechanical complications, some of which may be rapidly fatal [1]. Among the rarest and most devastating complications is cardiac perforation, leading to hemopericardium and acute cardiac tamponade. Although uncommon, this event has long been recognized as a highly lethal iatrogenic complication, often characterized by sudden clinical deterioration and, in some cases, delayed or missed ante-mortem diagnosis [2]. The underlying mechanisms are heterogeneous and include direct trauma from the guidewire or catheter during insertion, intracardiac mispositioning of the catheter

tip, persistent contact of the tip against the vascular or cardiac wall with late erosion of the wall, and delayed migration of the catheter with subsequent infusion into the pericardial sac. This complication may occur immediately after catheterization or may develop after an initially uneventful placement, thereby increasing the risk of diagnostic delay. In this context, correct tip placement and careful post-procedural verification remain the main preventive measures, particularly to avoid intracardiac placement and unfavorable tip orientation [3].

From a forensic perspective, catheter-related cardiac tamponade is particularly relevant because the diagnosis may occasionally be established only at autopsy, through demonstration of hemopericardium and identification of the perforation site. Accordingly, autopsy-based case reports may help clarify the mechanism and assess causation and preventability in potentially avoidable iatrogenic fatalities [1,4,5]. At the same time, when adequate antemortem imaging, procedural documentation, and clinical records are available, these sources may substantially contribute to the forensic assessment of therapy-related deaths and reduce the need for extensive post-mortem investigation. The present cases illustrate how the integration of the procedural circumstances, clinical course, and post-mortem findings can assist in reconstructing the mechanism of fatal catheter-related cardiac perforation and in distinguishing acute insertion-related injury from other potential mechanisms of catheter-associated cardiac tamponade.

2. Case Report

2.1. Case 1

A 65-year-old man with end-stage renal disease undergoing hemodialysis three times a week was admitted for elective replacement of a tunneled Tesio catheter in the right internal jugular vein due to its displacement from the skin. His past medical history included hypertension, peripheral arterial disease, secondary hyperparathyroidism, and severe ischemic heart disease, including previous myocardial infarction, chronic coronary artery disease, left ventricular aneurysmatic remodelling, and reduced left ventricular ejection fraction. During the guidewire-assisted replacement procedure, no difficulties were encountered with the first catheter, but the second catheter failed to advance sufficiently. Therefore, the guidewire of the first catheter was used to insert a double-lumen dialysis catheter. Although the newly inserted catheter was reported to work properly, the patient developed sudden hypotension and loss of consciousness prior to tunneling. The position of the catheter was not checked by imaging immediately after placement. Iatrogenic cardiac tamponade was immediately suspected. Echocardiography demonstrated a large pericardial effusion, and emergency pericardial drainage extracted about 200 mL of blood, with transient hemodynamic recovery. This was followed by recurrent episodes of pulseless electrical activity and cardiac arrest, rapid accumulation of hemopericardium, severe anemia, and progressive hemorrhagic shock. Bilateral pleural drainage, transfusions, vasoactive support, and emergency sternotomy were performed; approximately 1200 mL of blood was evacuated from the pericardial sac. No active bleeding from the heart and no cardiac perforation were recognized during the intervention, and no attempt was therefore made to repair the lesion. The catheter was left in situ. No further echocardiographic or CT examinations were performed after the intervention. Approximately two hours after the evacuation of the pericardial blood, the patient deteriorated again, required resuscitation, and died. The exact duration of the procedure was not available in the documentation reviewed. Post-mortem examination revealed the effects of multiple emergency procedures, including median sternotomy and bilateral thoracic tubes. Approximately 500 mL of residual blood was present within the pericardial sac, and both pleural cavities contained about 200 mL of serosanguineous fluid. Pericardial blood hematocrit and hemoglobin

levels were not measured. The heart weighed 675 g. A full-thickness punctate perforation with irregular margins and perifocal hemorrhagic infiltration was identified at the apex of the right ventricle, at the junction of the acute and obtuse margins (Figure 1A). Probing demonstrated a transmural tract extending through the epicardium, myocardium, and endocardium to the right ventricular lumen (Figure 1B). The catheter was found to be in an appropriate final position, anatomically distant from the right ventricular apical perforation.

