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CNS Infections 2
Do adjunctive corticosteroid and aspirin improve the outcome of tuberculous meningitis?

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Background: Corticosteroid and aspirin have been reported to improve the outcome of tuberculous meningitis (TBM) but their relative efficacy of corticosteroid, aspirin and combination of both have not been evaluated.
Objective: To compare the outcome of patients with TBM receiving adjunctive aspirin, aspirin + corticosteroids or none of these therapies.
Patients and methods: In a retrospective hospital based study, consecutive patients with TBM during 2008-2014 were included after ethical clearance. The diagnosis of TBM was based on clinical, cerebrospinal fluid (CSF), MRI, AFB, ELISA and PCR findings. The severity of meningitis was graded into stage I, II and III. They received 4 drugs (HRZE) antitubercular treatment with 150 mg/day aspirin (Group I) or prednisolone (0.5mg/kg/d) for 1mo plus aspirin (Group II) or none of these adjunctive therapies (Group III). Outcome was defined at 3 months into death, poor or good.
Results: 135 patients with TBM were included whose median age was 36.4 years and 49.6% were females. 29.6% patients had stage I meningitis, 54.8% stage II and 15.3% stage III meningitis. Group II patients had more severe illness compared to group I and III ($P<0.002$). At 3 months, 24% patients died; 18.2 % in group I, 18% in group II and 34.1 in group III. Complete recovery was more frequent in group II (48.8%) compared to group I (30.6%) and group III (25.9%). Death and functional outcome however were not significantly different in between the groups.
Conclusion: Adjunctive steroid plus aspirin therapy in TBM seems to offer survival and outcome benefit.

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CNS Infections 2
Subtests of the heidelberg neurological soft sign scale that discriminate HIV patients with and without hand

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I have obtained patient and Institutional Review Board approval, as necessary.
Background: Since the introduction of the antiretroviral therapy, Human Immunodeficiency Virus (HIV) mortality has decreased while the prevalence of milder forms of HIV-associated Neurocognitive Disorder (HAND) increased.

The diagnosis of milder forms of neurocognitive disorders is a clinical challenge, since they present with subtle symptoms in the early stages. Neurological Soft Signs (NSS) are subtle abnormalities in motor and sensory performance, usually present in the early stages of these disorders. We showed in a previous report of preliminary results, that persons living with HIV infection have more prevalence of NSS in comparison to healthy controls, and that in the HIV group, persons with HAND have higher NSS than HIV infected persons without cognitive disorders.
Objective: The aim of this study is to identify the specific subtests of the Heidelberg NSS Scale which could better discriminate between HAND (+) and HAND (-) and HIV patients in a cohort of Chilean HIV infected persons.
Materials and methods: 75 HIV patients without history of head injury trauma or opportunistic infections of the CNS were recruited from the HIV clinic, underwent a thorough clinical interview and neuropsychological testing. HAND was diagnosed using NIMH and NINDS criteria. All patients were assessed with the Heidelberger NSS Scale.
Results: In a preliminary analysis including 45 subjects we found motor coordination subtests to better discriminate HIV infected persons with from those without HAND.

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CNS Infections 2
Deficit sciatica post varicella zooster virus infection

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Introduction: Neurologic complications of varicella zoster virus (VZV) are rare and they are dominated by encephalitis. Direct mechanism is implicated. Myeloradiculitis involving indirect mechanism is rarely reported. Sciatica with neurological deficit can be the first manifestation.
Objective: Remembering through a case report the neurologic involvements of VZV infection implicated indirect mechanism.
Case report: A. N a 64-year-old was admitted for bilateral sciatica L5 and S1 with bladder disorders progressively installed two months after intercostal zozter. The examination showed flaccid areflexic paraparesis with a sensory level D10-D11. The spinal MRI showed medullar hypersignal D11-D12 on T2 weighting with meningeal and roots contrast enhancing. CSF study was consistent with aseptic lymphocytic meningitis with negative VZV PCR. The EMG showed lower limb polyradicular lesions. The diagnosis of post herpetic meningomyeloradiculitis was made. Patient improved under aciclovir and corticosteroid. He consulted six months later for recurrence of the same symptoms concomitant to corticosteroids tapering with extension of radiologic lesions. The resumption of corticosteroids was marked by clinical and radiological improvement.
Discussion: Neurological complications of VZV were observed in 5-10 % of cases affecting not only immunodeficient but also immunocompetent persons. Encephalitis and meningitis were the most comment involvements. Myeloradiculitis implicating indirect mechanism with immuno-allergic reaction secondary to antigenic mimetism was rarely reported. Treatment is based on corticosteroids.
Conclusion: VZV is a rare etiology of Myeloradiculitis caused by immunoallergic mechanism and whose only manifestation may be sciatica and neurologic deficit.

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