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The Artifacts of Death. CT Post Mortem Findings.

Running title

Post Mortem CT

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Post-mortem CT plays an increasing role in the evaluation of deceased patients (1) working as an ally with established pathology frameworks (2-4). There are a number of post-mortem specific artefacts that are not normally encountered by clinical radiologists and these have the potential to mimic or obscure underlying pathologies, or result in distraction for the radiologist. An adequate comprehension and appreciation of the 'normal' imaging appearances or so-called artefacts of death is vital to maximise the accuracy and utility of post-mortem CT. The intellectual approach to post-mortem CT is also different to clinical studies. Clinically much of the focus is forward looking, based upon determining what is wrong with the patient now and what can be done i.e. the effect of disease or trauma. Post-mortem imaging is more concerned with looking back to determine how and why did the patient reach this point i.e. the cause. The following review describes the post-mortem imaging process and introduces the common post-mortem imaging artefacts illustrated with representative examples.

CT Technique

At the Victorian Institute of Forensic Medicine (VIFM) the CT protocol is to perform the examination as rapidly as possible to minimise the degree of decompositional changes. The decedent is initially scanned within the body bag in the supine position (where possible) without any attempt to reposition the limbs or head, and has usually been undressed by mortuary technicians unless the case is a suspected homicide. Non-contrast CT is obtained from skull vertex to sternal notch with 1mm reconstructions in both soft tissue and bone kernels. A second scan is then performed from skull vertex to beyond the feet with 1.5mm soft tissue window reconstructions. The studies are then reviewed on a commercially available 3-D workstation. Due to these positioning factors, beam-hardening artefact occurs because of the limbs and interference may be present

due to metal both within and outside the patient (Figure 1). Post processing is therefore important to maximise image quality, especially as the vast majority of scans are performed without any exogenous contrast agents. Following the preliminary CT, the scan may be repeated to reduce these artefacts.

Rotatory subluxation cervical spine

The cervical spine and in particular the craniocervical junction has a highly varied appearance at PMCT based upon patient positioning. Due to the loss of muscle tone coupled with head rotation and variable degrees of extension, subluxation at the craniocervical and atlantoaxial articulations are common(5) (figure 2). This is frequently encountered in non-traumatic cases when it can be dismissed, while in the setting of trauma, a repeat scan with improved radiographic positioning may be necessary. The absence of blood or focal gas around the thecal sac and skull base is also a useful finding that the subluxation is more likely artifactual rather than pathologic.

Livor mortis

Livor mortis, also known as lividity and post-mortem hypostasis, relates to body fluids settling in dependent positions following cessation of cardiovascular circulation. This is evident on inspection of the body by reddish discolouration in the dependent aspect, while on CT is encountered as increased density in the lungs, dependent skin thickening and haematocrit formation in vessels. Haematocrit formation is most easily seen in large chambers, especially the right atrium (figure 3a). The pulmonary shifts of fluid are relatively constrained by the interlobular fissures (figure 3b) and appears as ground glass opacities with a relatively straight margin although early cases will be appreciable as a gradient of increasing density towards dependent aspects (6). It is important to remember that the pattern of lividity is dictated by the positioning of the body at the time of death (figure 3c) and many decedents are found in positions other than supine.

Decomposition

Decomposition refers to the breakdown of tissues and loss of cellular integrity that occurs following death. The two main processes are autolysis and putrefaction. The human body must be maintained within a narrow window of temperature, pH and oxygenation, and once outside this range, putrefaction rapidly commences.

Autolysis is the breakdown of cells by intracellular enzymatic processes with the rate being dictated by temperature (slows in cold conditions) and the concentration of the enzymes (rapid in enzymatically rich tissues such as the pancreas)(7). The fat around the pancreas is therefore frequently irregular which can pass towards the coeliac plexus, rendering it challenging to diagnose acute pancreatitis. A specific example of autolysis is so-called gastromalacia (figure 4a) whereby gastric acids cause the stomach (usually in the fundus where such fluid pools) to perforate leading to free gas and fluid in the peritoneal cavity often without other features of decomposition such as putrefaction. This phenomenon also rarely occurs in the posterior mediastinum (known as oesophagomalacia). To identify these cases it is important to note that free intraperitoneal or mediastinal gas is prominent, and gas is the mesenteric and portal venous systems is relatively minor.