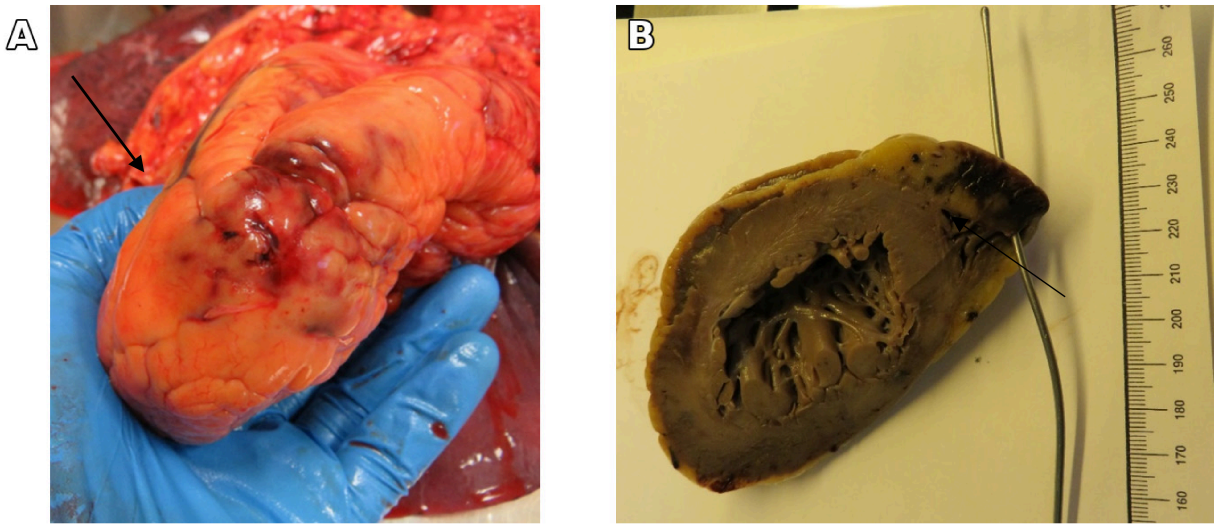


Figure 1. Case 1. (**A**) Full-thickness perforation of the right ventricle (black arrow), with irregular margins and perifocal hemorrhagic infiltration, located at the apex at the junction of the acute and obtuse margins. (**B**) Examination of the cardiac section following treatment with formalin, confirming a full-thickness perforation of the right ventricle (black arrow).

Histological examination confirmed a perforating lesion of the right ventricular apex with hemorrhagic infiltration of the adjacent subepicardial adipose tissue. The right ventricular wall measured 2 mm in thickness, whereas the left ventricular wall measured 16 mm and was mildly hypertrophic. The heart also exhibited severe chronic post-infarction ischemic cardiomyopathy, including an extensive posterior aneurysmal scar, a more recent anterolateral myocardial infarction, severe coronary atherosclerosis, and advanced nephrosclerosis. Overall, the clinicopathologic findings were consistent with fatal cardiac tamponade secondary to iatrogenic perforation of the right ventricle during guidewire-assisted replacement of a tunneled hemodialysis catheter. The morphology and location of the lesion, together with the sudden hemodynamic deterioration during catheterization, were considered most consistent with acute mechanical injury during the insertion procedure. In the absence of contemporaneous documentation of guidewire depth, transient excessive guidewire advancement into the right ventricle was considered the most plausible mechanism. The appropriate final catheter position did not exclude an insertion-related injury, particularly given the apical location of the perforation and the absence of a direct anatomical relationship between the catheter tip and the cardiac lesion. These findings support the possibility of a transient guidewire-induced injury occurring during catheter insertion rather than perforation by the catheter itself.

