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CASE REPORT

Post-mortem computed tomography differentiation of putrefactive gas and air embolism: A case of traumatic carotid-jugular arteriovenous fistula

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Abstract

Introduction: PMCT is superior to autopsy for identification of intravascular or extravascular gas pockets and their distribution. However, differentiation between air embolism and putrefactive gas can prove challenging due to overlapping imaging findings. *Case Report:* We report a case of a healthy young man who was involved in a fight, sustained a slash wound to the right side of his head by a kitchen knife and died at the scene. Pre-autopsy PMCT demonstrated complex fractures of the right mastoid bone extending to the right petrous apex and jugular bulb, exposing the right sigmoid sinus. There was also asymmetric intravascular air distribution suspicious of air embolism with ancillary findings of traumatic carotid-jugular pseudoaneurysm and arteriovenous fistulous formation. Post-mortem examination revealed a slash wound measuring 12x2 cm at the right side of the head, cutting through the scalp, right temporal bone, right temporal meninges, right sigmoid venous sinus and part of the right occipital lobe. No intracranial haemorrhage was found on both PMCT and autopsy. *Discussion:* PMCT findings of air embolism versus putrefactive air on PMCT are discussed in this case. Detailed history on mechanism, circumstances, time of death and careful analysis of intravascular and extravascular air distribution patterns on PMCT are essential in guiding differentiation of true fatal air embolism and "normal" post-mortem putrefactive air. Needless to say, it is recommended that PMCT be performed as early as possible after death to reduce the chances and presence of artifactual decomposition changes.

Keywords: Air embolism, putrefactive gas, postmortem computed tomography (PMCT)

INTRODUCTION

Air embolism is the ingress of air into veins or arteries and subsequent air diversion to distal sites along the circulatory system.[1,2] It is fatal if there is rapid rate of air entry into large vessels sufficient to cause vascular air-locks, obstruction and ischaemia distally with eventual cardiovascular collapse.[3,4] A high degree of suspicion is needed in cases of barotrauma, blunt or penetrating trauma or iatrogenic injuries caused by surgical interventions or air injections.[4]

Artifactual gas that may be confused with air embolism on post-mortem computed tomography (PMCT) result from putrefactive gas formation that begins soon after death or resuscitation artefacts that have no bearing in ascertaining the cause of death but mimic the appearance of air embolism. The amount and distribution of putrefactive gas are affected by extrinsic factors such as post-mortem interval, surrounding temperature, body exposure and intrinsic factors such as degree of blood loss, obesity, sepsis or underlying comorbidities.[2]

PMCT is superior to autopsy for identification of intravascular or extravascular air pockets and their distribution. However, differentiation between air embolism and putrefactive gas can prove challenging due to overlapping imaging

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findings. Detailed history on mechanism, circumstances and time of death as well as careful analysis of secondary PMCT signs are essential in guiding differentiation as we discuss in our case.

CASE REPORT

A 21-year-old previously healthy young man was involved in a fight, sustained a slash wound to the right side of his head by a kitchen knife and died at the scene. Non-contrast enhanced PMCT using Toshiba Aquilion 64-row detector helical CT scanner was performed prior to autopsy approximately six hours after the time of death. Head and neck were scanned first with these parameters: 135 kVp, 2925 mAs, FOV 320mm^2, Pitch 0.641, slice thickness of 1.0 mm and slice interval of 0.8 mm. Whole-body scan followed with these parameters: 120 kVp, 9080mAs, FOV 500 mm^2, Pitch 0.944, slice thickness of 2.0 mm and slice interval of 1.6 mm. Temporal bone reconstruction with slice thickness of 0.5 mm was included in the protocol.

Post-Mortem Computed Tomography (PMCT) Findings

PMCT demonstrated complex open right mastoid (temporal bone) comminuted fractures secondary to penetrating head trauma with mixed transverse and longitudinal fracture components extending to the right petrous apex and jugular bulb as well as exposing the right sigmoid sinus. No intracranial haemorrhage was appreciated. However, direct air communication between overlying right post-auricular subcutaneous laceration wound and the right sigmoid sinus was observed (Fig. 1a).

