Advanced Imaging-Based Medicolegal Death Investigation: Postmortem MRI and CT

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Abstract

Postmortem computed tomography (PMCT) is well established for medicolegal death investigation. There are deficiencies in PMCT that can be overcome with postmortem magnetic resonance imaging (PMMRI) especially in the brain, spinal cord, and heart, and in young children. MRI sequences need to be adapted to the deceased, mostly to compensate for body cooling. In this case of hypoxic ischemic brain injury, both PMCT and PMMRI provided information about the deceased. This included iodine leak into the basal ganglia following high-dose antemortem CT scanning confirmed on dual energy PMCT, and swelling and T2 hyperintensity in gray matter of the basal ganglia and cortex on PMMRI. These findings were confirmed as hypoxic ischemic encephalopathy at autopsy examination.

Introduction

Postmortem computed tomography (PMCT) has become the standard of practice at many forensic institutions for investigating cause and manner of death. All admissions to our institution are scanned from head to toe. PMCT is used to triage the body to decide if there is a probable cause of death or to exclude traumatic injury, and to aid in determining if autopsy is necessary to advance the diagnosis and further characterize pathology. PMCT-guided procedures can also be undertaken and include angiography, specimen sampling such as bladder aspiration or orogastric tube insertion, and organ biopsy. Pathologists use PMCT to guide the autopsy dissection, concentrating on the areas of suspected pathology and avoiding the need to open all body cavities such as the skull if CT findings are negative. Retrospective review of PMCT can aid in interpreting findings at autopsy. Magnetic resonance imaging (MRI) is rarely used in postmortem practice due to difficulties in access. However, it can provide more information as long as sequences are adapted to the deceased to compensate for reduced body temperature, decomposition, and absence of blood flow. Image interpretation in both PMCT and postmortem MRI (PMMRI) differs from clinical imaging due to changes that occur in the body after death, and requires specialized postmortem imaging experience.

Although PMCT is useful for the analysis of bones, chest, abdomen, and hemorrhage, it has limited application as intravenous radiographic contrast agent is not administered after death. Major areas of deficiency include the brain, spinal cord, heart, and young children, where there is minimal abdominal and peripheral fat to separate organs and other structures. MRI has excellent contrast resolution in these areas. It is therefore complementary to CT and can provide a complete analysis of the body contents for forensic purposes.

Case report

A middle-aged adult was found unconscious on the floor. Prolonged resuscitation ensued and they were transferred to a hospital emergency department. Imaging in hospital included a CT brain scan with angiography and a CT pulmonary angiogram (CTPA) with abdomen and pelvis examination using a total of 150 cc radiographic contrast (Omnipaque 350 iohexol: 113.25 g/150 mL, GE Healthcare Australia Pty Ltd, Melbourne, Australia). The CT brain scan showed diffuse brain swelling and no hemorrhage. The CTPA showed no pulmonary embolism, and examination of the abdomen/pelvis was otherwise normal. Clinical testing subsequently confirmed brain death, and the patient was extubated. Given the sudden and unexpected nature of the death, it was referred to the coroner for medicolegal death investigation, and the decedent was transferred to the Victorian Institute of Forensic Medicine (VIFM). A whole-body CT was performed on admission, followed by dedicated dual energy CT of the brain based on initial findings. A postmortem brain MRI was performed after the body had been stored in refrigeration at ~ 4 °C. A full autopsy examination was undertaken after the MRI.

Technique

The institute's mortuary-based CT scanner is a SOMATOM Drive with software version VB20A. The PMCT technique included whole-body imaging from head to toe, and dedicated imaging with a smaller field of view through the head and neck (see Table 1).

Dual energy PMCT of the head was performed (see Table 2) and analyzed on *syngo*.via (version VB80D) using the CT Dual Energy workflow and the Virtual Unenhanced application profile.

The institute's mortuary-based MRI system is a 3T MAGNETOM Vida with software version XA60. Images were analyzed on *syngo*.via (version VB80D) using the MR Basic workflow. For imaging parameters, see Table 3.

After review of the whole-body CT scan to exclude internal metallic hardware, the body bag is opened, and an external examination is undertaken to detect and remove any metallic devices or other accoutrements on the skin. The body bag is then closed, with any metallic zips moved to the foot end. The body is carefully transferred with a ceiling hoist onto the eDrive dockable table. The head is positioned in the 64-channel head/neck coil, and the table is maneuvered to the scanner and docked.

