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ACUTE DISSEMINATED ENCEPHALOMYELITIS AFTER TREATMENT OF SEVERE FALCIPARUM MALARIA

Sir,

A 2½-year-old girl was admitted with high-grade fever of 5 days’ duration and altered consciousness (decreased activity and speech) for 2 days. She was treated elsewhere with intravenous quinine (loading dose only) and dopamine (5 µg/ kg/ min) for malarial fever complicated with shock. There she had developed blackish-blue discoloration of the fingertips, toes, tips of the earlobes; and three similar-appearing patches on the arms and legs. The ambient atmospheric temperature was warm (month of August in Mumbai, India). On arrival at our center, she was still febrile and showed a marked neurological improvement.

Postmalarial neurological syndrome is defined by an acute onset of neurological abnormalities in patients recently recovered from severe Plasmodium falciparum malaria. The lack of malarial parasite on peripheral blood smear examination distinguishes postmalarial neurological syndrome from cerebral malaria. The time from eradication of the parasitemia to development of this syndrome varies from 4 days to 9 weeks.[1] Clinical features are convulsions, confusion-like state, psychosis, cerebellar ataxia, aphasia and myoclonus.[2]

Acute disseminated encephalomyelitis is an acute demyelinating disease occurring 1-3 weeks after a viral infection or vaccination.[3,4] The illness of our patient began after severe falciparum malaria complicated by a rapidly oncoming demyelinating illness after antimalarial therapy. To the best of our knowledge, there are only three cases of ADEM following treatment of malaria. The first had neurologic dysfunction, seizures and hyper-intense lesions in subcortical white matter on T2-weighted MRI images, after recovering from falciparum malaria.[5] The second case had neurological disturbances and multiple high-intensity lesions in the brain after recovery from Plasmodium vivax infection.[6] The third case presented with an ADEM-like illness after treatment of falciparum malaria. Identification of Varicella-Zoster virus infection using polymerase chain reaction on cerebrospinal spinal fluid suggested infection.[7] The neurological syndrome in our patient occurred after anti-malarial therapy and showed a marked neurological improvement.

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Plasmodium falciparum and Plasmodium vivax are the causative agents of falciparum malaria. The incidence of cerebral malaria varies from 5% to 20% in different geographical regions. Other complications of severe falciparum malaria include cerebral malaria, Plasmodium falciparum cerebral malaria (PFCM), and Plasmodium falciparum encephalopathy (PFE). PFCM is a distinct clinical entity with a high mortality rate. Patients with PFCM present with altered sensorium, fever, seizures, and focal neurological deficits. The mortality rate of PFCM is high, ranging from 50% to 90%.[8] The diagnosis of PFCM is based on clinical features and MRI findings. MRI is the imaging modality of choice for the diagnosis of cerebral malaria. MRI shows focal or diffuse hyperintensities on T2-weighted images in the subcortical white matter, corpus callosum and brain stem.[9] The neurological syndrome in our patient occurred after anti-malarial therapy and showed a marked neurological improvement.

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Gangrenous changes were noted in the digits of the hands and toes bilaterally. The spleen was palpable at 3 cm, the liver was palpable with a span of 8 cm, and the right kidney was palpable at the posterior axillary line. The fundi were normal with no retinal hemorrhages or exudates.

A Glasgow coma scale of 8 and exaggerated deep tendon reflexes were noted. The patient had altered sensorium with hypotensive (BP 70/40 mmHg) with acidotic respiratory failure, shock, malignancies (Hodgkin’s disease), collagen vascular diseases (systemic lupus erythematosus), use of vasopressors (dopamine, epinephrine), reaction to drugs (sulphamethazine, penicillin) and miscellaneous causes (like coma, ergotism, acquired hemolytic anemia), etc.[1] Among the infections, meningococcal, streptococcal, E. coli, Pseudomonas, Klebsiella, Proteus, viral gastroenteritis, varicella and disseminated tuberculosis are reported to cause SPG. Most of these conditions (except malaria and DIC) were excluded in our patient on the basis of clinical presentation and investigations.