A bilobed air filled out-pouching had also formed from an air-filled right jugular bulb (antero-medial) wall measuring 5.2 x 8.6 mm, representing a traumatic pseudoaneurysm. A tortuous fistulous air-filled connection was seen between this pseudo-aneurysm and the petrous internal carotid artery (ICA) measuring 11.2 mm in length and 1.4 mm in width. Generalised, near symmetrical intracranial, neck and subclavian arterial and venous air were also seen (Fig. 1b-c).

At the level of the thorax, pneumatisation of mediastinal vessels were most prominent along the superior vena cava, bilateral brachiocephalic veins and arteries, ascending aorta, arch or aorta and to lesser extent descending thoracic aorta, pulmonary veins and arteries. Significant air was seen in all cardiac chambers, more in the right heart chamber with air-fluid levels within the right atrium. At the level of abdomen,

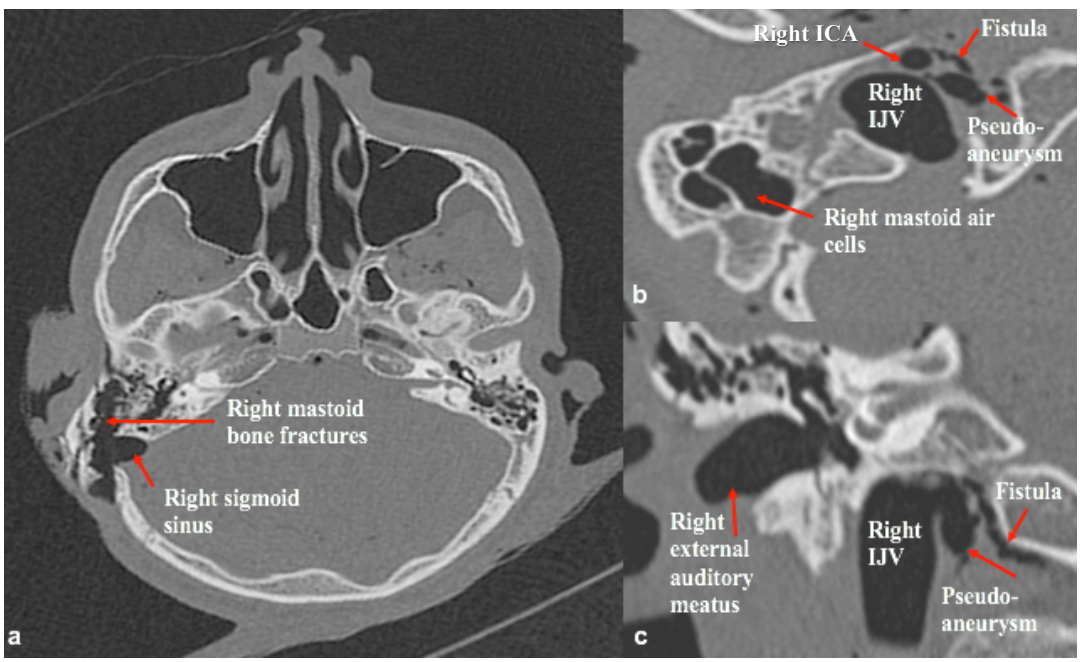

FIG 1. PMCT images showing the **(a)** Axial view of the right temporal (mastoid bone) comminuted fractures exposing the right sigmoid sinus to air, **(b)** axial and **(c)** coronal views of intravascular air within the right internal jugular vein (IJV) and right petrous internal carotid artery (ICA), an air-filled traumatic pseudoaneurysm and fistulous communication between the right IJV and ICA.

