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Letter to the Editor

Current therapeutic status of corticosteroid in human immunodeficiency virus patients with tuberculous meningitis

Sir,

Human immunodeficiency virus (HIV) poses risk for severe manifestation of TB. These may be in the form of pulmonary or extra pulmonary. According to the level of immunosupression there is risk of developing tuberculous meningitis (TBM). HIV alters pathogenesis, clinical features and prognosis throwing diagnostic and therapeutic challenges.[1]

There are multiple factors that determines the management of HIV coinfected TBM cases viz clinical features; drug interactions and choice of rifamycin; timing of ART; drug induced liver disease (DILI); use of corticosteroids and so on.

Clinical features

As compared to HIV uninfected patients, more lymphadenopathy, hepatosplenomegaly, altered consciousness and impaired cognition is seen. Increased CSF mycobacterium yield is increased due to high intra CNS dissemination.[1]

Timing of ART and IRIS

Paradoxical TB IRIS (immune reconstitution inflammatory syndrome) is frequently encountered during initiation of ART in HIV-TBM patients. It is an exacerbation of preexisting sign and symptoms of TB and hyperinflammatory response within three months of ART. It is immunity restoration attempt by the body.[2]

Choice of rifamycin

Interaction between ATT and ART and overlapping of side effects is one of the major concerns. Since rifampicin is an integral part of ART but it induces CYP450 therefore the level of protease inhibitors and nevirapine is reduced.[3]

Rifabutin which is a rifamycin with less CYP450 property hence less effect on serum level of ATT drugs is often advocated.

DILI-drug induced liver injury

Hepatitis is 20% more common in HIV TBM as compared to TBM not infected with HIV.[4] There is several fold increases in AST/ALT. Therapy is stopped if level of liver enzyme is ≥10 times the upper normal limit.

Corticosteroid use

Along with ATT, corticosteroid is also added as an adjunct. Corticosteroid reduces inflammation in subarachnoid space and also cerebral edema ICT and Incidence of paradoxical reaction. However, it suppresses the immunity and symptoms resulting in growth of the bacteria. Penetration of drugs into subarachnoid space is affected due to reduction in meningeal inflammation.[5] Despite these limitations, it is been used in TBM including HIV TBM as it is observed that corticosteroid may reduce mortality.

Evidence to show the beneficial role corticosteroid is on the basis of Vietnam trial that recruited 545 TBM patients. Out of these, 98 were HIV TBM. There was reduction of death risk at 9 months in HIV TBM cases but not significant (p=0.08). Since number of patients recruited was small it was difficult to comment upon the effect of treatment. This small benefit was the basis of using corticosteroid in management of HIV TBM by many international guidelines. Overall, it was observed that in TBM corticosteroid may reduce mortality but not the morbidity.

LTA4 hydrolase an enzyme that affect leukotriene B (chemoattractant of neutrophils and macrophages) decides inflammation in TBM. LTAH4 has two genotype TT and CC. Desired effect of corticosteroid is appreciated only in LTA4H TT genotype.[5]

Therefore, due to paucity of sufficient data, beneficial role of corticosteroid in HIV infected TBM patient remains uncertain.

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