2.2. Case 2

A 94-year-old woman was hospitalized after a right hip fracture caused by a fall. She initially underwent fracture fixation; then, because of a subsequent wound infection, the implant was removed, followed by bipolar hemiarthroplasty, revision surgery, and later replacement of mobile prosthetic components. She had been receiving prolonged antibiotic therapy. In order to facilitate continued intravenous treatment, a central venous catheter in

the right subclavian vein was positioned. Shortly before the procedure, she was conscious and responsive. The procedure reportedly lasted approximately 10 min. Immediately after catheter placement, the patient developed cardiac arrest. Cardiopulmonary resuscitation was continued for approximately 45 min but was unsuccessful, and death was declared thereafter. Post-mortem computed tomography (PMCT) was performed before autopsy, documenting the position and course of the central venous catheter prior to any potential post-mortem manipulation. At autopsy, a central venous catheter was present in the right infraclavicular region, with adjacent subcutaneous hemorrhagic infiltration. The pericardial sac contained 270 mL of dark red blood, partly fluid and partly clotted, consistent with hemopericardium and cardiac tamponade. Pericardial fluid hematocrit and hemoglobin levels were not measured. The heart weighed 330 g. The left ventricular wall measured 16 mm in thickness and was mildly hypertrophic, whereas the right ventricular wall was within the normal thickness range. At the apex of the right ventricle, at the transition between the anterior and posterior wall, there was an irregular epicardial defect approximately 0.5 × 0.2 cm with mild surrounding hemorrhagic infiltration (Figure 2A). A corresponding irregular hemorrhagic defect was identified on the endocardial side of the right ventricle, with demonstrable continuity between the two lesions, indicating a full-thickness perforation of the right ventricular wall (Figure 2B). No histological examination of the perforation was performed.

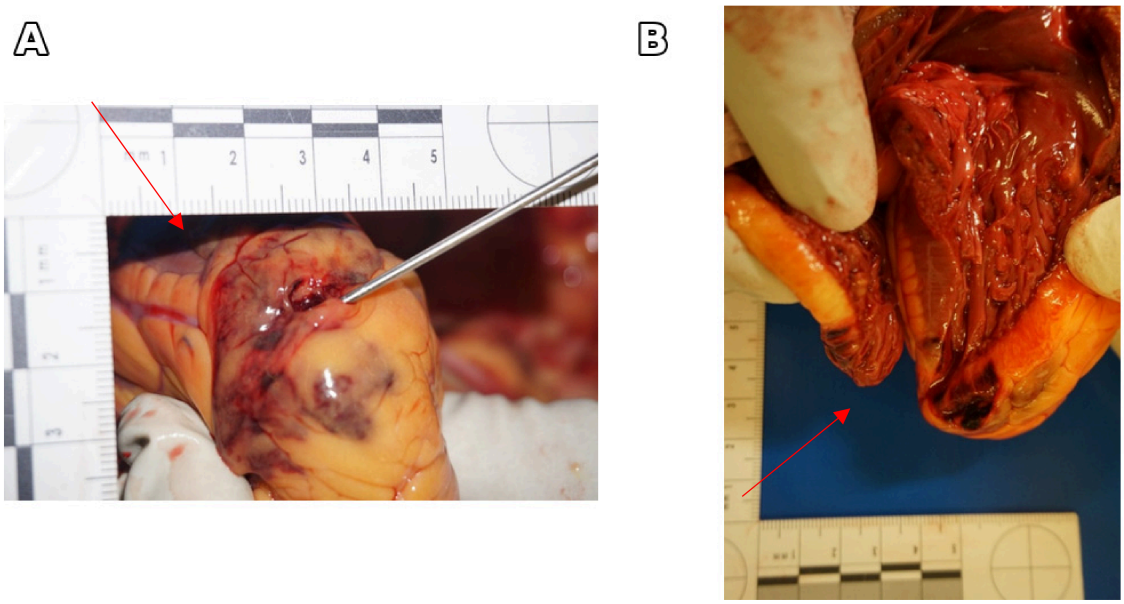


Figure 2. Case 2. (**A**) Full-thickness perforation of the right ventricle (red arrow), located at the apex at the transition between the anterior and posterior wall, with surrounding hemorrhagic infiltration. (**B**) Corresponding irregular hemorrhagic defect on the endocardial side of the right ventricle (red arrow), extending from the defect illustrated in (**A**), with demonstrable continuity between the two lesions.

