The comparison of the value of CT imaging and selected MRI sequences (including DWI) in the evaluation of axonal injuries

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Summary

Background: Diffuse axonal injuries of the brain consist in the damage (overstretching or torsion) of white matter axons, as a result of the forces of energy waves, evoked in the moment of injury, together with its accelerating-retarding inertia effect. Patients with DAI are most frequently the casualties of high speed car accidents. Diffuse axonal injuries of the brain are one of the most common acute brain injuries, with lesions typically occurring in the periventricular white matter, corpus callosum, and on the borderline of the white and grey matter, subventricularly. The diagnosis of axonal injuries is difficult, as the majority of lesions found in DAI are of microscopic nature.

Material/Methods: The material included the evaluation of 8 patients with craniocerebral injuries, normal results of brain CT (or showing slight posttraumatic lesions), and in severe neurological clinical state (continuing coma), which was all suggestive of a diffuse axonal injury. The patients were subjected to brain MRI studies within an MRI trauma protocol including FLAIR and DWI sequences, as well as sagittal T2-weighed images, which shortened the diagnostic examination time and was sufficient for the visualisation of DAI-specific lesions.

Results: On MRI examination, seven patients were diagnosed with diffuse foci of high signal intensity, located in corpus callosum, basal ganglia, thalamus and brain stem, although the CT examination results were normal or revealing minor changes. The foci were most prominent in DWI images. DWI sequence showed a diffuse cytotoxic oedema of white matter in one case, in which the CT results were normal.

Conclusions: The MRI examination with DWI should become a basic diagnostic tool in DAI. Due to patients' severe condition, the diagnostic process should be shortened. This could be done with the use of some selected sequences and projections of brain MRI, including transverse DWI and FLAIR, as well as T2-weighed images in sagittal plane, which reduces the time of the examination by approx. 12–15 minutes. Correct and quick diagnosis of a diffuse axonal injury is of major therapeutic and prognostic importance.

Key words: DAI – diffuse axonal injury • MR- DWI – diffusion weighted imaging

PDF file: http://www.polradiol.com/fulltxt.php?ICID=878428

Background

Diffuse axonal injuries are one of the most frequent brain injuries which lead to a prolonged coma, patient's vegetative state, and subsequent death. The majority of patients who underwent the diffuse axonal brain injury are socially excluded.

Treatment outcomes depend to a large extent on a quick diagnosis and evaluation of lesion extension.

A major role in the early diagnosis of diffuse axonal injuries is played by imaging studies. Until recently, the basic imaging method was computed tomography. However, due to the fact that some lesions occurring in DAI cannot be
visualised on CT examination, or it is impossible to evaluate their extension precisely, the value of CT in DAI diagnostics is limited.

The actual degree of brain damage in the course of diffuse axonal injuries is higher and more severe than what is shown by the image. At the microscopic level, although there is no complete interruption of axon continuity during the injury, the degenerative processes are activated and lead to a subsequent cytoskeletal disintegration, organelle accumulation, axon myelin sheath disorders and parenchymal atrophy, in the long run.

This nervous cell damage, delayed (in relation to the moment of injury), and further progressing, is a major mechanism of DAI lesions.

No significant predilections towards DAI were found with respect to sex or age – DAI may occur in every age and in both sexes. The only significant factor is the mechanism of injury, including a rapid rotation, resulting in shearing forces (‘shearing injury’).

Among recently introduced new imaging methods aiming at a faster and more precise diagnosis of diffuse axonal brain injuries, the MRI DWI imaging seems to be the method of choice.

Due to patients’ health state that excludes long-lasting, standard MRI examinations, we developed so called ‘posttraumatic MRI protocol’ which involves transverse DWI and FLAIR sequences, as well as sagittal T2-weighed images. Thus, the time of the examination does not exceed 15 minutes. Posttraumatic MRI has always been preceded by CT exam, in order to exclude intracranial bleeding and other extensive posttraumatic CNS damages.

