Neuroradiological Findings in Non-Accidental Trauma — Educational Pictorial Review

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Outline

1. Simulation-SIM as assessment tool
2. Clinical and Neuroradiological findings of Non-Accidental Trauma (NAT)
3. Pitfalls
   - Congenital infections
   - Birth related injuries e.g. subdural tentorial hematomas
4. Search pattern to prevent observational errors
5. Assertive guidelines to call NAT to avoid cognitive errors
6. Recommended imaging plan for a follow up
Background

➢ Computer aided simulation (SIM) was developed and designed to test residents for readiness for call

➢ 8 hour simulation of 65 emergent & critical care cases of varying degrees of difficulty, including normal studies using full DICOM image sets

➢ SIM was taken by 127 first (R1) & second (R2) year residents from 16 USA radiology training programs
Results

➢ 75% of the residents either called the study normal (observational error) or gave an incorrect diagnosis (cognitive error)

➢ No significant difference between R1 and R2 residents with an average scores of 17 versus 25% respectively

**Conclusion**: Significant observational and cognitive gap exists in detecting and differentiating NAT from other disease entities
Neuro NAT Manifestations

Brain:
- Axial loading injury - Skull fracture
- High impact trauma - Skull base fracture & brain injury
- Penetrating trauma
- Shaking Injury - Diffuse axonal injury & SDH
- Shearing injury
- Venous tethering
- Asphyxiation/Hypoxic brain injury
- Long term sequelae - Global atrophy

Spine
- Axial loading injury - Vertebral compression fracture
- Spinal cord injury
Fig 1 - Extensive scalp hematoma (white arrows) with associated skull fracture (red arrows)
Fig 2 - Right sided posterior rib fractures (yellow arrows), better identified on zooming (Fig 3) and changing windows (Fig 4)

75% of the residents failed to make the diagnosis indicating the need to revisit the radiology of NAT
Prominent extra-axial spaces (between purple arrows) predispose for extra-axial bleeding after getting shaken. Repetitive shaking leads to mixed age of extra-axial blood products (white arrows).

Larger subdural hemorrhage (SDH) (white arrows) is an uncommon spontaneous event in infants. Other types of blood collections include: subpial (yellow arrow) & intradural (pink arrow) hemorrhage and subdural hygromas (purple arrows).
Traditional Radiology Teaching of Neuro NAT

Fig 1 & 2 - Axial loaded skull fracture (red arrows)

Typical transverse fracture of the temporal bone (red arrows)

Axial CT (Fig 3) shows transverse oriented fracture (pink arrow) with its complete extent better demonstrated with on the 3D reformation (Fig 4)
Cervical Spine/Cord Injury

- Most commonly from a shaking/”whiplash” type mechanism
- May damage the lower brainstem and upper cervical cord
  - Could present as apnea and hypoxia.
- Subdural and epidural cord hematomas, cord contusion and ligamentous rupture
- Cervical spine MRI must be performed if there is any clinical suspicion of shaking-type injury
Long segment spinal cord edema

Sagittal (Fig 1) & axial (Fig 2) T2 images reveal spinal cord edema extending from the foramen magnum to C7 level (red arrows)

Axial loaded associated spinal column injury

Lateral lumbar spine X-ray shows typical compression deformity of a vertebral body (white arrow)

**Teaching point:** Cervical spine CT without bony injury is clearly underestimates the extent of cord injury
Shaking Injury

- **Diffuse Axonal Injury** preferentially affects
  - Gray-white matter junctions
  - Corpus callosum & basal ganglia
  - Dorsolateral aspect of the pons & upper brainstem

- **Shearing Injury** causes
  - Disruption of delicate cortical bridging veins as they leave the cortex to enter the dural venous sinuses, most commonly the superior sagittal sinus
  - Subdural hematomas & hygromas
**Teaching Point:** Diffuse axonal injury from shaking is usually multifocal and most often affects the gray-white matter junction, the corpus callosum, basal ganglia and the dorsolateral pons & upper brainstem.

Multiple areas of restricted diffusion along the right tentorial incisura, in the mesial right temporal lobe, right temporal pole, anterior frontal lobes bilaterally and adjacent to the cribriform plate (red arrows)
P1 Perforator (Shear) Injury

DAI in a 2-Month-Old with History of SDH

**Teaching Point:** Diffuse axonal injury is often subtle requiring special attention to SWI and DWI sequences

P1 perforator distribution edema: a shear effect not a post herniation effect (yellow arrow)

Figs 1 & 2 show a focus of microhemorrhage (white arrow) in the right frontal convexity and a left frontal acute SDH (red arrows) with foci of acute restriction in the left frontal lobe (yellow arrows) in Fig 3 & 4.
3-Month-Old with Seizures and Periorbital Bruising

**Teaching Point:** Patients with diffuse atrophy from repetitive trauma may require contrast to differential acute from chronic injury.