At autopsy, the course of the central venous catheter could be followed from the right subclavian vein through the superior vena cava, with the catheter tip located at the venous-atrial junction. Additional findings included bilateral pulmonary edema with marked emphysematous hyperinflation, left-sided pleural effusion, mild cerebral edema, severe three-vessel coronary atherosclerosis, previous myocardial scarring, chronic congestive changes, and chronic kidney disease. Overall, the autopsy findings were consistent with fatal cardiac tamponade due to an acute iatrogenic perforation of the right ventricle during central venous catheter placement. Although the catheter tip was found to be

in the appropriate position, the morphology and location of the right ventricular defect, together with the immediate post-procedural collapse, were considered most consistent with an acute mechanical perforation occurring during catheter insertion. In the absence of contemporaneous documentation of guidewire depth, transient excessive advancement of the guidewire was considered the most plausible mechanism. The final catheter tip was therefore anatomically distant from the site of right ventricular perforation, with no direct correspondence between the final catheter position and the myocardial lesion. This finding supports an insertion-related injury occurring during guidewire advancement rather than perforation caused by the final catheter position. The extensive pre-existing cardiac, pulmonary, and renal disease likely reduced the patient's physiological reserve and negatively influenced the fatal outcome but did not provide an alternative explanation for the mechanism of death.

3. Discussion

Central venous catheter-related cardiac tamponade is uncommon but remains one of the most catastrophic complications of venous access [6]. The pathogenesis of CVC-related tamponade is heterogeneous. Early events are usually attributed to direct perforation by the guidewire, dilator, or catheter during insertion, whereas late events are more often linked to persistent tip abutment, erosion of the vascular or cardiac wall, catheter migration, or intrapericardial infusion [7–9]. The literature includes fatal guidewire-related perforations during jugular or dialysis catheterization [8,10], perforation despite the use of a soft J-tipped wire in a structurally vulnerable right ventricle [11], and late tamponade after apparently uneventful placement or from later catheter migration [2,5,9]. The two cases presented fit more closely with an acute mechanical mechanism than with delayed erosion. In both cases, hemodynamic collapse occurred during or immediately after the procedure, and autopsy demonstrated hemopericardium associated with a transmural defect of the right ventricular apex. In the first case, histology demonstrated hemorrhagic infiltration adjacent to the perforation, but no specific cellular reaction allowing more precise histological dating of the lesion was documented. Therefore, the acute nature of the injury was inferred primarily from the temporal relationship with the catheterization procedure, the morphology and location of the perforation, and the clinical course. In the second case, PMCT performed before autopsy documented the catheter position prior to dissection, strengthening the reliability of the finding that the final catheter tip was located at the venous–atrial junction. The right ventricular perforation was instead located at the apex, with no direct anatomical correspondence between the final catheter position and the site of myocardial injury. Alternative mechanisms, including direct perforation by the dilator or catheter during insertion, cannot be completely excluded. However, the immediate onset of cardiac arrest following catheter placement favors an acute insertion-related injury rather than delayed catheter-related erosion or migration. Taken together, the temporal sequence, hemopericardium, full-thickness apical perforation, and absence of another anatomical source of bleeding support an acute mechanical injury occurring during catheter insertion. Among the possible mechanisms, transient excessive advancement of the guidewire was considered the most plausible. In that respect, our observations are more consistent with the early guidewire-mediated mechanism described in case reports by Cavatorta et al., Lee et al., and Hiroshima et al. than with the delayed erosion model [8,10,11]. The issue of tip position deserves a nuanced interpretation. For standard CVCs, correct tip positioning and avoidance of acute contact with the vessel wall are important preventive measures [4,12]. These considerations concern the final catheter position and should be distinguished from the separate risk of uncontrolled guidewire advancement during insertion.

Schuster et al. proposed the carina as a simple and reliable radiographic landmark for correct placement [13], and Stonelake and Bodenham further showed that, particularly for left-sided catheters, a position below the carina is more likely to be satisfactory considering both depth and angulation [14]. Mahlon and Yoon provided a complementary anatomical basis by defining normative dimensions of the superior vena cava and the venous–atrial junction area on CT angiography [15]. At the same time, Pittiruti and Lamperti questioned the simplistic dogma that any right atrial tip automatically explains late tamponade in adults [2]. These apparently divergent observations are not contradictory. Rather, they suggest that final tip position should be interpreted in relation to catheter type and function, while remaining distinct from the separate problem of uncontrolled guidewire advancement during insertion.