Qualified for posttraumatic MRI examination were the patients with no or slight lesions on CT examination, which did not match patient’s severe neurological condition – a prolonged coma.

Material and Methods

The study group involved 8 patients – casualties of road traffic, with a predominant prolonged coma in the clinical picture of most of them – unconscious from the moment of the accident, with no other, or with minor injuries (4 patients without any other injuries, 3 fractures of the limbs, 1 haematoma of soft tissues of the neck). Only one patient was conscious but confused – he was answering the questions with random words. Most of the patients were under the influence of anaesthesia and sedation administered right after the injury, or they revealed symptoms of brain coma; the GCS score ranged from 3–7 points but it was unreliable in some of the cases, due to the administered sedatives. One patient, with retained limited consciousness, was breathing on his own. Other patients were intubated and mechanically ventilated. One patient had to be reanimated after a circulatory arrest at the scene of the accident (he was diagnosed with cytotoxic brain oedema due to ischaemia and hypoxaemia of the brain). During their hospitalisation at an Intensive Care Unit, the patients were mechanically ventilated with different systems of ventilation – the invasiveness of ventilation was gradually decreased.

The study group included 2 women and 6 men in the age of 8–50 years (mean age 22.4 years). Patients were subjected to the CT examination without contrast enhancement, in order to exclude intracranial bleeding and other injuries in the first 24 hours after the procedure, and the MRI examination according to so called ‘trauma protocol’, within 24–72 hours from the moment of injury.

The CT examination was performed with the apparatus by Siemens: Emotion 6; the technique used was sequential, with the slices being 4 mm thick, with bone and tissue ‘window’.

The MRI examination was carried out with Signa LX apparatus (GE Medical System), with magnetic field intensity of 1.5 T, and with the use of a basic coil for head examinations. Diffusion images were registered in echo-planar technique, with b value equal to 1000 s/mm², slice thickness =5 mm, no intervals between the slices, the number of impulses =1, TR =8000 ms, and TE =95 ms.

Results

CT examinations of 7 patients revealed single (2–5), small (approx. 5–10 mm) haemorrhagic foci. The majority of lesions were located in brain hemispheres, in the frontal and parietal lobes mainly, at the borderline of cortex and brain tissue. One of the patients had his haemorrhagic focus revealed in the brain stem, and the other one in thalamus. In one case there were no changes of the CT image.

In the MRI examination of 7 patients, the DWI images revealed multiple (>5) foci of higher signal intensity – the largest one of approx. 30 mm, located in four cases in corpus callosum. Two of these cases had some additional foci in the brain stem. Other foci could be found at the borderline of the white and grey matter of the brain hemispheres (diffused), in the frontal and parietal lobes, in the basal ganglia and thalamus (single foci). They all corresponded to the postraumatic brain damage of diffuse axonal type.

One patient, with a normal baseline CT, was subjected to the MRI examination on the third posttraumatic day, and diagnosed with an extensive, diffuse cytotoxic oedema of the white matter resulting from the ischaemic lesions – with additional typical foci of a recent ischaemia in basal ganglia (the patient was reanimated after circulatory arrest following the accident).

Lesions of different intensity were also visible in FLAIR sequence (proper visualisation of corpus callosum lesions) and in sagittal projection, in T2-weiged images.

For statistical purposes, the foci of brain damage present in the analysed cases were divided into four groups/regions and summed up. The first group consisted of lesions of the periventricular white matter (PWM), the second one: subcortical foci (SF), the third one: foci in corpus callosum (CC), and the fourth one: foci of the brain stem (BS).
The CT revealed 8 ‘locations’ with apparent pathologies in 5 patients, which makes together 16 lesions: 3 in periventricular white matter, 12 in subcortical structures, 0 in corpus callosum, 1 in brain stem.