The basic causative factors for SPG include low cardiac output, vasospastic conditions and small-vessel obstruction. The etiological factors in our case can be explained by the triple insult of the hyperparasitemia, shock and vasospastic action of dopamine. All the cases of SPG with malaria reported in literature had evidence of DIC. Elevated D-dimers reflect the process in our patient. Heavy malarial parasitemia leads to sluggish microcirculation as parasitized red cells along with unaffected erythrocytes form microaggregates (rosettes) and attach to different endothelial cell receptors (cytoadherence).[4] Tendency to form rosettes may differ among different individuals due to host genetic differences such as complement receptor 1 polymorphisms, differences in heparin sulfate molecule type or density on the infected erythrocyte surface or differences in prevalence of other blood group determinants.[5]

The parasites alter the lipid distribution across the red cells activating the intrinsic coagulation system. The parasites also increase the risk of DIC.[6] A positive DIC screen is found in all cases of SPG manifesting with gangrene. The parasites disrupt the capillary networks but the areas of ischemia were demarcated, and two digits of her left hand got auto-amputated. The patient was transfused a total of 60 cc/kg of packed red cells over a week. Limb elevation, early physiotherapy for the toes and fingers and inter-digital packing and protection from trauma were instituted. Over a period of next 1½ months, the gangrene became well demarcated, and two digits of her left hand got auto-amputated distally. She was referred for further surgical management.

Symmetrical peripheral gangrene (SPG) is a rare clinical condition manifesting with acral ischemic damage in two or more extremities, without evidence of obstruction or vasculitis of the relevant artery. SPG has been reported in DIC, low cardiac output states (myocardial infarction, congestive cardiac failure, shock), malignancies (Hodgkin’s disease), collagen vascular diseases (systemic lupus erythematosus), use of vasopressors (dopamine, epinephrine), reaction to drugs (sulphamethazine, penicillin) and miscellaneous causes (like coma, ergotism, acquired hemolytic anemia), etc.[1] Among the infections, meningococcal, streptococcal, E. coli, Pseudomonas, Klebsiella, Proteus, viral gastroenteritis, varicella and disseminated tuberculosis are reported to cause SPG. Most of these conditions (except malaria and DIC) were excluded in our patient on the basis of clinical presentation and investigations.

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The parasites alter the lipid distribution across the red cells activating the intrinsic coagulation and the complement pathways leading to DIC.[6] Additionally, occlusion of small blood vessels with fall in the intra-luminal pressure below a certain critical value (36-60 mmHg) has been demonstrated.[6] This occurs in cases of shock and hypovolemia.[6] In our patient, the disease was arrested in the pro-coagulant phase, which left fibrin deposits in the dermal and acral capillary networks but the areas of ischemia did not extend. This process is irreversible, as barring a few cases, the gangrene leads to amputation of the digits.[7]

Various treatments, viz., epoprostenol (prostacyclin), tissue plasminogen activator,[7] aspirin, vasodilators and sympathetic blockade have been suggested.[6] Such modalities, however, are generally unsatisfactory. Though used in our patient, the role of pentoxiphylline is controversial owing to its side effects like bleeding, drowsiness and hypotension. The primary treatment of this condition includes treating the underlying cause, treatment of the DIC (with heparin) to prevent extension of SPG, avoiding use of dopamine, prompt recognition and treatment of shock and preventing extension of gangrene by avoiding infection and trauma. This case is being reported for the unusual occurrence of SPG in a pediatric patient of severe malarial fever.

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**ABSTRACT**

Internet, from a long time, has opened up a myriad resource of knowledge and applications for academicians, researchers and clinicians alike in all health care professions across the globe. Basic endocrinologists are exploring through bench-top protocols to understand endocrine system and to design therapeutic interventions. Clinicians are required to continuously look for new developments relating to investigation, diagnosis and therapeutic options in their everyday practice for better quality of life of patients. All these require managing a large body of information. Now, these innovative technologies have opened up newer avenues for endocrinologists. As internet is serving the backbone for modern technologies, it is of utmost necessity to use and refine internet applications for future endocrinologists. Increasingly, easy access to internet has dramatically reduced barriers in sharing information among basic and clinical endocrinologists. Considering the growing scope for endocrinologists in the use of internet, it is necessary to understand internet as a source of information and backbone of modern applications. This review illustrates the expanding roles of the internet for endocrinologists and provides a ready-to-use compilation of useful academic, research, clinical resources, and is expected to introduce, stimulate and guide endocrinologists into the realm of WWW.

**Key words:** Endocrinology, internet, World Wide Web

**INTRODUCTION**

Internet, the ever-expanding storehouse of information, contains more than 800 million pages, encompassing about six terabytes of text data on over three million servers,[1] and there is exponential growth in the number of people getting access to the internet.[2] Its power is most strongly understood by scientists, as more and more information is made available through the internet, whether it is about gene sequences, experimental data, chromosome maps or complete journal papers.[3] Internet has emerged as the most powerful platform for exchange of scientific knowledge.[4] It represents significant advancements for the retrieval and dissemination of reliable scientific literature in well-organized, user-friendly formats. Immediate access to all scientific literature has long been a dream of scientists,