Putrefaction is related to bacterial growth and fermentation (7) being an anaerobic process and first evident in bacteria rich sites, most typically the gut. Upon inspection of the body, putrefaction is first noted by greenish discolouration of the skin in the right iliac fossa, with this related to the close anatomical relationship of the bacteria rich caecum. As an anaerobic process, gas is produced, which with breakdown of mucosal integrity results in pneumatosis, followed by gas within the mesenteric/portal venous system (figure 4b), right heart, body wall (figure 4c) and eventually free peritoneal gas. The rate of putrefaction is highly variable and the gaseous by-products makes it challenging to detect gut ischemia and even pre-mortem hollow viscus perforation. Unusual distributions/locations of gas can raise the suggestion of pre-mortem pathology at that site, but unfortunately unlike in pre-mortem imaging, this is neither sensitive nor specific. The rate of putrefaction may be modified by the body's location, for example being quicker in areas exposed to heat or areas that are well insulated.

More advanced cases of decomposition can develop generalized liquefaction and saponification especially in the thoracic and abdominal cavities. Soaponification is secondary to hydrolysis and hydrogenation of fat and typically occurs in damp environments with enough heat for microorganisms to flourish. This process results in fluid-fluid levels, typically within the peritoneal cavity, that may be missed or be mistaken for blood using standard viewing windows. It is important to measure the Hounsfield density of the components as the dependent aspect will be close to water (0-10) and the anti-dependent aspect having a negative number (figure 4d) in the range of -20 to -100 which with incorrect window settings may be mistaken for air.

The final stage of decomposition is skeletalisation, at which point the soft tissues have decayed, been consumed by animals or insects, or dried to the point that the skeleton is exposed. At this point, medical devices and implants may move or fall out of the body cavity (figure 4e).

Blood

Upon cessation of the circulation, sedimentation occurs with cellular elements becoming dependent relative to serum(8). The non-cellular elements can leak from the vascular system, increasing the apparent haematocrit (6) as the dense iron containing components cluster in the dependent vessel, leading to an increased Housfeld Unit. Clotting and fibrinolysis processes may occur simultaneously (6) and post-mortem clot is most frequently encountered in cardiac chambers and large vessels (figure 5a). These post-mortem clots render it challenging to differentiate ante-mortem thrombosis and emboli. In our experience if the clot conforms to the shape of the vessel it is likely post-mortem, while if it is irregular or cord like then it is more likely, but not invariably, ante-mortem. In cases of haemopericardium, the clot can form a shell around the heart with a separate encasing serum layer (so-called hammer heart)(figure 5b) which is thought to occur when the haemopericardium develops while cardiac pulsation persists as this produces dynamic pressure variation that models the clot. In contrast haemopericardium producing sudden cardiac death tends to layer and form a haematocrit, although unfortunately hammer heart is not a specific sign as pulsation may be provided by well-administered chest compressions or the blood may not clot if the patient is anticoagulated.

Blood beyond the vascular system has 2 distinct patterns. In the peritoneal cavity it tends to remain liquid producing an haematocrit especially in the pelvis and paracolic gutters (figure 5c). Otherwise clotted blood tends to retain its high radio-density despite decomposition and can provide a clue to cause of death. A careful search for the site of haemorrhage may reveal a sentinel clot or a pathologic appearing vessel. The vessels themselves become collapsed after death as the patient no longer has blood pressure. This alters the morphology of vessels, which can become concave while aneurysms may have a lax margin similar to a collapsed rubber band (figure 5d). The absence of intravascular pressure allows fluid and gas to easily track into potential spaces and recesses. A classic example is in patients with haemopericardium who upon collapse of the aorta have blood track into the peri-aortic peri-cardiac recesses. It is important to recognise this as the blood in the recess, coupled with the collapsed wall of

the ascending aorta, can be mistaken as a type A dissection with rupture - the so-called pseudo-dissection sign (figure 5e).