Scan parameter	Routine head	Routine whole body
Scan range	Vertex to T1	Vertex to toes
Slice thickness	1 mm	1.5 mm
Slice increment	0.8 mm	1.0 mm
FOV	300 mm	500 mm
Soft Tissue		
Kernel	H31s	B30f
Window	Base orbita	Abdomen
Bone		
Kernel	H70h	Vertex to toes
Window	Bone	
Lung		
Kernel		B70f
Window		Lung
Eff mAs	420	280
kV	120	120
Pitch	0.55	0.6
Care Dose4D	off	off
Care kV	off	off
Rotation time	1 sec	0.5 sec
Detector config.	128 x 0.6	128 x 0.6

Table 1: PMCT whole-body imaging.

Parameter	Dual energy head
Care kV	Tube A 100 / Tube B Sn 140
Quality ref mAs	119
Pitch	0.6
FOV	300
Detector config.	32 x 0.6
Slice thickness	1.5 mm
Slice increment	1.0 mm
Reconstruction kernel (s)	Qr 40
Reconstruction window	Cerebrum

Table 2: Dual energy PMCT of the head

Sequence	MPRAGE	T2 TSE axial	T2 TSE coronal	T2 TSE dark-fluid axial_fs	PD TSE axial	T2 SWI	Resolve (segmented DWI) axial
Echo time (ms)	2.32	87	87	80	11	20	TE1: 75 TE2: 125
Repetition time (ms)	2300	3600	3600	9000	2290	30	5650
Slice thickness (mm)	0.9	3.0	3.5	3.0	3.0	2.0	3.0
Acquisition matrix	256 x 256	312 x 512	312 x 512	174 x 304	243 x 400	270 x 352	200 x 200
Imaged field of view (mm)	240 x 240	187 x 230	187 x 230	187 x 230	187 x 230	196 x 230	230 x 230
Receiver bandwidth (Hz/pixel)	200	222	222	289	223	100	781
Acquisition time (min:sec)	5.21	2.18	2.47	1.50	2.00	1.45	3.48
Acceleration	GRAPPA 2	GRAPPA 4	GRAPPA 4	GRAPPA 2	GRAPPA 2	Wave CAIPI: 6	SMS:2,: GRAPPA 2
AI (Deep Resolve Boost, Deep Resolve Sharp)	n/a	DRB:med; DRS	DRB:med;	50, 800	0.5 × 0.5 × 3.0	1.5 × 1.5 × 4.0	1.0 × 1.0 × 3.0
Inversion time (ms)*	900	n/a	n/a	Variable*	n/a	n/a	n/a
Flip angle	8	150	150	150	150	15	180
b values (s/mm²)	n/a	n/a	n/a	n/a	n/a	n/a	b0; b1500; Calc b2000

Table 3: Imaging parameters
* = depends on body temperature (see Fig. 4)
n/a = not applicable

Image review

The PMCT of the brain shows bilateral symmetrical hyperdensity in the basal ganglia, including caudate heads and lentiform nuclei. On dual energy CT (DECT), this hyperdensity is shown to represent iodine and disappears on the virtual non-contrast reconstruction (Fig. 1).

