

CASE REPORT

Autopsy in A Case of Severe Malaria (Plasmodium Falciparum)- Opening Pandora's Box of Salient Malarial Pathophysiology

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ABSTRACT

Malaria is a potentially lethal infectious disease caused by a unicellular protozoan parasite, Plasmodium, transmitted by the female Anopheles mosquito¹. Severe malaria, caused by *Pl. falciparum*, has a high mortality rate and is a major cause of death due to malaria². As complete pathological autopsies are scarce in India, findings of disseminated malaria are rarely well documented. We report a 36-year-old male from a non-endemic area with a work station in Bihar, endemic for *Pl. falciparum*, who died of severe malaria with MODS. Diagnosis was based on the provided history, very eminent autopsy table findings, and classic histopathological examination findings.

KEY WORDS: Plasmodium falciparum, cerebral malaria, severe malaria, DIC in malaria

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INTRODUCTION

Malaria, infamously known as swamp fever or paludism, is among the leading causes of infectious parasitic deaths in the world since antiquity, most of them in Africa. 7% of cases reported worldwide are from Asia³. In India, malaria is quotidian in most states and UTs except a few snow-clad northern regions⁴.

Diagnosis of severe malaria is based on the predefined WHO criteria⁵ of the presence of any one of the following criteria, with the presence of asexual forms of Plasmodium falciparum in the blood. 1. Impaired consciousness/coma. 2. Prostration 3. Multiple convulsions 4. Respiratory distress/acidotic breathing 5. Circulatory collapse/shock 6. Clinical jaundice with organ dysfunction 7. Abnormal bleed 8. Hemoglobinuria 9. Hypoglycemia 10. Metabolic acidosis 11. Severe normocytic anaemia 12. Renal impairment 13. Hyperparasitemia(>10%).

CASE PRESENTATION

A 36-year-old soldier from Jammu (UT of Jammu & Kashmir), posted in Bihar, on vacation, died and was sent to

the Department of Forensic Medicine and Toxicology, GMC-J, to undergo a postmortem examination for determination of the exact cause of death. Six days after arrival in his native village in Jammu District, he developed high-grade fever with severe myalgia and chills. His family noted conspicuous yellow discolouration of his sclera and skin. He was checked by a local doctor and diagnosed with S. S. Bilirubin of 10mg/dl and positive Optimal Test for *Pl. falciparum*. He was referred to GMC-J and subsequently admitted to the emergency department. He suffered a fresh onset of seizures, hematuria, coma and respiratory distress (was on assisted mechanical ventilation); however, he succumbed to the parasitism. During admission, his Hb and HCT were markedly decreased with a low normal MCV and MCH, Serum Bilirubin shot past 17mg/dl, had metabolic acidosis on ABG, and his Blood Urea levels were high.

For Insurance claims, we needed an autopsy for the exact cause of his untimely death, as falciparum is hyperendemic

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in Bihar, his workplace. Thus, we got a chance to an academic treasure-trove of an autopsy, by mere fortuity.

RESULTS

EXTERNAL EXAMINATION: External examination was done approximately. 18 to 22 hours postmortem. The corpse was a well-nourished young male, 36 years old, 5'8'' ft in length, with prominent icterus of skin all over the body. Rigour mortis had fully developed in all 4 limbs. Livor mortis was seen on the back, except for contact pallor, reddish purple. No sign of decomposition was noted. No injuries were seen externally. Purpuric blotchy patches were seen on his face and chest- an ominous sign of DIC. He had crusted herpetic lesions over his lips, peri-orbitally and around his nose. Hospitalisation artefacts were also noted.

AUTOPSY FINDINGS

HEAD: Brain and skull were intact, with no injuries. The brain had marked venous congestion with unambiguous straining and a tawny hue to the external surface. Marked oedema with flattening of sulci and gyri was noted. The brain weighed 1.4kg and on C/S, multiple profuse petechial haemorrhages were seen in the white matter, giving it a salt & pepper appearance.



Fig. 1: (a) Classic cerebral oedema with flattening of sulci & gyri, and marked congestion of cerebral vessels (unfixed); (b) Fixed brain on grossing showing white matter petechial haemorrhages with insert of closeup section of same.

NECK, TRACHEA AND OESOPHAGUS:

The mucosa of the oesophagus as well as the trachea was hyperemic with blood-tinged frothy mucus present.