Agonal period

The time over which a patient dies, including the period of resuscitation is referred to as the agonal period and it is vital to remember that changes from extrinsic factors frequently occur during this phase. Examples include resuscitation and other medical interventions, while further changes may occur within the mortuary itself. A classic mortuary fracture is breaking an ankylosed spine during routine decedent handeling. The dynamic intervention around the time of dying needs to be remembered and recognised. Rib fractures in the anterior ribs bilaterally, frequently with a buckle type morphology are regularly encountered in patients who have received chest compressions(9) (figure 6a). Likewise CPR can result in cardiac rupture, which in our experience is typically right atrial or ventricular and usually in patients with massive pulmonary embolism, where the obstructed outflow tract produces high right heart pressure with CPR (figure 6b). Pneumothorax is also an often encountered complication as are cases of pulmonary and upper abdominal visceral laceration/contusion (figure 6c), and a history, along with other patterns of injury can be useful to differentiate resuscitation injuries, from preceding trauma including ventilation. At times though a clear distinction cannot be made. PMCT is far more sensitive and specific for the detection of ectopic gas such as pneumothorax and pneumopericardium compared with autopsy.

Airway intervention may result in dislodged teeth that can be 'aspirated' (figure 6d) and at times large objects such as entire dentures (figure 6e) can become wedged in the areodigestive tract. Various lines, tubes and catheters may be inserted and need to be closely inspected to determine if they are in the correct position (figure 6f).

Gas is frequently encountered in the inguinal fossae and eyes secondary to post-mortem blood and vitreous samples being taken for toxicology purposes. Prominent arterial system gas is encountered in divers who die while submerged. Retrieving the body to the surface reduces pressure and allows the dissolved systemic gases to leave the solution. This can lead to difficulty in diagnosing gas embolism as a cause of death (figure 7).

Lung changes

The pulmonary parenchyma and airways are challenging to assess in the post-mortem setting. Around 50% of patients will aspirate at the time of death (figure 8a), due to loss of muscle tone within the lower oesophageal sphincter, and thus fluid and debris is commonly encountered in both major and minor airways. At times, the absence of aspiration can support a cause of death, for example in cases of choking (figure 8b). The lungs are poorly distended and coupled with aspiration and lividity changes may obscure nodules, consolidation and lymph nodes. When the pulmonary density changes are asymmetric, central or in an anti-dependent position, it increases the likelihood of them being pathologic, however it is vital to determine how the body was positioned when it was found, as this may be significantly different to how it is positioned on the CT scanner.

Several groups describe post-mortem ventilation lung CT using continuous positive pressures delivered via multiple means including endotracheal tubes, laryngeal masks, tracheostomies and continuous positive airway pressure masks (10, 11). These studies have demonstrated reduced conspicuity of lividity and improved detection of nodules and pneumonia. The ability to use supraglottic masks increases the practicality given the challenge of intubating bodies with rigor mortis.

Brain changes

Many of the post-mortem brain changes will be familiar to radiologists as the findings are similar to those encountered in brain death studies. Changes on PMCT after one to two days include a loss of grey white matter differentiation with diffuse reduction in parenchymal density. This can result in vessels appearing hyperdense (figure 9a) and the cortical venous involvement can result is a pseudosubarachnoid haemorrhage appearance (figure 9b). These signs are usually symmetric, often prominent over the cerebral convexities and sylvian fissures and less evident in the posterior fossa and around the craniocervical junction. It is important to ensure there is no intraaxial or intraventricular element of haemorrhage and that no skull fractures are present. As the brain softens it sags to the dependent portion of the skull with gas being located in the anti-dependent portion and vessels(8).

Conclusion

An appreciation of the artefacts associated with dying and the post-mortem state is important as these are limitations of post-mortem CT, and if not recognised can lead to misdiagnosis.