Figs 1 & 2 show dilated subarachnoid spaces (red arrows) with bridging veins (orange arrows) separating the subdural hygroma (pink arrow) from diffuse brain atrophy. Post-contrasted T1 images (Figs 3 & 4) reveal extensive subdural enhancement with pial irritation suggestive of mixed age subdural & subarachnoid hemorrhage (white arrows).
Hypoxic Ischemic Injury & Strangulation

- Asphyxiation/Hypoxic brain injury
  - Strangulation
  - Smothering
  - Apnea associated with brain stem stretch injuries in infants

- Affects…
  - Whole brain if severe
  - High ATP utilization zones if less severe
Hypoxic Ischemic Injury & Strangulation

➢ Hypoxic ischemic injury (HIE)
  • Spares the basal ganglia, thalami brainstem and cerebellum

➢ Strangulation
  • May affect unilateral watershed territory due to extrinsic compression of carotid artery
  • Hemorrhagic laminar necrosis may occur 7-10 days after initial insult
  • Must perform MRA to assess for vascular injury
10 Month Old with Seizures

Teaching Point: Strangulation often presents unilaterally and should trigger MRA of the neck to assess for vascular injury.

Fig 1 shows bilateral, frontal SDH (red arrows) with parafalcine extension (white arrow) and focal areas of diffusion restriction in the subcortical white matter in the right MCA/ACA watershed zone (yellow arrows in Figs 2 & 3).
Initial CT scan (Fig 1) read as Normal. Follow up CT 24 hours later (Fig 2) reveals loss of grey white matter differentiation, diffuse hypodensity of the white matter and complete effacement of the sulci consistent with diffuse cerebral edema.

**Teaching Point:** Causes of diffuse cerebral swelling include Diffuse primary injury (e.g. DAI) and metabolic derangement.
Teaching Point: Serial imaging & comparisons to priors, if available, are critical for identification of early signs of diffuse edema / brain injury.

Figs 1&2 show a large subgaleal hematoma (orange arrow) & upward transtentorial herniation (red arrow) due to diffuse edema causing effacement of the prepontine cisterns (yellow arrow) and diffuse sulcal effacement (pink arrows).
Direct Impact Trauma

- High likelihood of skull fracture, best seen on 3D reconstruction
- Focal parenchymal contusions & lacerations near scalp hematoma or fracture
- Traumatic SDH & subarachnoid hemorrhage tend to stem from direct disruption of vessels at fracture site

Cortical contusion
Unresponsive & Apneic Child

**Initial CT**
Axial, non-contrast CT images reveal diffuse interhemispheric and tentorial SDH (red arrows) and developing subdural hygromas (yellow arrows) with no overt skull fractures or soft tissue hematoma.
Follow up MRI
T1 (Fig 1 & 3) and FLAIR (Fig 2) show acute SDH (red arrows) superior to the transverse sinuses with significant enlargement of the panhemispheric subdural fluid collections (yellow arrows), as compared to the initial CT with minimal blood foci on T2* gradient imaging (white arrows in Fig 4)
Stabbing/Penetrating Trauma

- Similar potential findings as direct impact trauma

- Other complications include
  - Intracerebral hematoma
  - Posttraumatic aneurysm
  - Carotid-cavernous fistula
  - Arterial occlusion or venous thrombosis
  - CSF leakage

- CT is recommended due to possible retained metallic foreign bodies
30-Day-Old with Trauma

Coronal (Fig 1) and axial (Figs 2 & 3) CT images show hyperdensities in the subarachnoid spaces (red arrows) likely related to subcortical venous stasis and acute SDH & subarachnoid hemorrhage along the vertex. This is associated with diffuse loss of gray-white differentiation (yellow arrows) & effacement of the ventricles and sulci.

**Teaching Point:** Even when imaging clearly shows diffuse cerebral edema & HIE with its poor prognosis complete documentation of findings (e.g. fractures) is still needed for medical legal purposes.
Initial CT
Fig 1 shows extensive right facial & scalp hematoma (red arrows) with comminuted and displaced right parietal bone fracture (yellow arrows in Fig 2) and a small subjacent hemorrhagic contusion and edema (white arrow in Fig 3). This wedge-shaped defect in the parietal lobe in association with high energy impact is concerning for entrapped brain tissue in the adjacent fracture fragments.
MRI follow up 3 days later
T1 and DWI images confirm the posttraumatic wedge-shaped defect extending from the right parietal cortex to the right ventricular trigone (red arrows) with a focus of entrapped brain tissue at the fracture site (yellow arrow) and small volume extra axial blood (white arrow) on the T2* gradient sequence.

