FATAL CEREBRAL MALARIA IN A BURN PATIENT - A CASE REPORT

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SUMMARY: A 25 year old male with 50% deep burns died on the 10th post burn day due to cerebral malaria. The clinical features and post mortem findings are described. The need to think of another cause for symptoms which appear like septicaemia particularly in an unresponsive patient is emphasised.

CASE REPORT

A 25 year old male sustained multiple injuries in a bomb blast. He had 50% deep burns over the face and neck with patchy areas of involvement in both upper and lower limbs. In addition he had multiple lacerated wounds over forehead, neck and limbs with foreign bodies in both the eyes. Skiagram of the right hand revealed multiple foreign bodies with subluxation of the first carpometacarpal joint. General resuscitation measures were instituted. Wound debridement was done. Nonviable right index and middle fingers were amputated and the subluxated first metacarpal was fixed with a K-wire. The patient appeared to make steady progress. On the 7th post burn day, he developed clouding of consciousness. Crepitations were heard over both lungs fields. The next day he was unconscious and febrile (39°C). Blood examinations were not conclusive. On the 9th post burn day he was deeply comatose with a temperature of 40°C. The blood smear on that day was positive for Plasmodium falciparum. The cerebrospinal fluid showed increase in lymphocytes and proteins. He was immediately put on parenteral quinine and corticosteroids. The patient died the next day.

Post mortem was done. The brain was congested with petechial haemorrhages throughout the subcortical white matter (Fig 1). Brain section revealed red blood corpuscles with rings of Plasmodium falciparum (Fig 2). Capillaries were found to be stuffed with pigment laden macrophages (Fig 3).

(Fig 1) Brain of the patient showing petechial haemorrhages throughout the subcortical white matter.

(Fig 2) Brain section showing rings of Plasmodium falciparum in R.B.C.s.

(Fig 3) Brain section showing capillaries stuffed with pigment laden macrophages.
of malaria are delay in the diagnosis and inappropriate treatment.

Plasmodium falciparum malaria may become fatal due to the development of high parasite densities in the blood. The pathogenesis of cerebral malaria is complex. Brain sections of the patient show capillaries congested with the parasitised red cells. Due to stasis in the cerebral circulation, necrotic lesions in the cerebral tissues are seen. Stasis also leads to anaerobic glycolysis and lactic acidosis. These changes were present in our patient.

The changes in behaviour, rise in temperature and loss of consciousness started on the 7th post burn day and simulated toxæmia and septicaemia of burns. Any non-immune patient with falciparum infection may show changes in behavioral pattern and cerebral malaria must immediately be thought of. There is urgency in instituting appropriate treatment. Failure to find malarial parasites in the blood does not exclude the diagnosis and it is important to repeat the examination. Anti-malarial drugs must be given. Corticosteroids are supposed to reduce the cerebral edema and might be beneficial in management. The incubation period for Plasmodium falciparum infection is 8 to 15 days and probably the patient was infected before the accident. Conditions like cerebral malaria in burn patients must be rare, and hence this presentation.

References

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