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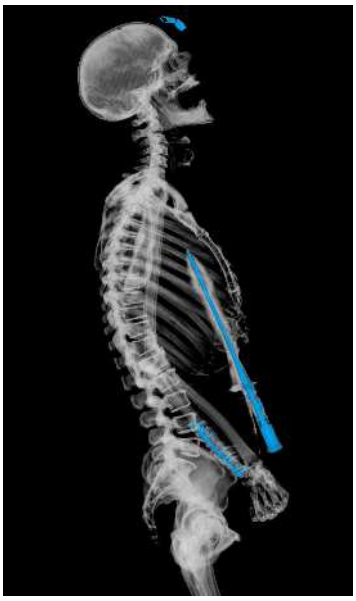

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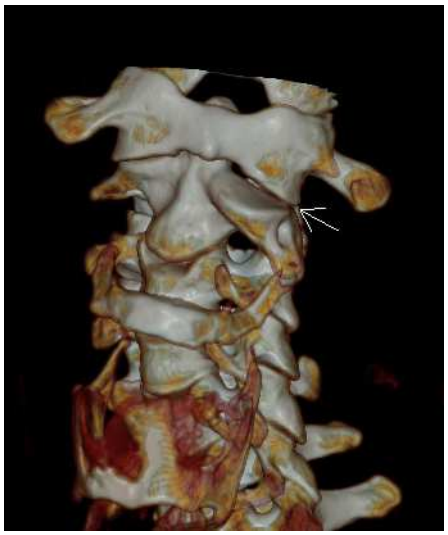

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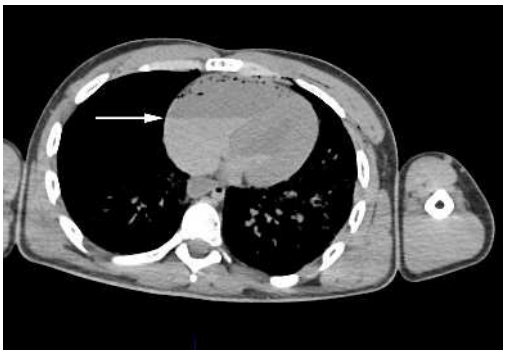

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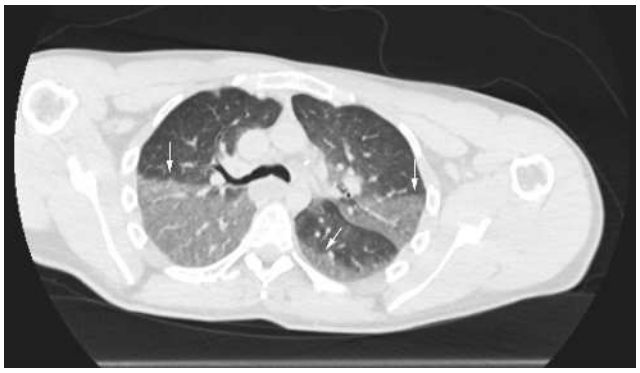

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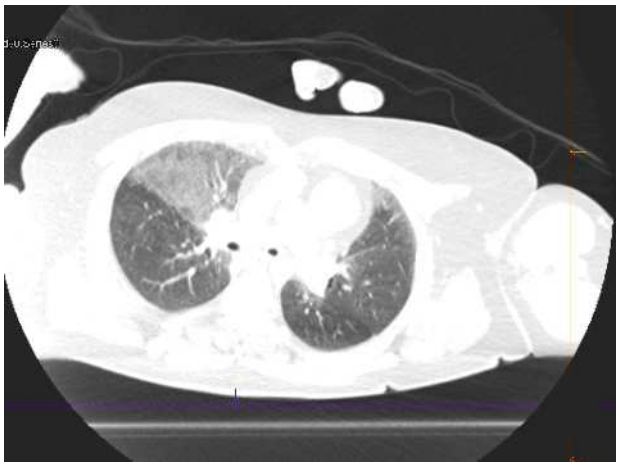

