Clinical History:
Polytraumatized 17 year-old girl who came to the emergency room with low Glasgow Coma Scale score (7/15) and a previous blunt head trauma without apparent thoracoabdominal injuries.

Imaging Findings:

1. CT EMERGENCY ROOM (Fig. 1)
- Multiple tiny hyperdense foci (microhaemorrhage) in the gray–white matter junction of the right frontal lobe and left parietal lobe, body and splenium of corpus callosum and right thalamus.
- Subtle intraventricular haemorrhage in the right occipital horn.

2. CT AFTER 2 DAYS (Fig. 2)
- More conspicuous intraventricular haemorrhage (Fig. 2a; yellow arrow).
- Persistent tiny hyperdense foci (microhaemorrhage) in the gray–white matter junction of the right frontal lobe and left parietal lobe, body and splenium of corpus callosum and new hyperdense foci in the right medial temporal lobe (Fig. 2a; red arrows).
- Increasing hypodensity and volume of the body/splenium of corpus callosum (Fig. 2b; red arrows).

3. MRI FINDINGS (Fig. 3; Fig. 4)
- Haemorrhagic lesions: Multiple supra and infratentorial susceptibility artefacts on SWI images (Fig. 3a; Fig. 4).
- Non-haemorrhagic lesions: Multiple hyperintense areas on FLAIR sequences (Fig. 3c)
- Prognosis: The region demonstrating restricted diffusion is assumed to have suffered irreversible injury. Note the
Discussion:

1. BACKGROUND [1, 2]

Primary traumatic brain injury describes the lesion sustained to the brain parenchyma at the moment of trauma. Contusions are the most common intra-axial injuries. Other primary lesions are: focal brain injuries (lacerations), haemorrhage, diffuse axonal injury (DAI), or penetrating injuries/blast injuries. Secondary TBI results from processes initiated by the trauma, as for example brain swelling, cerebral hypoxia, raised intracranial pressure, or hypothalamic–pituitary dysfunction among others.

We will focus on DAI, one of the most disabling conditions of primary traumatic brain. DAI occurs as a result of acceleration–deceleration/rotational force, and is typically located in the long white matter fibre tracts. Cortex and white-matter have different densities and therefore rotate at different speeds during closed head injury, leading to misaligned axons or stretched axons (rarely sheared). The stretching of axons causes depolarization, metabolic alterations, cellular swelling, cytotoxic oedema, and apoptosis.

2. CLINICAL PERSPECTIVE [1]

We should consider DAI when traumatic patients present with a loss of consciousness without or with minimal lesions on CT.

3. IMAGING PERSPECTIVE [3, 4, 5]

DAI typically presents with haemorrhagic and non-haemorrhagic lesions.

CT findings

- Often without relevant findings
- Tiny hyperdense foci (microhaemorrhage) in:
  1. Gray matter–white matter, especially frontotemporal lobes.
  2. Corpus callosum, especially splenium.
  3. Brainstem, especially dorsolateral midbrain and upper pons.
  4. Less common: Deep gray matter, basal ganglia and internal/external capsule, tegmentum, fornix, corona radiata and cerebellar peduncles
- Intraventricular haemorrhage correlates with DAI
- MRI is superior to CT in detecting haemorrhagic and non-haemorrhagic DAI lesions.

MRI findings

- Haemorrhagic lesions: SWI is the best tool to detect haemorrhagic DAI.
- Non-haemorrhagic lesion: FLAIR is the best current tool to detect small and non-haemorrhagic parenchymal lesions, displayed as hyperintense lesions.
- Prognosis: The region demonstrating restricted diffusion (DWI) is assumed to have suffered irreversible injury. Schaefer et al. demonstrated that the volume of altered DWI shows a stronger correlation with clinical outcome and Glasgow coma scale than FLAIR. DTI (Diffusion tensor imaging) analyses water motion in order to evaluate the integrity of white matter tracts. Neuronal disruption can be implied by reductions in fractional anisotropy (FA).

Nuclear medicine findings

- PET may show hypometabolism in cingulate gyrus, lingual gyrus, and cuneus.

4. OUTCOME [4, 6]

Different degrees of DAI have been shown to be present in 80–90% of patients with traumatic brain injury. Prognosis, however, is highly dependent on the extent and location of the injury.
greatly depends on its extension (Grade I to III).

Grading: Adams and Gennarelli staging
Stage 1 (mild): Frontal and temporal lobe grey-white matter interface lesions
Stage 2: Lesions in lobar white-matter and corpus callosum
Stage 3 (severe): Lesions of dorsolateral midbrain and upper pons

5. TAKE HOME MESSAGE

Most of the lesions are microscopic and non-haemorrhagic.

**Differential Diagnosis List:** Diffuse axonal injury grade III, Demyelinating disease (non-hemorrhagic lesions), Chronic hypertension, Cerebral amyloid angiopathy

**Final Diagnosis:** Diffuse axonal injury grade III

References:

Lee S, Baek HJ, Jung HK et al. (2017) Interpretations of diffusion-weighted MR imaging by radiology residents in the emergency department: is diagnostic performance influenced by the level of residency training?. Radiol Med Jan;122(1):35-42 (PMID: 27670660)
Figure 1

Description: Multiple tiny hyperdense foci (microhaemorrhage, red arrows). From the top to the bottom: Junction between gray–white matter (frontal lobes), corpus callosum (splenium) and right thalamus. Note the intraventricular haemorrhage within the right occipital horn. Origin: J.A Prat-Matifoll; Radiology department; Vall Hebron Hospital
Description: More conspicuous intraventricular haemorrhage (yellow arrow) and microhaemorrhages in the splenium and right temporal lobe (red arrows). Origin: J.A Prat-Matifoll; Radiology department; Vall Hebron Hospital
Description: Intraventricular haemorrhage (yellow arrow) and increasing hypodensity of posterior corpus callosum, caused by oedematous changes (red arrows). Origin: J.A Prat-Matifoll; Radiology department; Vall Hebron Hospital
**Description:** Multiple magnetic susceptibility artefacts (haemorrhages). Mainly located in the gray matter–white matter junction of frontotemporal lobes, body/splenium of corpus callosum and right dorsolateral midbrain.

Note the affection of the right thalamus (white arrow). **Origin:** J.A Prat-Matifoll; Radiology department; Vall Hebron Hospital
Description: Tiny hyperintense dots on T1WI in the subarachnoidal space, compatible with subarachnoidal haemorrhage as well as right laminar subdural haemorrhage (yellow arrows). Origin: J.A Prat-Matifoll; Radiology department; Vall Hebron Hospital
Description: Conspicuous hyperintense areas on T2-FLAIR sequences demonstrating altered frontotemporal cortico-subcortical junctions, body/splenium of corpus callosum and right thalamus (orange arrow). Origin: J.A Prat-Matifoll; Radiology department; Vall Hebron Hospital
Description: Restricted diffusion within the body and splenium of corpus callosum (red arrows), probably secondary to an underlying cytotoxic oedema and irreversible injury in this location.

Origin: J.A Prat-Matifoll; Radiology department; Vall Hebron Hospital
Description: Note the presence of tiny magnetic susceptibility artefacts on SWI (susceptibility weighted imaging) in the right midbrain and right cerebellar peduncle/dentate nucleus (red arrows). These lesions correspond to a stage 3 (severe) diffuse axonal injury. Origin: J.A Prat-Matifoll; Radiology department; Vall Hebron Hospital