The MRI, performed according to a shortened brain trauma protocol (sagittal T2-weighted projection, as well as transverse FLAIR and DWI images) showed 20 ‘locations’ with apparent pathologies in all 8 patients, which makes together 64 lesions: 18 in periventricular white matter, 31 in subcortical structures, 12 in corpus callosum, 3 in brain stem (Table 1).

The statistical comparison of the number of foci revealed on CT and MRI was significant (Wilcoxon test for matched pairs); P<0.001. Similar results were obtained in the comparison of the number of lesions within the following brain regions: periventricular white matter (PWM), subcortical foci (SF), corpus callosum (CC), and brain stem (BS), P<0.05 (Figure 1).

According to Adams’ classification (discussed below) and its localisation and prognostic value (of further survival), two patients were diagnosed with DAIIs of the first degree (mild). Four patients, with diffuse foci not only in brain hemispheres, thalamus, and basal ganglia, but also in corpus callosum, were diagnosed with II degree DAI (moderate); two patients with diffuse foci in the brain hemispheres and corpus callosum, but also in the brain stem, were diagnosed with DAI of the third degree (severe). In total, DAI was revealed in 7 patients.

One patient had a diffuse, cytotoxic posttraumatic oedema of the white matter which could not be classified with Adams’ scale.

Three patients died – 2 of DAI of the II degree and 1 of DAI of the III degree; the patient with cytotoxic oedema was in the vegetative state at the time of data processing.

Discussion

Pathophysiologists were the first ones who pointed to extensive, posttraumatic injuries of the white matter – such injuries were described as early as in 1943 by Holbourn [1], and in 1956 by Strich [2,3]. In 1982, Adams et al. [3] and Gennarelli et al. introduced the name ‘diffuse axonal injury’ [4]; nowadays, it is commonly accepted and used [5,6].

Diffuse axonal injuries are one of the most frequent primary neuronal damages in patients with acute brain injuries – second the most frequent, right after posttraumatic haematomas [7,8].

DAI consists in tearing of axons within the white matter of the brain, which occurs during an injury with concomitant rotary acceleration.

The cause of brain tissue damage in such traumas is the difference in the inertia of the grey and white matter, leading to the injury of axons at the borderline of the white and grey matter and tearing of small blood vessels.

Table 1. Listing of the number and locations of the foci revealed on CT and MRI examinations in particular patients with DAI.

| Patient's data | Periventricular white matter | Subcortical foci | Corpus callosum | Brain stem | Other |
|---------------|-----------------------------|-----------------|-----------------|------------|-------|
|               | CT  | MRI | CT  | MRI | CT  | MRI | CT  | MRI |                |
| MM            | –   | –   | 4   | 6   | –   | 4   | –   | –   |                |
| WP            | –   | 2   | –   | 2   | –   | 1   | –   | –   |                |
| MD            | –   | –   | –   | –   | –   | –   | –   | –   | Only cytotoxic oedema on MRI |
| KJ            | 2   | 4   | 3   | 8   | –   | 2   | 1   | 2   |                |
| KT            | –   | –   | 2   | 4   | –   | –   | –   | –   |                |
| PW            | –   | 2   | –   | 6   | –   | 3   | –   | –   |                |
| AR            | 1   | 3   | 2   | 3   | –   | –   | –   | 1   |                |
| CA            | –   | 4   | 1   | 2   | –   | –   | –   | –   |                |
| Total number of foci | 3   | 18  | 12  | 31  | –   | 13  | 1   | 3   |
The most frequent locations of DAI injuries are: corpus callosum and subcortical white matter; Brain oedemas are also common in these injuries.

Until recently, the frequency of DAI was hard to evaluate, as it was impossible to visualise them in imaging studies, while the lesions were confirmed by histopathological post-mortem studies (as the injuries of DAI type are a frequent cause of posttraumatic coma, leading to a vegetative state and subsequent death.).