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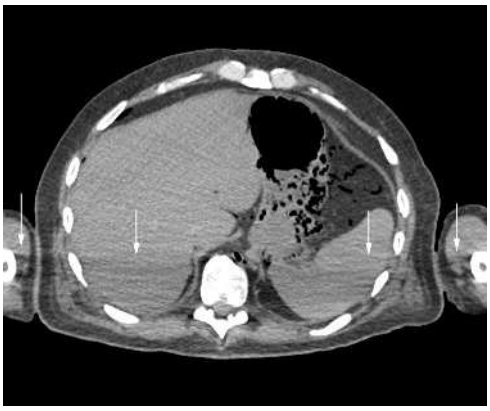

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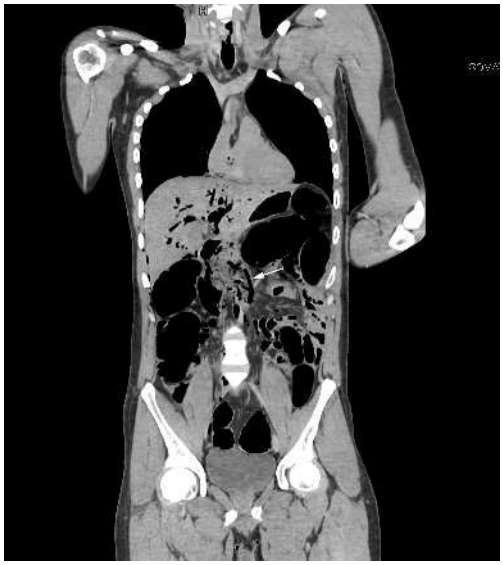

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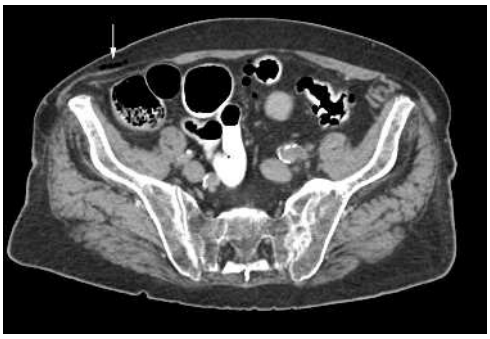

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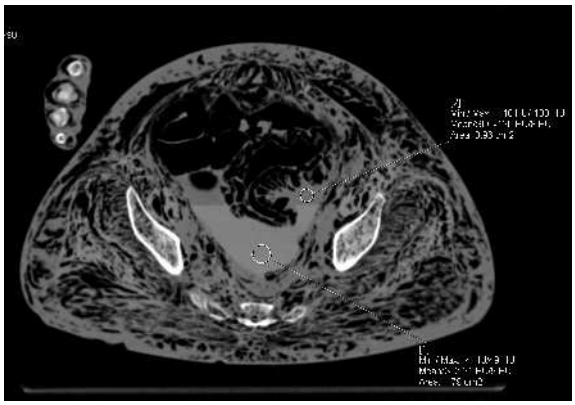

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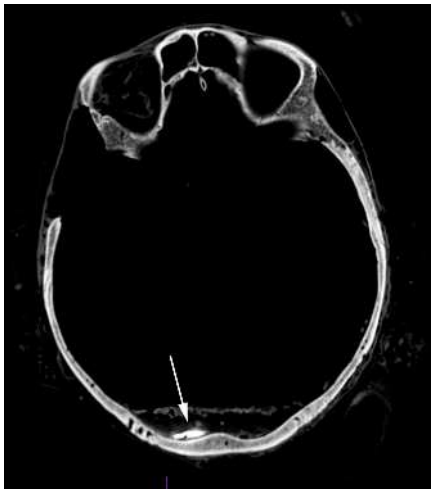

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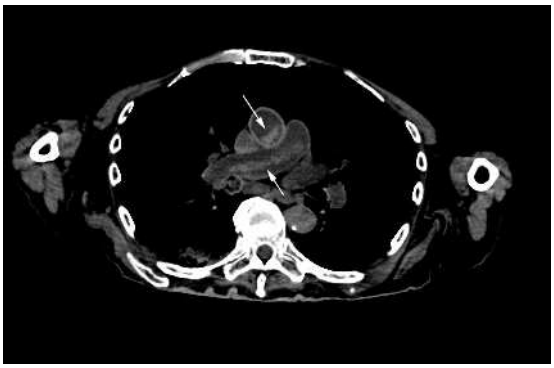