THORAX: There was blood-stained fluid flooding out of the opening of the chest cavity (approx. 100ml). There was no injury to the chest wall.

Lungs: Upper and mid lobes were congested and frankly heavy, with a dusky hue to the surface. Following fixation on C/S, petechial haemorrhages were seen. Lower lobes were dark with hematoma formation on the back surfaces. The right and left lungs weighed 650 g and 530 g, respectively.

Heart: The heart had increased pericardial fat deposition. The circumference of the tricuspid valve was 9.5cm, the mitral valve 9cm, the pulmonary trunk valve 5cm and the aortic valve 4.8cm, respectively. The RV wall was 2.8mm in thickness, and the left ventricular wall was 11mm thick. The anterior wall of the main aortic trunk showed atheromatous deposits on gross examination. Coronary vessels appeared normal. The heart weighed 340 g. A huge gob of PM clot, 4x4x3cm, was found in the right atrium, beautifully demonstrating chicken fat on black currant jelly appearance.

ABDOMEN AND PELVIS

The stomach contained 50 mL of brownish liquid. Spleen was prodigiously enlarged, inky black and extremely fragile to touch, measuring 15x8x6cm. The liver was overtly enlarged, grey to slate black, with a marked stretching and snagging of the capsule. It weighed 1.46kg. Duodenum, intestines, colon and pancreas showed no remarkable changes, except for the tawny hue to serosal surfaces. Kidneys were edematous and mildly enlarged. On longitudinal section, the pyramids were abnormally hyperemic, and the renal pelvis had some petechial haemorrhages. Both testes were congested.

HISTOPATHOLOGY

Histopathological examination was done at the PG Department of Pathology, GMC-Jammu. The following findings were noted:

BRAIN: Capillary channels were congested and jammed up by RBCs in a conglomeration. Ring forms of falciparum were seen in these RBCs along with numerous fairly conventional ring haemorrhages, aka "Durck's granulomas", surrounding the capillaries like a halo.

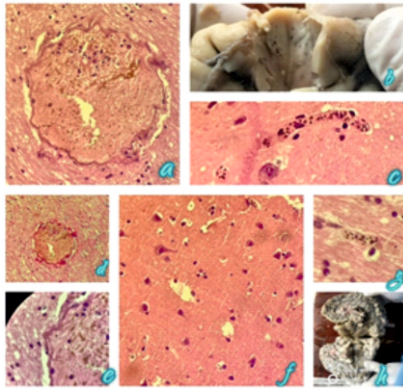


Fig. 2: HPE of the cerebrum (a, e) Quintessential capillary occlusion;(b, h) Gross appearance of brain with punctate hemorrhages and greyish discoloration of cerebral hemispheres (c, g) High power magnification showing parasitized capillary; (d) Scanner view of classic Durck's granuloma; (f) Ischemic changes with red neurons.

HEART: The myocardial morphology was not distorted, but the accompanying clot showed the presence of ring forms of the falciparum parasite. The aorta and LAD artery showed atheromatous plaques with no significant lumen narrowing.

LIVER, SPLEEN & LUNGS: showed exodus of the malarial pigment, hemozoin, everywhere in parenchyma and presence of ring forms in parasitised RBCs in capillary lumens. Phagocytic cells showed marked hyperplasia with dogmatic malarial pigment depositions. GB showed a thinned-out mucosal lining.

KIDNEY: The kidneys showed focal areas of tubular necrosis.

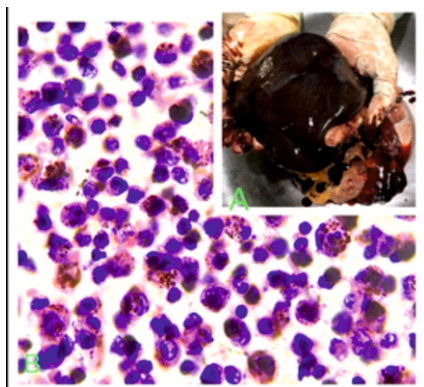


Fig. 3: (A) Typical inky black enlarged, fragile spleen(unfixed); (B) HPE of spleen with parasitized rbc's in venous sinuses and hemozoin blotting of Littoral macrophages.