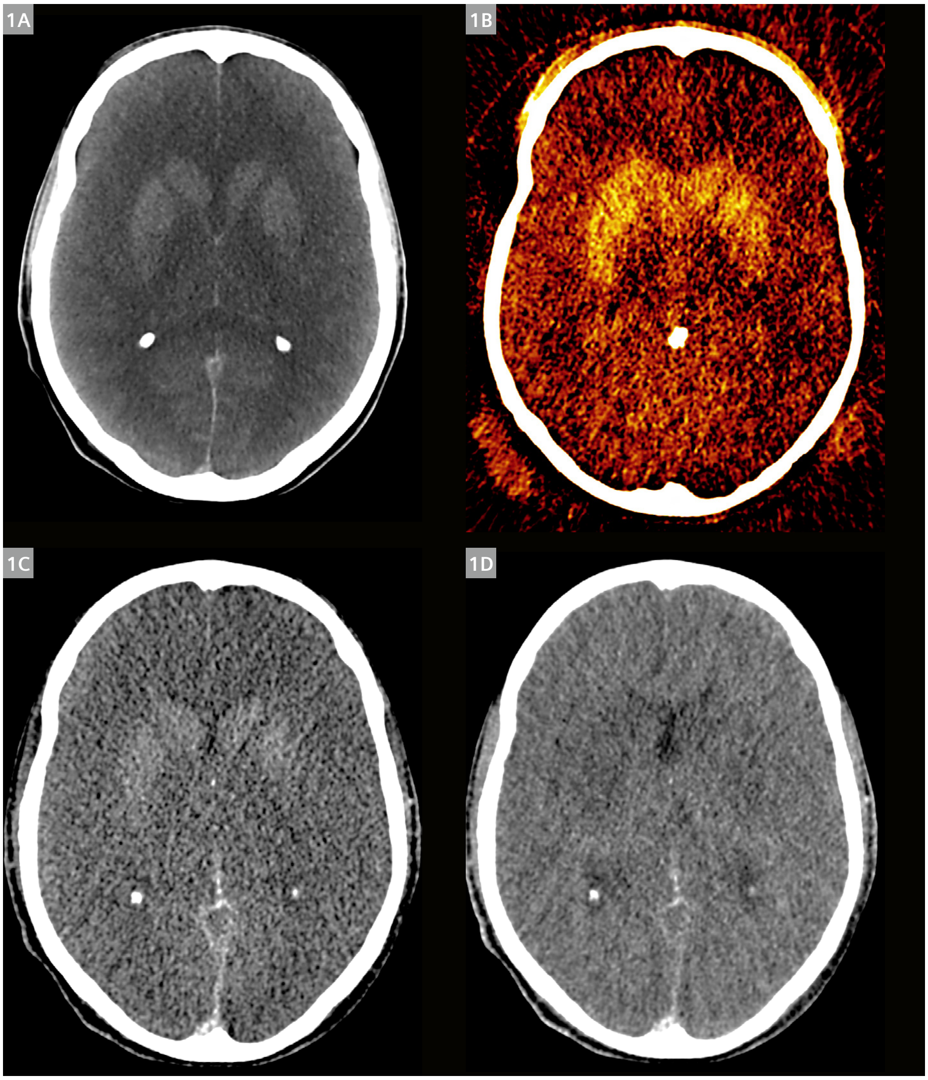
The PMMRI of the brain demonstrates swelling and increased T2 signal in the basal ganglia (Fig. 2). Further swelling and increased T2 signal indicative of edema is present within the cortical gray matter of both the frontal and parietal lobes, indicative of diffuse hypoxic ischemic brain injury. This was confirmed at autopsy.

Superimposition of colorized axial T2 TSE PMMRI and axial PMCT using *syngo*.via (Fig. 3).

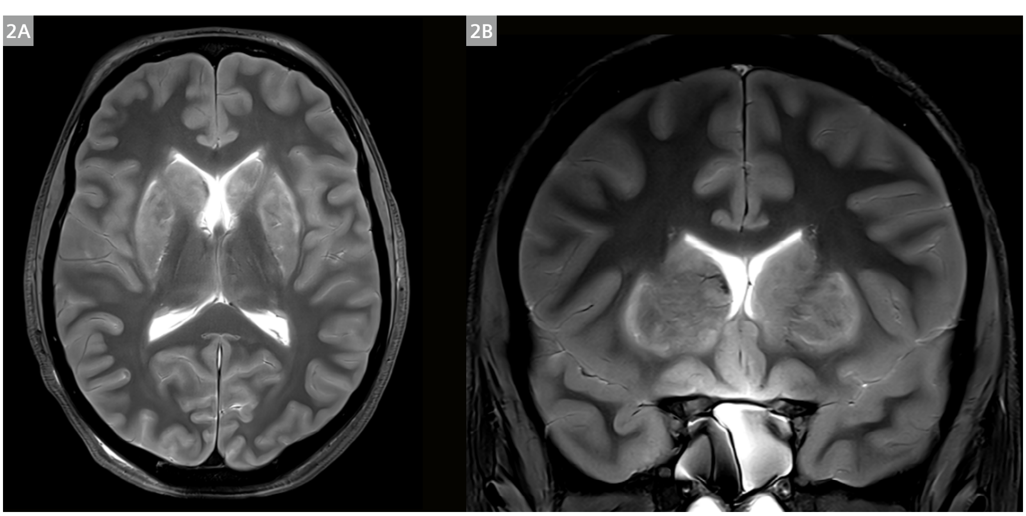
Series of FLAIR sequences with differing inversion times (constant TR and TE) showing varying effects on T2 contrast and signal within the cerebrospinal fluid (CSF) to find the null point, i.e., net zero transverse magnetization of water (Fig. 4).