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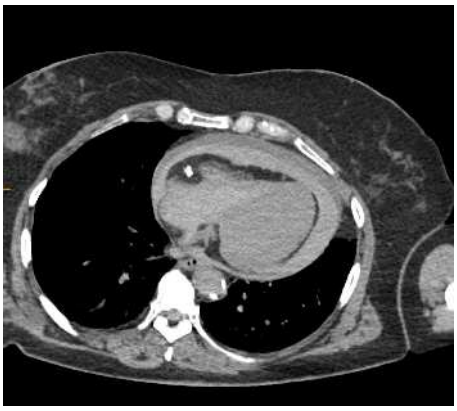

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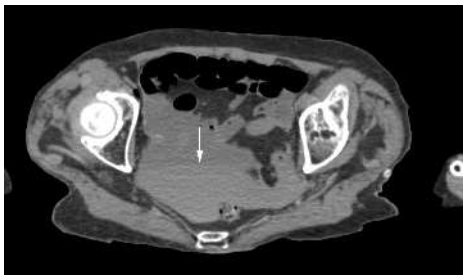

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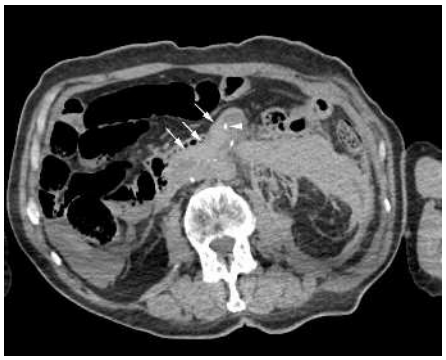

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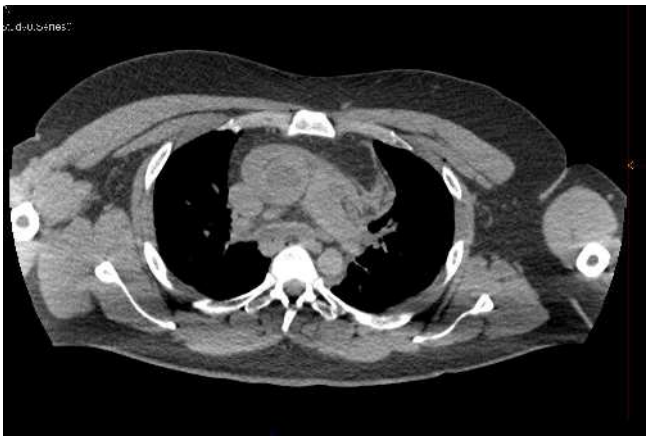

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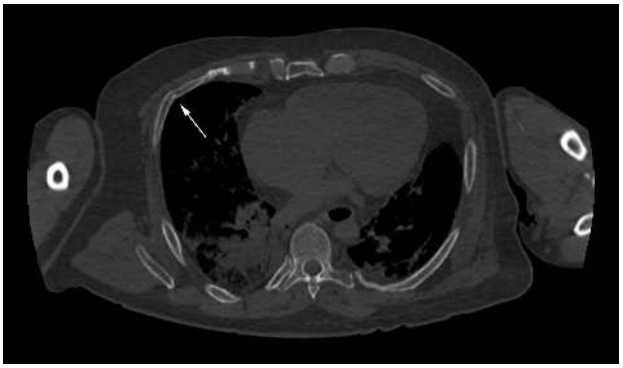

