脊椎硬腦膜外動靜脈瘻管：四病例報告 DP074-BN
Spinal Extradural Arteriovenous Fistula: four cases report

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PURPOSE: Spinal extradural arteriovenous fistulas (AVFs) are rare conditions. They are characterized by the presence of an arteriovenous communication, localized to the paraspinal soft tissues and the epidural venous plexus. They could be asymptomatic, or caused compressive myelopathy by mass effect of the enlarged epidural varices. Moreover, when retrograde intradural venous drainage and subsequent venous congestion develops, they could cause progressive myelopathy.
CASES REPORT: The author reports on 4 cases of extradural AVFs. Clinical findings, diagnostic evaluation, treatment and outcome are discussed.
Two cases of spinal extradural AVFs have extradural fistulous locations (both at L3 level) and large epidural venous lakes with intradural venous drainage. They presented with progressive myelopathy. Transarterial embolization (TAE) with N-butyl cyanoacrylate (NBCA) was performed to occlude the fistula and the venous lake in both cases successfully. Regression of cord edema was noted and the symptoms were relieved in one patient and improved in another. The third case presents with cervical radiculomyelopathy. Angiography revealed an extradural AVF at T12 level with epidural venous drainage but without intradural venous drainage. TAE with NBCA was performed to occlude the fistula successfully. The symptoms completely resolved. The fourth case was an incidental finding. Spinal extradural AVFs at cervical level were noted without intradural venous drainage or mass effect on surround tissue. No treatment was needed.
CONCLUSION: Spinal extradural AVFs are rare malformations which differ from dural AV fistulae in many ways. Careful differentiation and evaluation with selective spinal angiography was important before the treatment to be performed.

反覆性顱內結核瘤：罕見病例 DP075-BN
A Rare Presentation of TB: Repeated Attack of Brain Tuberculoma

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There are various forms of central nervous system infection, such as meningitis, tuberculoma, abscess, cerebritis, military tuberculosis, and spinal tuberculous arachnoiditis. The way of scattering included hematogenous dissemination, rupture of a Rich focus into CSF space, or from CSF space to adjacent parenchyma.

There is a 59 year-old male, who had history of hypertension, arrhythmia, old infarction in the midbrain, and old traumatic SAH. He went to the ER because of left side weakness and numbness with ataxic gait and drop foot. The emergent CT didn't show new lesion. But the brain MRI showed cluster of tiny, enhancing nodules in the periventricular white matter of right temporal lobe and bilateral parietal lobes with perifocal white matter edema. The stereotaxic aspiration was done with result of necrotizing granulomatous inflammation and negative finding in PAS, GMS, and AFS. But the sputum AFS was positive. He was treated as tuberculosis with anti-TB drugs. The symptom was subsided quickly. But, 3 months later, he admitted again because of left limb apraxia. The follow-up MRI showed improvement of the primary lesions but with some newly lesions in the right frontal corona radiate, suspect with recurrent tuberculosis. Therefore, the anti-TB drugs were kept used.However,4 months later, another new symptom of headache and progressively memory loss were progressed. The follow-up MRI showed disappear of previous lesions, but with cluster new enhancing nodules in the left frontal-parietal lobes. Then, he was kept treating with rifampicin in the OPD. Then, the symptoms subsided again. The last follow-up MRI showed disappearance of all brain parenchymal tuberculoma lesions.