White cord syndrome

Presence of intramedullary MRI hyperintensity signal on T2 weighted image in a patient with unexplained neurological deficits following a spinal cord decompression.

Epidemiology

“White cord syndrome” is a very rare condition.

Etiology

It is thought to be the result of acute reperfusion of chronically areas of spinal cord ischemia.

Diagnosis

Its hallmark is the presence of intramedullary MRI hyperintensity signal on T2 weighted image in a patient with unexplained neurologic deficits following a spinal cord decompression.

Treatment

In previous reports patients have improved following steroid therapy and acute rehabilitation [1].

Case reports

2018

Antwi et al. report an additional case of this complication in a 68-year-old man who developed acute left-sided hemiparesis after posterior cervical fusion for cervical spondylotic myelopathy. The patient improved with high dose steroid therapy [2].

2017

A 64-years old male patient with severe neck pain irradiated to both arms, gait disorder and urinary incontinence. He showed spastic tetraparesis, grip weakness and positive bilateral Hoffmann's reflex, with a Nurick scale score of 3 and a Japanese Orthopaedic Association scale (JOA) of 13, Grade I. MRI imaging documented multiple cervical stenosis with voluminous C3–C4 and C5–C6 disc herniations associated to T2-hyperintense myelomalacic area at C3–C4 level.

Patient underwent double-level ACDF with microsurgical discectomy according to Smith Robinson technique and following anterior arthrodesis, first in C5–C6 with the placement of a titanium cage with intrabody screws (Zero P®, Depuy Synthes – Johnson & Johnson – US), then in C3–C4 level with a stand-alone titanium cage (Cervios®, Depuy Synthes – Johnson & Johnson – US). A diamond drill was used to remove osteophythes in both interbody spaces so to increase spinal cord decompression. An autologous fibrin glue was used to ameliorate haemostasis and fusion.

No surgical, nor anaesthesiological complications were observed, all neural structures were respected and intra-operative x-ray showed the correct placement of both cages. During the closure time of the superficial planes, somatosensory and motor evoked potentials suddenly decreased in voltage. When awakened, the patient showed a severe tetraparesis with complete paraplegia and severe motor weakness to upper limbs with diffuse spastic hypertonia.

A neck collar was then placed and an immediate cervical-spine CT imaging confirmed the correct execution of ACDF.

A following cervical MRI showed an enlarged T2-hyperintense area in C5–C6 level

This ischemic-edematous lesion was supposed to be a case of “white cord syndrome” imputable to a mechanism of improper cord reperfusion. A two-days NASCIS III protocol was then performed.

Three days after, a partial recovery in prehensile strength on the right hand (3/5 Medical Research Council Scale, MRC), a partial recovery in flexion of right arm (2/5 MRC), and in flexion of both legs on thighs (2/5 MRC) were observed.

Seven days after the procedure the patient was transferred to a high specialized Rehabilitation Unit with a Nurick score of 4 and a JOA of 6 [3].

2013

Chin et al. report a case of complete loss of somatosensory evoked potentials (SSEPs) during elective ACDF at C4-5 and C5-6 followed by postoperative C6 incomplete tetraplegia without any discernible technical cause. A postoperative MRI demonstrated a large area of high signal changes on T2-weighted MRI intrinsic to the cord “white cord syndrome” but no residual compression. This was considered consistent with spinal cord gliosis with possible acute edema. The acute decompression of the herniated disc resulted in cord expansion and rush-in reperfusion. We postulate that this may have led to disruption in the blood brain barrier (BBB) and triggered a cascade of reperfusion injuries resulting in acute neurologic dysfunction. At 16 months postoperatively our patient is recovering slowly and is now a Nurick Grade 4 [4].

References

[1] , [2]
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