Patients with DAI are most commonly the casualties of high-speed car accidents which lead to intracranial dislocations of brain structures as a result of accelerating-retarding inertia forces. Diffuse axonal injuries are not caused by mechanical tearing but by overstretching and torsion of axons, due to energy waves caused by the injury and penetrating brain structures [9]. At this rate of injury formation, a direct trauma is not required and the lesions may appear in locations distant from the point of direct impact.

Axon injuries lead to axoplasmic effusion into intercellular compartments, and the creation of ball-shaped lesions, so called retraction balls, that appear as early as 3 hours after the injury, and their number increases by the end of the first 24 hours [2]. Damaged but uninterrupted axons undergo posttraumatic depolarisation, with ionic escape, increased aminocid release, and other metabolic disorders, including accelerated glycolysis and accumulation of lactates.

This leads to an inhibition of axoplasmatic transport with an interruption of synaptic connections, and, after a few weeks, there appears Waller degeneration of the nerve fibre and a diffused impaired inflow of impulses stimuli [2,9].

DAI lesions appear mainly in the white matter, around the ventricular system, in corpus callosum, in the region of the thalamus, hippocampus, dorsally-lateral part of the mesencephalon and the superior part of the pons. Corpus callosum is particularly prone to DAI. Initially, it was believed that the injuries of corpus callosum are transmitted from the cerebral falk, but model studies have shown that these are the linear (lateral) and rotary forces (‘shearing forces’) that transmit the shock wave energy in the direction opposite to the head movement and are responsible for the injury of this structure [9,10]. Moreover, the dislocation of intracranial structures in the direction opposite to the direction of the head movement leads to the tension of vessels penetrating brain structures and supplying basal ganglia, which results in microhaemorrhages with diffuse posttraumatic injury of blood vessels (diffuse vascular injury) and concomitant injury of intracerebral tracts, as well as clinical picture of brain stem injury. This type of brain injuries is responsible for severe clinical course and is called ‘shearing injuries’ [2,6,9]. Some extravasations become macroscopically visible. They are most common in frontal and temporal lobes, anteriorly, as well as periventricularly [2,8,9,11].

According to Adams, DAI may be categorised into three groups, depending on the severity of injury, its anatomical location and patient’s survival odds [12,13]:

First degree (mild injury) – diffuse lesions, in perisagittal regions of the white matter of frontal lobes, in periventricular regions of frontal lobes, and in periventricular regions of temporal lobes. Foci in parietal lobes, or internal and external capsule, are less frequent.

Second degree (moderate injury) involves lesions of the first degree and, additionally, lesions in corpus callosum, including haemorrhagic lesions, which significantly deteriorate the prognosis and decrease the odds of survival.

Third degree (severe injury) involves changes of the second degree and, additionally, lesions within the brain stem, pons, and cerebellum, often leading to a vegetative state or death.

Owing to the progress in CNS imaging, including CT and MRI diagnostic procedures, the injuries of DAI type are increasingly better visualised and diagnosed.

Lesions in diffuse axonal injuries are better visualised in MRI images than in CT images.

DAI symptoms found on CT are slight or absent. Only the DAI with macroscopic haemorrhagic lesions is visible in CT images – and that is the cause of low sensitivity of this technique in DAI diagnostics [8–10].

CT examination visualises fine, punctate foci of increased density, at the borderline of the white and grey matter, corresponding to haemorrhagic foci.

Right after the injury, the lesions may be poorly visible on CT but detectable on MRI. On MRI examination, the non-haemorrhagic DAI reveals multiple foci of high signal intensity in T2-weighed images and FLAIR sequence; haemorrhagic DAI reveals additionally the foci of low signal intensity, described as ‘black points’ and ‘corresponding to the foci of haemorrhage’ [6,8].