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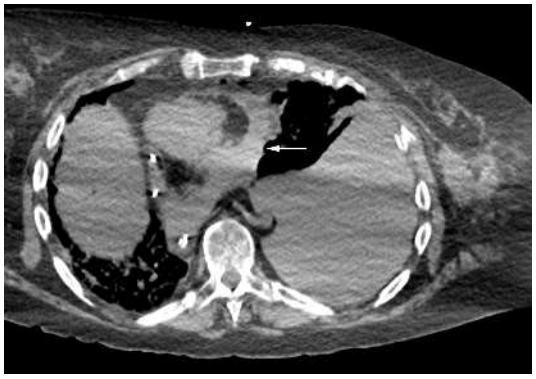

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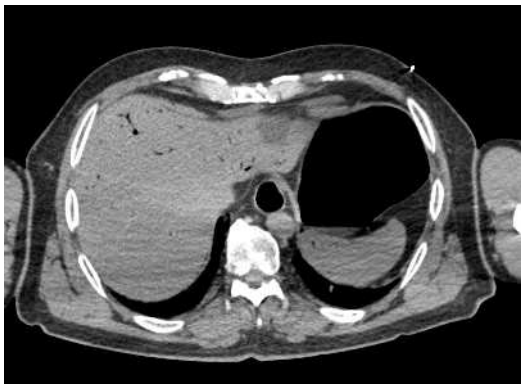

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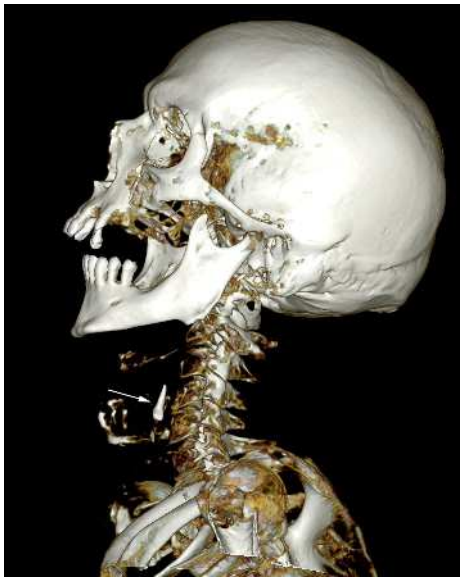

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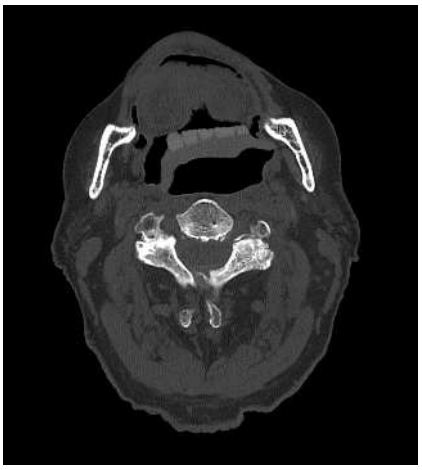

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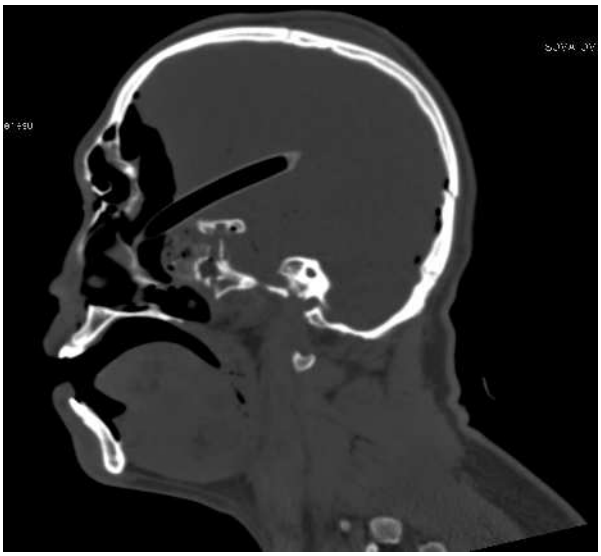