From a practical standpoint, the primary preventive message is therefore broader than simple post-procedural tip confirmation. Prevention requires control of the entire insertion process, including careful attention to guidewire depth, avoidance of forceful advancement, prompt withdrawal of the guidewire when cardiac irritation is suspected, and verification of catheter position. Although no single guidewire depth can be considered universally safe for all patients and access routes, published data support limiting guidewire advancement to approximately 18 cm for right-sided approaches [16–18], while insertion to 20 cm has been associated with an increased incidence of guidewire-induced arrhythmia compared with 15 or 17.5 cm [19]. These values should be regarded as practical safety thresholds rather than absolute universal limits, given the influence of vascular access route, patient anatomy, device characteristics, and procedural technique. Unexpected resistance should be regarded as a procedural warning sign rather than an indication to apply additional force; the guidewire should not be advanced against resistance. New-onset atrial or ventricular ectopy, tachyarrhythmia, or other ECG changes during guidewire advancement should prompt immediate reassessment and withdrawal of the guidewire until its position is clarified. Sudden hypotension, loss of consciousness, or acute hemodynamic deterioration should be considered major warning signs requiring immediate interruption of the procedure and evaluation for mechanical complications, including cardiac perforation and tamponade. In the present cases, the exact depth of guidewire advancement was not documented and therefore cannot be retrospectively quantified. No specific arrhythmia or resistance during guidewire advancement was documented in the available procedural records.

These considerations are also consistent with current vascular access safety guidance, which emphasizes standardized insertion practices, meticulous procedural documentation, real-time ultrasound guidance for central venous access, appropriate catheter tip positioning, and early recognition of complications [20]. Our cases also suggest that patient-related factors may modulate the perforation threshold without constituting an independent cause of death. In the first patient, severe chronic ischemic cardiomyopathy and marked thinning of the right ventricular wall likely reduced tissue resistance to mechanical injury. In the second, extreme age and advanced cardiac, pulmonary, and renal disease reduced physiological reserve and worsened tolerance to tamponade and hemorrhagic collapse. Hiroshima et al. described a similar interaction between guidewire trauma and structurally vulnerable myocardium, reinforcing the concept that severe pre-existing disease may facilitate perforation while not replacing the iatrogenic mechanism itself [11].

From a forensic perspective, these two cases illustrate the contribution of autopsy in suspected catheter-related deaths. dos Santos Modelli and Cavalcanti reported one early and one late fatal tamponade, both diagnosed only at cadaveric examination [5], while Valente-Aguiar et al. similarly showed how post-mortem investigation can demonstrate the lethal mechanism in otherwise clinically elusive iatrogenic deaths [1]. In the present

cases, autopsy provided direct morphological confirmation of hemopericardium and right ventricular perforation, while also helping to reconstruct the causal sequence, distinguish acute injury resulting from a medical procedure from the underlying chronic disease, and rule out alternative sources of fatal hemorrhage. However, autopsy should be considered within the broader framework of the medico-legal investigation rather than as invariably indispensable. When sufficient antemortem imaging, procedural documentation, and medical records are available, these sources may substantially contribute to the forensic assessment of therapy-related deaths and may reduce the need for extensive post-mortem examination. In Case 2, PMCT performed before autopsy documented the catheter position prior to dissection, strengthening the assessment of its final position. In Case 1, no imaging was performed immediately after catheter placement, and no further echocardiographic or CT examination was performed after the intervention. No additional antemortem or post-mortem imaging or photographic documentation of the catheter position was available for Case 1. This represents a limitation of the investigation and prevents independent radiological confirmation of the catheter position and its anatomical relationship to the cardiac lesion.