Lesions in DAI are found in locations typical for the severity of the injury, which was covered by Adams’ classification. In mild injuries, the lesions are limited to the subcortical white matter within the perisagittal and periventricular regions. In patients with more severe injuries, we may observe (additionally) corpus callosum damage, especially in its posterior part. In the most severe cases, the DAI involves the dorsally-lateral part of mesencephalon and the superior part of the pons, apart from the lesions of white matter of the lobes and of corpus callosum [6,11,13].

The diffusion-weighted imaging is a technique producing an image depending on the diffusion of extracellular and extravascular water particles. Diffusion is a physical property of particles that depends on their thermal energy (Brown’s movements). The movements are present at the microscopic level and proceed in all directions. On DWI images, the regions of increased diffusion are dark, while the regions of decreased diffusion are bright (hyperintensive). To exclude artefactive hyperintensive foci in DWI sequences, i.e. so called T2 shine-through effect, the DWI images are compared with the maps of ADC (apparent diffusion coefficient).
In brain injuries, including DAI's, some parts of the brain reveal a decreased perfusion rate due to a diffuse injury of nervous cells and small blood vessels, which results in an inhibited free exchange of water particles in the process of diffusion. As a consequence, these regions generate an intense MRI signal during the diffusion examination and the lesions appear just after a few minutes from the moment of nervous cells injury.

DWI is one of the most sensitive methods visualising the injuries of white matter [9,11,14] which is extremely useful in the detection of axonal injury foci.

A more recent imaging technique, using the diffusion measurements with DTI (diffusion tensor imaging), is applied for the visualisation of neuronal tracts in the white matter of the brain. The technique is based on the knowledge that water particles move much easier along the nervous fibres than across them. Thus, the movement of water particles within the white matter shows the direction of nervous tracts and allows for their mapping with simultaneous evaluation of the degree of injury, which is the most frequent in axonal injuries with lesions within corpus callosum [15,16].

One more modern imaging method useful in the evaluation of results resulting from axonal brain injuries, is the SWI (susceptibility-weighted imaging) – an imaging method based on the evaluation of magnetic susceptibility, which depends on the properties of a given tissue, i.e. on the degree of its longitudinal magnetisation in external magnetic field. Its distribution is evaluated in T2-weighted images that are very sensitive to local heterogeneity of the magnetic field, resulting from the presence of structures of significantly different magnetic susceptibility, e.g. posthaemorrhagic hemosiderin deposits. This is useful in the evaluation of haemorrhagic foci in DAI [17].

A supplementary diagnostic method of diffuse axonal injury is the MRS (magnetic resonance spectroscopy) which may reveal a decreased NAA/Cr ratio [18,19]. The NAA concentration is an indicator of neuronal and axonal density in the examined nervous tissue. Diffuse axonal injury results in the damage of nervous cells, and their subsequent degeneration and atrophy [18,19].

All these methods aim at a comprehensive evaluation of the extent of DAI injuries, with a prognostic assessment of patient's survival odds and potential brain dysfunctions. Lesions within corpus callosum, resulting from cellular degeneration, may lead to the disorders of motor coordination; lesions within the brain stem, to activation system injuries; lesions within the white matter of temporal lobes, to memory disorders, disorientation, behaviour disorders, depression; and the lesions of the white matter of frontal lobes, to dementia.

Diagnosis of axonal injuries should not base solely on CT examination.

Reasonable seems the change of the diagnostic procedure algorithm in patients with suspected DAI, and the performance of an MRI examination (apart from a standard CT) according to the shortened protocol and with the DWI option. This would not extend the time of the diagnostic work-up substantially (which is always of importance in posttraumatic patients) and would ensure a right diagnosis of the brain injury with a prognostic evaluation including future posttraumatic neurological defects.

Conclusions

1. CT method is not sufficient for brain DAI evaluation.
2. MRI is more sensitive than CT in brain DAI detection.
3. Diffusion-weighted imaging allows for a successful and comprehensive diagnosis of brain axonal injuries.
4. New MR imaging methods – DTI, SWI, and MRS – constitute important supplementary methods of DAI diagnosis.