Discussion

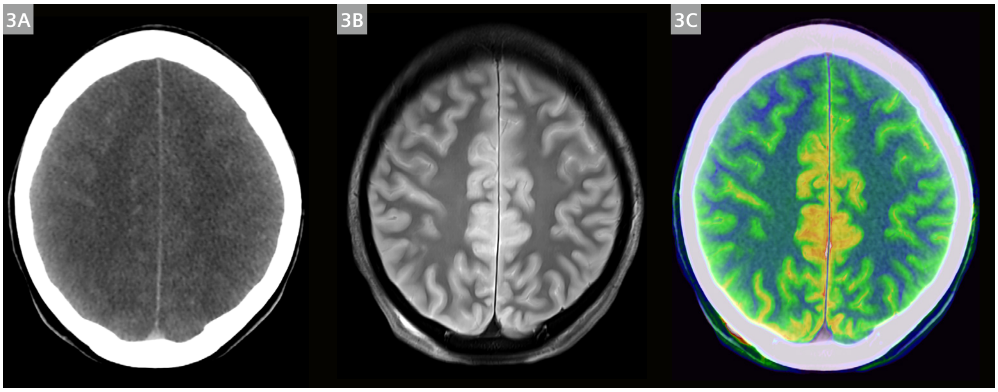
Hypoxic ischemic injury to the brain occurs when there is generalized inadequate oxygenation leading to cell swelling and death (so-called hypoxic ischemic encephalopathy or HIE). This is often associated with cardiac arrest. Gray matter is more metabolically active than white, making it sensitive to hypoxemia and more likely to be affected. This includes the basal ganglia and cortical gray matter. On CT imaging, hypoxemia is manifested as edema within gray matter and the resultant swelling. Normally, intravenously injected radiographic contrast does not enter the brain tissue due to an intact blood-brain barrier that restricts it to the meninges and blood vessels. If the barrier is disrupted, as it is in hypoxic injury, the radiographic contrast can leak into the brain, especially the basal ganglia, leading to a phenomenon known as hyperdense basal ganglia (HBG).



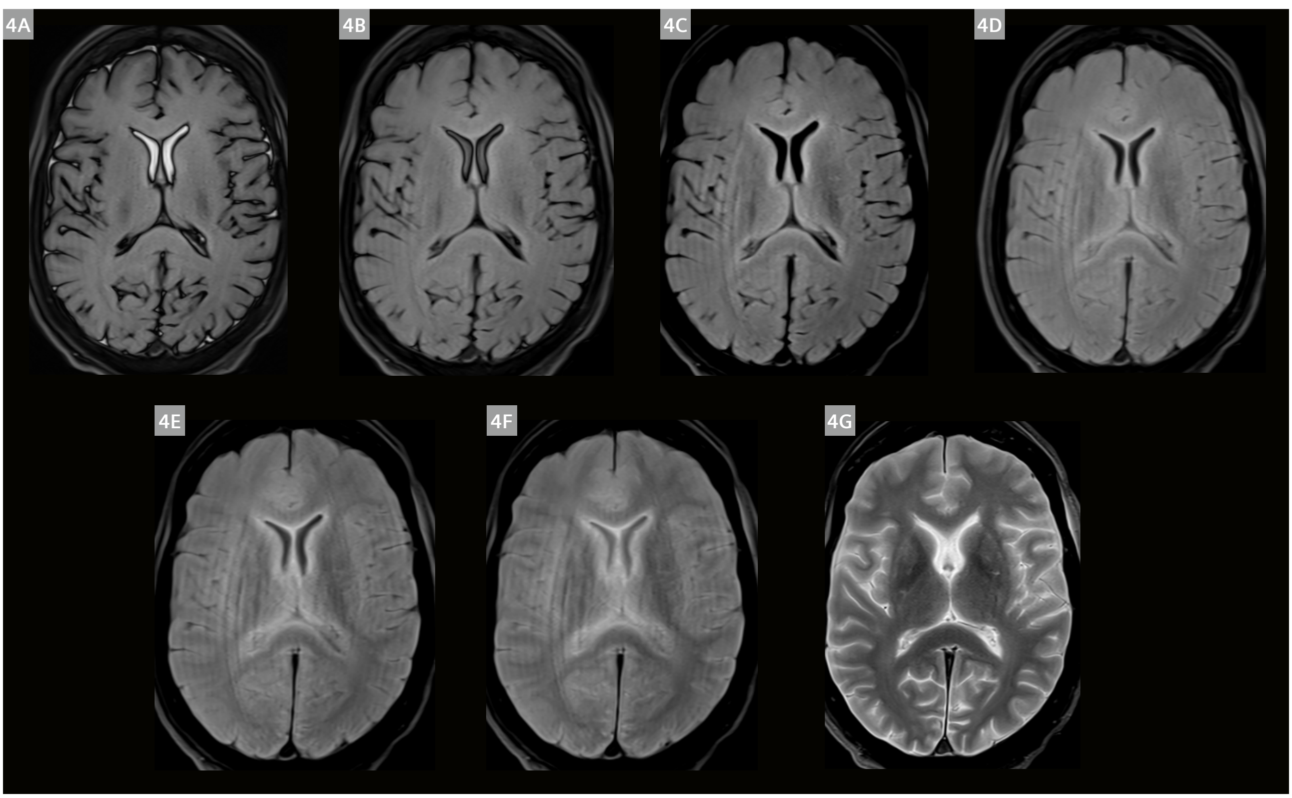
1 Axial PMCT brain **(1A)** showing bilateral symmetrical hyperdensity of basal ganglia. Axial PM DECT brain with iodine overlay **(1B)** shows that this hyperdensity is iodine deposition with a concentration of 1.5 mg/mL. Side-by-side comparison of DECT with iodine **(1C)** and virtual non-contrast **(1D)** showing loss of hyperdensity in the basal ganglia indicating the presence of iodine. Note how high-density blood in the superior sagittal sinus does not show loss of density on virtual non-contrast (VNC).



