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Short Communication

Autoimmune Mechanism for Post-Malaria Neurological Syndrome

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DESCRIPTION

The appearance of a syndrome marked by new neurological and/or psychiatric manifestations following recovery from recent malaria infection with no other aetiological cause is increasingly recognized as a distinct entity known as Post Malaria Neurological Syndrome (PMNS) [1]. It is a rare complication of malaria which may pose a diagnostic challenge to the clinicians [2]. Most patients present with prominent psychiatric manifestations progressing to encephalopathy of variable severity. Cases of PMNS have mostly been described in adults following severe *Plasmodium falciparum* infection [1]. Very limited numbers of cases have been reported in the paediatric population due to lack of awareness among clinicians regarding this rare entity [3].

The diagnostic criteria for PMNS are: history of recent symptomatic malaria infection with parasites cleared from peripheral blood (and in cases of cerebral malaria, full recovery of consciousness); and development of neurological or psychiatric symptoms within two months after acute illness [1]. This is in contrast to cerebral malaria or relapse of malaria, which occurs during parasitemia.

The clinical manifestations of post malaria neurological disorders are diverse and include three distinct neurological syndromes: a Delayed Cerebellar Syndrome (DCS), an Acute Demyelinating Polyneuropathy (AIDP) and an Acute Disseminated Encephalopathy (ADE) [2]. Clinical features of PMNS include psychosis, visual hallucinations, catatonia, confusion, apathy, tremors, inappropriate smile, motor aphasia, fever, generalized seizures and impaired consciousness. The onset of symptoms occurs following a latent period of 2-60 days after the clearance of parasitemia. Other viral, bacterial and metabolic causes of altered consciousness should be ruled out before considering the diagnosis of PMNS [2]. Although most cases of PMNS are self-limiting with complete resolution and requiring no specific treatment; steroid therapy has been used in severe cases with good outcomes [4].

The pathogenesis of this syndrome remains unclear. Previously most cases of PMNS had been attributed to mefloquine therapy; however the occurrence of PMNS in patients without any history of mefloquine use does not support this hypothesis [1]. emergence of symptoms in the post-infectious phase following clearance of parasitemia and dramatic response to corticosteroids indicates that the underlying aetiology may possibly be immune–mediated rather than direct neural invasion by the parasite [3,4]. Recent reports also suggest a likely immune mechanism with a possible association with other post-infectious encephalopathies, either Acute Disseminated Encephalomyelitis (ADEM) or autoimmune encephalitis [5,6].

CONCLUSION

The classical lesions of ADEM are widespread areas of demyelination in the deep subcortical and periventricular white matter while in autoimmune encephalitis, brain MRI may be unremarkable or show nonspecific signal hyperintensities. In contrast to ADEM, psychiatric manifestations are more commonly associated with PMNS as in autoimmune encephalitis. Therefore, patients of PMNS with normal brain MRIs could be a part of autoimmune encephalitis. The detection of antibodies against the Voltage-Gated-Potassium-Channel (anti-VGKC) and neurexin-3α in some patients of PMNS provides evidence in favour of underlying autoimmune aetiology. There is a wide array of antibodies associated with autoimmune encephalitis. Additional studies are required to ascertain the nature of antibodies involved in PMNS. Whether PMNS is a part of autoimmune encephalitis or an entity distinct from it, needs to be explored.

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