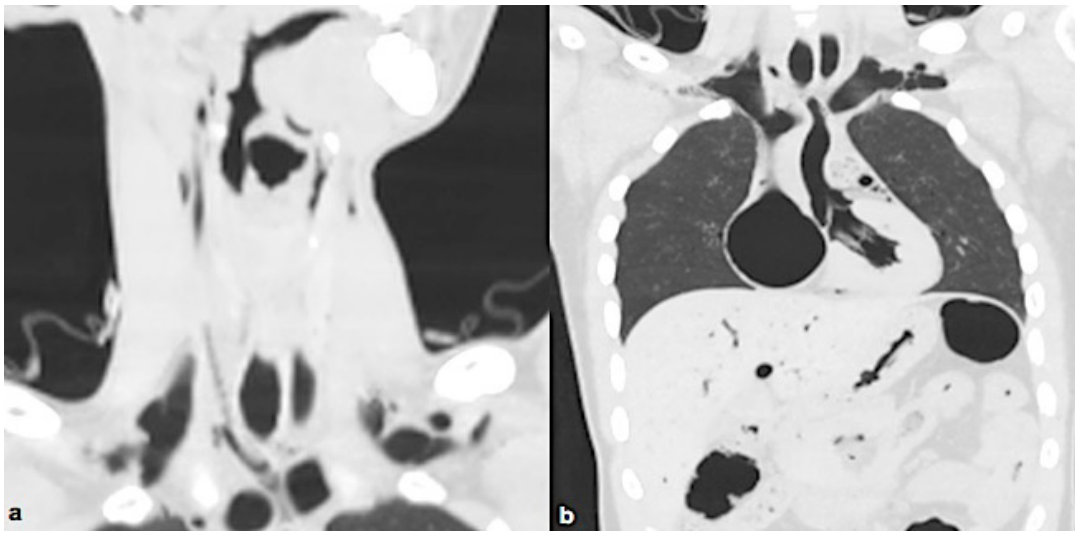

FIG. 2: Pneumatisation of **(a)** large neck **(b)** mediastinal vessels, heart chambers and portomesenteric air.

minimal portomesenteric intravascular air was seen extending into the left liver lobe portal veins. No pneumatosis intestinalis was observed. Minimal intravascular air was also seen along the collapsed/flattened abdominal aorta, common, internal and external iliac arteries. No extravascular air i.e. pneumothorax, pneumomediastinum, pneumoperitoneum or subcutaneous emphysema, external penetrating wounds or internal organ injury were seen elsewhere in the body.

Autopsy Findings

The deceased was thin built with height of 165 cm and weight of 60 kg. The body showed no signs of decomposition on external and internal inspection. On external examination, the deceased was covered in blood with pale conjunctiva and nail-beds as well as faint hypostatic lividity at the back. Examination of the heart showed subendocardial haemorrhage. These were signs of blood loss. No injury over both hands, forearms, or legs indicative of defensive wounds or struggling marks were seen. A slash wound measuring 12 x 2 cm at the right side of head, cutting through the scalp, right temporal bone, meninges, right sigmoid venous sinus and part of right occipital lobe approximately 0.5 cm in size was also seen (Figure 3). There was no

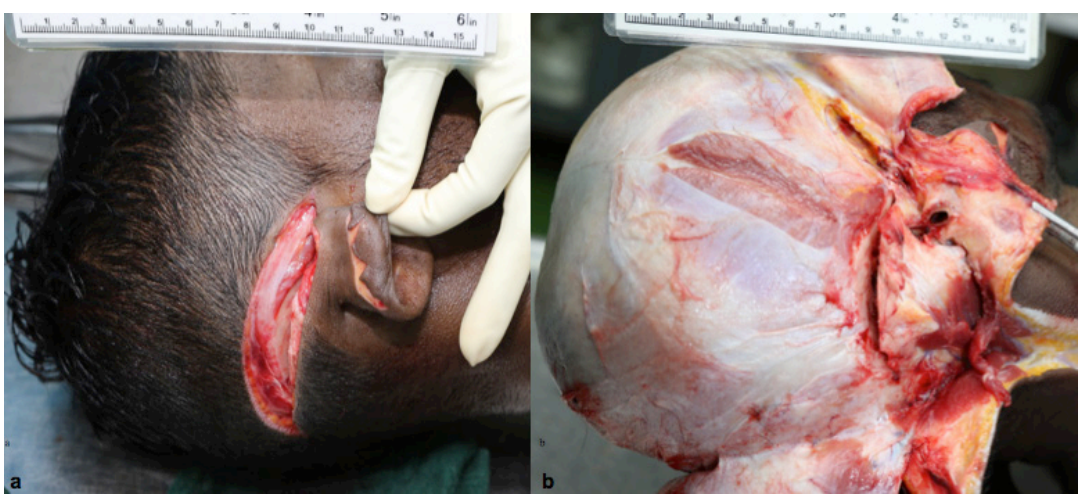

FIG. 3: (a) Slash wound at right temporal region with incised partial thickness wound posterior to the right external ear. (b) Right temporal bone fracture corresponding to PMCT findings.

intracranial hemorrhage or cerebral edema. All other internal organs are unremarkable.