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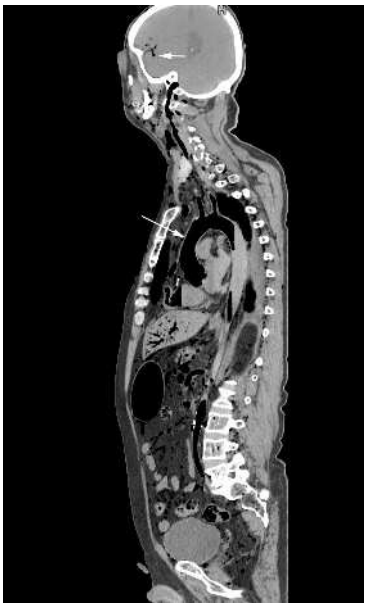

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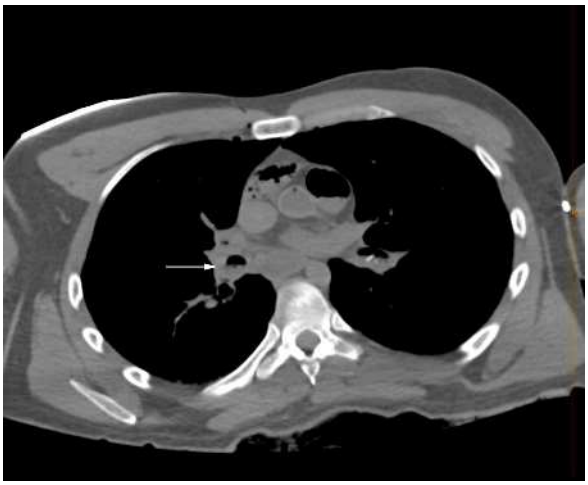

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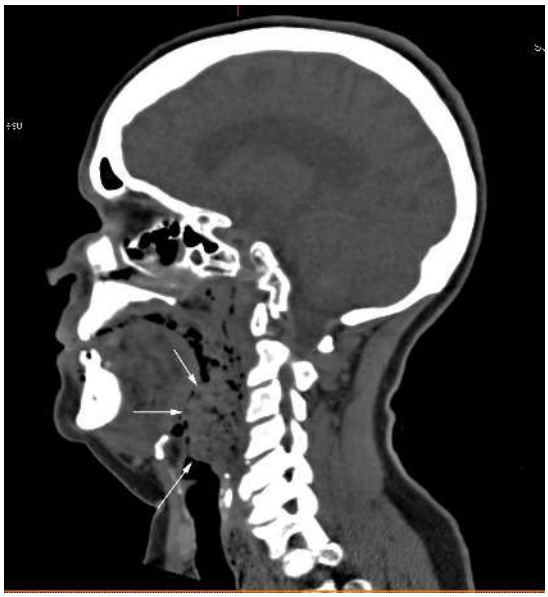

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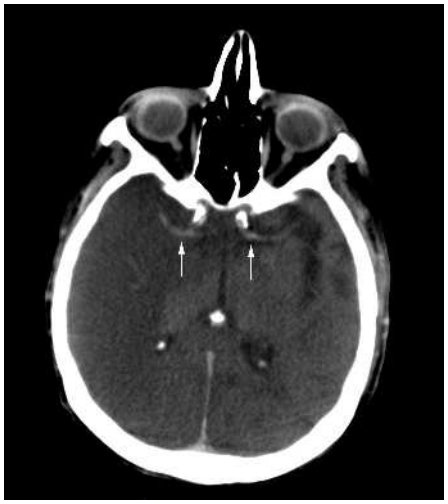

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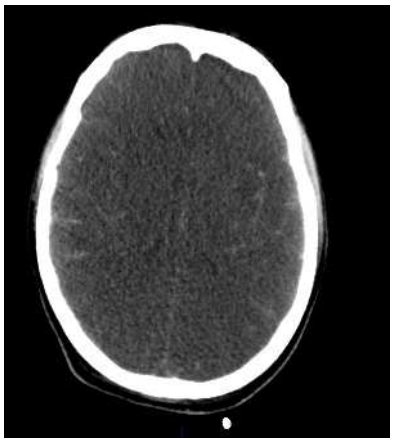

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