The present investigation has several methodological limitations. In Case 1, no post-mortem CT or other imaging documenting the catheter position was available, and no photographic documentation of the catheter position could be retrieved retrospectively. In Case 2, although PMCT documented the final catheter position before autopsy, histological examination of the perforation was not performed. These limitations should be considered when interpreting the mechanism of injury. Nevertheless, the immediate temporal relationship between catheterization and clinical deterioration, together with the autopsy demonstration of hemopericardium and full-thickness right ventricular perforation, supports the reconstruction of an acute mechanical injury related to the insertion procedure. Accordingly, the interpretation of the catheter position and the reconstruction of the mechanism should be based on the integration of imaging, procedural, clinical, and post-mortem findings rather than on the final catheter position alone.

An additional medico-legal consideration concerns the classification of deaths resulting from medical treatment. In jurisdictions that recognize "therapeutic complication" as a manner of death, this designation is used for fatalities resulting from predictable complications of appropriate medical therapy and should be distinguished from medical malpractice. The occurrence of a therapeutic complication does not, in itself, establish that a procedure was performed negligently. Rather, the medico-legal assessment requires a separate evaluation of whether the intervention was appropriately indicated, whether it was performed in accordance with accepted professional standards and available safety recommendations, and whether any deviation from those standards contributed causally to the fatal outcome [16,17].

In the present cases, the post-mortem findings support an acute mechanical perforation of the right ventricle occurring during central venous catheterization. However, the autopsy findings alone cannot establish whether the presumed excessive advancement of the guidewire resulted from a specific departure from accepted procedural practice. Such a determination would require a sufficiently detailed reconstruction of the procedure, including the operator's technique, guidewire handling, procedural documentation, monitoring, and compliance with relevant safety standards. This distinction is particularly important in forensic practice, where establishing the cause and mechanism of death should remain conceptually separate from determining whether professional conduct met the applicable legal or professional standard. The legal assessment of professional liability is also jurisdiction-dependent and may involve additional statutory and jurisprudential criteria beyond the medical assessment of causation and adherence to professional standards.

4. Conclusions

The present cases reinforce that fatal cardiac tamponade after central venous catheterization, although rare, remains a clinically and forensically significant event. In both patients, the temporal sequence, autopsy findings, and lesion morphology supported an acute iatrogenic perforation of the right ventricle, with transient excessive advancement of the guidewire during catheter insertion considered the most plausible mechanism. Pre-existing cardiac and systemic disease likely reduced physiological reserve but did not provide an alternative explanation for death. An apparently acceptable final catheter position does not exclude a transient insertion-phase perforation. The absence of complete procedural imaging and documentation in Case 1 and the lack of histological examination of the perforation in Case 2 represent limitations of the present investigation. Accordingly, post-mortem findings should be interpreted together with the clinical and procedural evidence, particularly when antemortem imaging or procedural documentation is incomplete, to reconstruct the mechanism of death and assess therapy-related fatalities.

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Institutional Review Board Statement: This study was performed from data from human cadavers. This article does not contain any studies with (living) human participants or animals performed by any of the authors. The subjects involved in this study underwent a judicial autopsy in order to identify the cause of death. In Italy, forensic autopsies and related post-mortem investigations that are performed within the framework of a judicial proceeding (either upon prosecutorial order or under the authority of the judiciary) do not require approval by an Institutional Review Board (IRB) or Ethics Committee. Such procedures are conducted under the provisions of the Italian Code of Criminal Procedure (Articles 359–360, 116, 224 et seq.), which govern medico-legal examinations performed for judicial purposes. The case concerns two deceased subjects within a legal/forensic context. Prosecutors opened an investigation, and a judicial autopsy was ordered. Therefore, ethics committee approval was not required. The investigation was conducted in accordance with the principles set forth in the Declaration of Helsinki (1975, as revised in 2013).

Informed Consent Statement: Patient consent was waived. This study was conducted in accordance with international ethical guidelines, including the Declaration of Helsinki. Informed consent for participation is not required as part of a public verdict and per local legislation on deceased subjects [Italian Legislative Decree 10 August 2018, n. 101, Provisions for the implementation of national legislation to the provisions of Regulation (EU) 2016/679 of the European Parliament and of the Council of 27 April 2016, on the protection of individuals with regard to the processing of personal data and on the free movement of such data and repealing Directive 9546/EC.

Data Availability Statement: The original contributions presented in this study are included in the article. Further inquiries can be directed to the corresponding author.

Conflicts of Interest: The authors declare no conflicts of interest.

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