Teaching Point: High velocity direct impact trauma tends to have a more focal distribution.
Linear blood in subcortical location on CT is consistent with brain laceration (**red arrow**)

Widespread cortical & subcortical brain lacerations (**yellow arrows**) on DWI

**Vertex tethering / laceration**
Initial CT
Fig 1 & 3 reveal a slightly depressed occipital bone fracture extending into the right temporal bone (red arrows). Fig 2 shows a small right cerebellar hemorrhage (yellow arrows) and a possible infarct (pink arrow). Fig 4 demonstrates right parietal occipital brain laceration (white arrow) subjacent to the skull fracture.
31 Month-Old Boy after “Fall”

Follow up MRI
Fig 1 & 2 illustrate increased conspicuity of the brain laceration (red arrows) with a new focus of diffusion restriction in the anterior temporal pole (yellow arrow). Fig 3 reveals more extensive right cerebellar hemorrhage (pink arrow) than indicated on the initial CT.

Teaching Point: CT may underestimate the extent of hemorrhage, as well as more subtle injuries away from the impact site.
Long-Term Sequelae

- Metabolic insults lead to global atrophy, most commonly following diffuse cerebral edema (if the patient survives)

Unexplained macrocephaly related to chronic subdural effusions (red arrows) producing an increased cranial volume

Unexplained atrophy (pink arrows) & acute parafalcine SDH (yellow arrows)
Axial CT post NAT(Fig 1 & 2) shows decreased basal ganglia density (red arrows) & sulcal effacement (yellow arrows) related to hypoxic effects (confirmed at autopsy). In addition, there is a focal acute cortical hemorrhage (orange arrow).

Initial DWI sequence shows few scattered foci of restricted diffusion (red arrows) with subtle involvement of the pons (yellow arrows) that becomes markedly more apparent on the follow up DWI 10 days later.
**Patient 1 - HSV Infection Simulating NAT**

- Multifocal areas of diffusion restriction (**red arrows**) consistent with DAI in this patient with suspicion for NAT that were proven to be caused HSV infection.

**Patient 2 - Birth Related SDH Simulating NAT**

- Tentorial SDH (**yellow arrows**) was seen on a MRI done for postnatal HIE in this premature infant which should not be interpreted as NAT on follow up MRI.

**Pitfalls in the diagnosis of NAT:**

**Patient 1** - Multifocal areas of diffusion restriction (**red arrows**) consistent with DAI in this patient with suspicion for NAT that were proven to be caused HSV infection.

**Patient 2** – Tentorial SDH (**yellow arrows**) was seen on a MRI done for postnatal HIE in this premature infant which should not be interpreted as NAT on follow up MRI.
Importance of Follow up: Fatal sequelae of NAT

Initial non contrast CT (Fig 1) shows a subdural catheter in the left parietal region in this unresponsive patient with known NAT. CT done 2 days later (Figs 2 - 4) reveal a large left MCA infarct (yellow arrows) with uncal herniation (orange arrow)

Non contrast CT performed 9 days after the initial CT (Fig 5 & 6) shows hemorrhagic transformation of the left MCA infarct (white arrows).

Teaching Point: Routine follow up CTs are required in unresponsive patients in whom the clinical signs and symptoms are unreliable to detect the fatal complications.
Proposed Imaging Protocol

by Jaspan et al.

Suspected NAT

Day 0

CT

Days 1-2

Skull radiographs and Cranial US

If neurologic symptoms & normal/equivocal CT

Day 3-4

MRI: T1, T2, FLAIR and DWI
T2*GRE or SWI

Continued follow-up, as indicated

CT

At 10 days
If intracranial abnormalities

Suspected Vascular Injury

MRA
Conclusions

Traditional outlook for NAT in Neuroradiology

- Subdural collections in different stages
- Multiple fractures of skull; Multiple Wormian bones/Sutural diastases; Classic transverse fracture of the temporal bone

Additional findings which all the Neuroradiologists need to look for are - Axial loading injury and spinal cord injury in spine

In brain a range of injury pattern - DAI, Shearing injury, hypoxic injury, P1 perforator injury, delayed manifestations like large arterial territorial stroke, venous tethering and laceration