2 T2 TSE axial **(2A)** and coronal **(2B)** PMMRI showing bilateral symmetrical hyperintensity and swelling in basal ganglia indicative of hypoxic ischemic encephalopathy (HIE).



3 Axial PMCT **(3A)** shows minor swelling of the medial cortex. T2 TSE axial PMMRI **(3B)** shows significant swelling and abnormal T2 signal in the medial cortices. Fused colorized T2 TSE axial PMMRI and PMCT **(3C)**.



4 Axial T2 TSE dark-fluid FLAIR sequences with a TR of 9000, a TE of 80 ms, and a TI of **(4A)** 1000, **(4B)** 1200, **(4C)** 1400, **(4D)** 1600, **(4E)** 1800, **(4F)** 2000, and **(4G)** 2500 msec. The TI of 1400 ms produces the best signal suppression in CSF.

On PMCT, HBG is detected if an individual has an episode of severe hypoxic injury, is resuscitated to reestablish blood flow and oxygen to the brain, and then undergoes post collapse radiological imaging using large volumes of IV radiographic contrast (e.g., coronary angiography or a CT pan scan). Although not necessarily seen at the time of the antemortem CT, the contrast will progressively leak into the brain at sites where the blood-brain barrier is disrupted, such as the basal ganglia, and can be detected on PMCT as HBG.

Dual energy CT can detect iodine in tissues and can be used to confirm that this hyperdensity is iodine, distinguishing it from other causes such as nonketotic hyperglycemia, calcification, hemorrhage, and copper deposition (Wilson disease).

MR imaging is temperature sensitive, especially for T1-weighted sequences, leading to low signal intensity and tissue contrast [1]. T2 contrast and signal intensity are less affected. Deceased persons are routinely stored in fridges at ~ 4 °C. The temperature of the decedent at the time of scanning varies depending on body habitus and duration of refrigeration. If necessary, this can be quantified using rectal or forehead temperature measurements [2]. Limited T1-weighted sequences are performed in PMMRI and are not relied upon for diagnosis, apart from detection of methemoglobin and fat. T2-weighted sequences on PMMRI are most useful without flow artifacts. This means that time-of-flight MR angiography is not possible, but CSF is always universally white and brain edges are extremely sharp as there is no brain pulsation. FLAIR imaging is problematic due to temperature sensitivity, meaning inversion times differ from clinical practice [3]. We routinely perform a series of rapid preliminary FLAIR sequences with variable TI (1000–1600 ms) to determine the “sweet spot” or null point of CSF signal (Fig. 4). Once determined, this TI is then used for a full diagnostic sequence. A TI of 1400 ms is most commonly used on our magnet at 3T on bodies taken directly from refrigeration. This contrasts with the typical TI of 2500 ms used in clinical cases at body temperature.

Despite immediate changes to the body that occur following death, swelling and abnormal T2 signal in the brain on MRI are not a feature until decomposition progresses. It is somewhat counterintuitive that pathology such as HIE can be readily detected as swelling and increased T2 signal on PMMRI, although diffusion-weighted imaging is more problematic. This means that PMMRI of the brain is a particularly useful investigative tool for many fatal neurological conditions such as infarction, mass lesions, hemorrhage, and HIE.

Conclusion

Postmortem imaging is now highly sophisticated. It uses all the CT and MRI techniques available in clinical radiology, albeit with adjustments to allow for changes in the body after death and refrigeration. In medicolegal practice, interpretation must also be tempered by the requirements of investigators and the criminal justice system to answer questions, which may not always be obvious to clinically trained medical specialists, whose focus is diagnostic and therapeutic rather than forensic. Imaging specialists must therefore be experts in interpreting imaging of deceased persons, have an understanding of the law, and be able to engage with legal professionals and investigators, including providing expert evidence in court.

References

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