Diffuse axonal injury

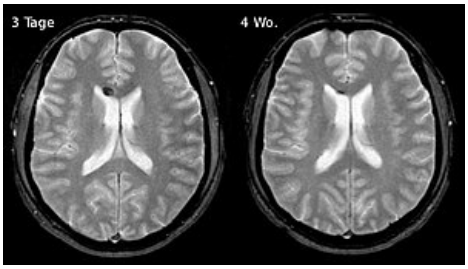
Diffuse axonal injury (DAP) is a multiple microscopic traumatic involvement of axons . Gray and white matter have a different specific weight, and therefore their mutual shear movement occurs during an injury. Axons (white matter) become stretched, damaged and then degenerate.

The myelin sheaths remain primarily intact. The axon is not mechanically severed, but axoplasm constantly flows from it, axon lysis occurs about 24 hours after the injury. On the proximal stumps, sacs filled with axonoplasm are formed, which are visible histologically after a few days. Glia gradually disappear and atrophy of the brain tissue begins, which is macroscopically visible as early as three weeks after the injury. The brain tissue is gradually replaced by cerebrospinal fluid , the brain ventricles enlarge.

There is no clear line between coma and diffuse axonal damage. The previously stated criterion that coma is functional damage and DAP (diffuse axonal injury) organic damage can no longer be used. An organic cause probably also contributes to severe comas, as evidenced by the long convalescence in post-coma syndrome. Diffuse axonal damage is the cause of up to 50% of long-term unconsciousness. Today it is considered the most common brain injury , which is combined to a greater or lesser extent with other types.

Etiology

- Acceleration mechanism of injury, arises as a result of rotational accelerations after impact;
- axons are mainly damaged because they run across the direction of the applied shearing forces.



SWI image of a diffuse axonal injury with small petechiae (right) − in the most severe shearing injury, small vessels are also injured and this is a sign of a serious prognosis.

Physical forces acting on axons during acceleration and deceleration

1. shear forces;
2. tension forces.

The degree of damage to axons depends on the intensity of the applied forces (an overload of at least 80 G is required to cause DAP), which act on the axons once or when smaller injuries are accumulated (for example, in sports injuries).

Classification

Depending on the severity of the damage

1. **Mild** diffuse axonal injury: reversible;
2. **severe** diffuse axonal injury: irreversible.

According to the speed of creation

1. **Primary – shearing injury** – the most severe degree of damage, the forces acting on the axons are so strong that the axons are torn suddenly, at the moment of injury .
2. **Secondary – axonolysis** develops gradually, organelles accumulate at the place of Ranvier notch on the axon and enzymatic activity increases, axonolysis occurs in 24 hours, followed by autolysis of the neuron body and surrounding glia. It can be demonstrated histologically in up to 72 hours. [1]

Clinical picture

The basic symptom is immediate and long-lasting unconsciousness . The severity and consequences depend on the number of damaged axons and the location of the lesion.

- **The neurological findings** are very varied according to the affected nerve pathways;
- disorders of consciousness with focal symptoms;
- there may be decortication rigidity as a reflection of involvement of motor pathway axons at the level of the capsula interna ;
- may be decerebrate rigidity as a result of brainstem involvement ;
- axons in the white matter of the brain , in the corpus callosum , around III. chambers;
- in the case of extensive involvement, deep unconsciousness may occur, trunk lesions appear; severe conditions are often accompanied by hemodynamic swelling and an increase in intracranial pressure .

Diagnostics

- History (head injury, prolonged unconsciousness - more than a few minutes), usually negative CT scan (in the most severe cases, small hemorrhages in the white matter can be detected). The main finding on CT is

generalized brain edema with disappearance of gyrification and reduction of the ventricles.

Auxiliary examinations

- **MRI – small focal changes especially in the area of the corpus callosum , in the subcortical white matter, basal ganglia , thalamus and pontine .**

Therapy

- Treatment focuses on the treatment of accompanying intracranial hypertension (mannitol, corticosteroids, furosemide, barbiturates), there is no specific therapy for axon damage itself.
- Hospitalization in ICU, artificial ventilation (prevents hypoxia of the brain), continuous monitoring of intracranial pressure (prevention of edema), provision of internal environment, nootropic (piracetam , approx. 12 g per day).
- Prevention and treatment of secondary infections (humoral and cellular immunity is greatly weakened in unconsciousness).

Prognosis

- Bad, it is not possible to adjust ad integrum, there is a persistence of various degrees of physical and psychological deficit;
- part of the patients remains in a so-called vegetative coma and a non-negligible part of the patients dies.

Links

related articles

- Craniocerebral trauma
- Comoce

Source

- BENEŠ, Jiří. *Study materials* [online]. ©2010. [feeling. 2009]. < http://jirben.wz.cz (http://jirben.wz.cz/) >.

Reference

1. Source: BENEŠ, Jiří. *Study materials* [online]. [feeling. 2009]. < http://jirben.wz.cz >.
2. ↑Jump up to:a b c Neurosurgery lecture, 5/19/2011, Doc. MD Ing. Jaroslav Plas, ÚVN Prague.
3. ↑Jump up to:a b c d . SAMES, M, et al. *Neurosurgery.* 1st edition. Prague: Jessenius Maxdorf, 2005. ISBN 80-7345-072-0 .
4. ↑ AMBLER, Zdenek. *Basics of neurology.* 6th edition. Prague: Galén, 2006. pp. 171-181. ISBN 80-7262-433-4 .
5. ↑ NEVŠÍMALOVÁ, Soňa, Evžen RŮŽIČKA and Jiří TICHÝ. *Neurology.* 1st edition. Prague: Galén, 2005. pp. 163-170. ISBN 80-7262-160-

1. Přednáška z neurochirurgie, 19. 5. 2011, Doc. MUDr. Ing. Jaroslav Plas, ÚVN Praha.

Literature

- NEVŠÍMALOVÁ, Soňa, Evžen RŮŽIČKA and Jiří TICHÝ. *Neurology.* 1st edition. Prague: Galén, 2005. pp. 163-170. ISBN 80-7262-160-2 .
- AMBLER, Zdenek. *Basics of neurology.* 6th edition. Prague: Galén, 2006. pp. 171-181. ISBN 80-7262-433-4 .
- SAMES, M, et al. *Neurosurgery.* 1st edition. Prague: Jessenius Maxdorf, 2005. ISBN 80-7345-072-0 .
- ZEMAN, Miroslav, et al. *Special surgery.* 2nd edition. Prague: Galén, 2004. 575 pp. ISBN 80-7262-260-9 .

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