DISCUSSION

The demonstration of intracranial air with PMCT is an important element of medico-legal opinions.[5] We first consider the mechanism of injury that favored fatal air embolism as the ultimate cause of death. The slash wound that caused right mastoid bone (base of skull) fracture introduced a sudden high pressure, allowing rapid air ingress into the exposed entry point i.e. the right sigmoid sinus. Transected vein walls adhere to adjacent tissues, preventing collapse, but create negative intravenous air pressure that produces an air-suction effect.[6] Venous air emboli are then transported to cardiac chambers forming intracardiac air locks, reducing blood flow from the right ventricle into the pulmonary circulation which then lead to rapid asphyxiation due to oxygen deprivation, arrhythmias, sudden drops in blood pressure, shock and sudden death.[7-9] In our case, PMCT demonstrated continuous air passage into the right jugular bulb, systemic veins, pulmonary circulation followed by systemic and peripheral arterial circulation.

Air embolism should be considered when there is significantly increased air within the systemic arteries compared to the pulmonary circulation as seen in this case.[10] Arterial air has been reported to be more fatal compared to venous gas embolism as it can lead to neurologic and cardiovascular collapse.[11] Makino *et al*. suggested that entrance of air within the arterial system could be due to intrapulmonary shunts opened when pulmonary pressure increases.[12] In addition to this, presence of bi-hemispheric intra-arterial air within the Circle of Willis polygon could also be explained by retrograde passage of air via the traumatic pseudoaneurysm and carotico-jugular fistulous communication incidentally found on PMCT. The formation of arteriovenous fistula and pseudoaneurysm likely resulted from traumatic dissection of vessels.[8] Ho *et al*. postulated that up to 2mls of air within the cerebral circulation was sufficient to cause obstruction, ischaemia, hypoxia and ultimate death.[13]

There have been suggestions that PMCT has significant diagnostic value if performed up to 24 hours after death to avoid putrefactive gas formation after death.[14] Putrefaction begins gradually and intravenously after death, before spreading to the arterial system, its rate depending on intrinsic and extrinsic variables and may appear from 3 to 6 hours postmortem.[15] In our case, PMCT was done six hours after death. Underlying diseases such as hepatic abscesses, inflammatory bowel disease, necrotising enterocolitis, cholangitis and mesenteric ischaemia are known to accelerate the rate of putrefaction.[16] However, in this case, the victim was a fit, young man with no prior co-morbids, fever or sepsis. His body was found in a shaded cool area and fully clothed, extrinsic factors to consider in estimating the rate of decomposition. Other than established rigor and livoris, no decomposition changes were noted during autopsy.

Takizawa *et al*. stated that air in deep organs such as the heart, liver and deep abdominal vessels related to the digestive system were more likely attributed to putrefaction.[17] PMCT interpretation was limited by the fact that actual rate of putrefactive gas formation was unknown. Specialised gas composition analysis by means of gas chromatography-mass spectrometry and thermal conductivity were also not available in our facility, but some pathologists have not found this to be helpful, considering confounders such as time between PMCT and autopsy sampling of material, variations in transport and storage.[18] Notwithstanding, the quantity of air within the heart, large mediastinal vessels, neck and head were disproportionately increased compared to air within the liver, abdominal organs and soft tissue fascial planes. Correlating with history and autopsy findings, the non-uniform air distributions inferred that the intravascular air source was likely from a process not solely due to putrefaction.[19]

In conclusion, air embolism is a life-threatening event and an established cause of sudden death. The presence, quantification and passage of air embolism is best evaluated with PMCT but should be interpreted with caution. Differentiation from normal putrefactive intravascular air production can be challenging on imaging alone but is possible as illustrated in this case, when considering the history, mechanism of injury, site of air ingress, pattern of air distribution, decomposition changes and autopsy findings. Needless to say, it is recommended that PMCT to be performed as early as possible to reduce the chances and presence of artifactual decomposition changes.

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Author's contribution: WYL is the main author and coordinator for this case report. AN performed the autopsy and was co-writer for forensic pathology aspect of the case. MK is corresponding author for publication. MK and SSF were tasked for revision of the script, permission and accountability of the case study to ensure accuracy and integrity of the study. All authors read and approved the final version of the manuscript.

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