DISCUSSION

In India, prevalent forms of malaria are: *P. vivax* (25-30%), *P. falciparum* (25-30%), mixed *P. vivax* and *P. falciparum* (4-8%) and in focal regions in Karnataka, *P. malariae* (<1%). *P. ovale* is restricted to West Africa. *P. knowlesi* is a zoonotic infection where macaque monkeys serve as primary hosts, which is confined to Southeast Asia. However, most mortalities are of severe *P. falciparum*⁶. The reason for this is that *P. falciparum* has a purported unique property to induce cell membrane changes in RBCs at EMP with the formation of 'knobs' via the 'var' gene products⁷. This causes the RBCs to adhere to capillary endothelial cells via CD36, E-selectin, I-CAM, and V-CAM on the endothelium, thereby causing widespread thrombosis. Erythrocytic schizogony is in capillaries of vital organs, and only ring forms are usually seen in peripheral circulation. The *P. falciparum* shows egalitarianism, thereby attacking RBCs of all ages impartially, elucidating the severity of anaemia.

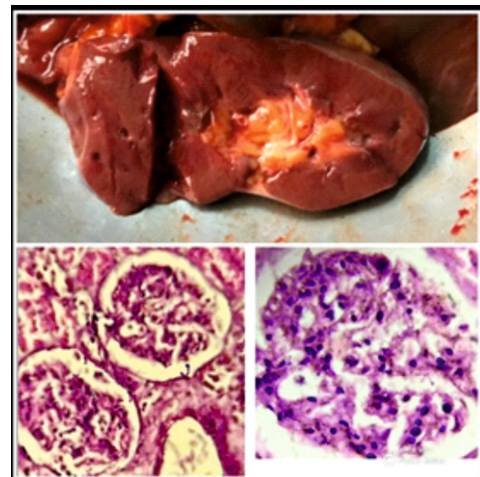


Fig. 4: Dusky discoloration with congestion of Kidney. Microscopic Examination showing dusting of the glomeruli with hemozoin pigment.

HPE was very important here to grasp the essence of the disseminated spread of the parasite. We incorporated the H&E method of staining. Malarial pigment was seen in brain capillaries, heart and the PM clot, liver, spleen, B/L lungs, and kidney, which confirmed the disseminated presence of the protozoa, explaining his rainbow spectrum of signs and symptoms!

The brain had haemorrhages, especially in the white matter, which differ from viral encephalitis, where hyperemia is

mostly in the substantia grisea. Brain capillaries were clogged with RBC clusters. Parasitisation and black malarial pigment were only seen in intraluminal RBCs. Durck's granuloma, i.e. a reparative reaction of glial cells arranged circumferentially around the blocked capillary lumen like an aureole!

Liver has a typical slate grey colour because of hemozoin pigment diffusely spread into the parenchyma⁸. The most common cause of splenomegaly in the tropics is malaria. The red pulp- both venous sinuses and cords of Billroth expand. Littoral cells are deeply pigmented, befitting their black discolouration. Gross and microscopic examination of the kidney revealed early tubular necrosis, explaining acidosis on ABG, but not the typical pigmentation of glomeruli, still pertinent with falciparum malaria⁹.

According to WHO criteria, cerebral malaria as a cause of death should be based on the presence of malarial pigment in the brain, which must be confirmed histologically. The amount of pigment depends on sequestration density, but death still could be due to failure of other vital organs¹⁰.

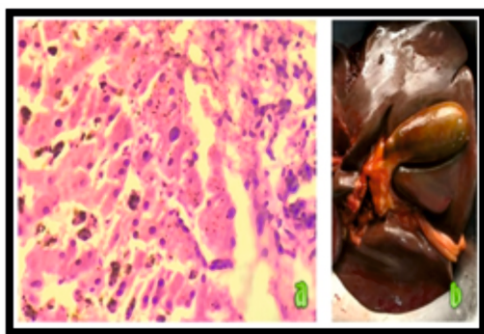


Fig. 5: Tawny discoloration of liver with massive hepatomegaly (unfixed); Histopathological picture of the liver with parasitized RBCs in sinusoidal spaces.

CONCLUSION

In our prodigious case, most criteria of the WHO for severe classification of malaria were all in attendance. The cause of death was MODS caused by disseminated falciparum malaria infection. Our study, therefore, further reinforces HPE as an indispensable requisite of a necropsy, rightly

justifying the name of the discipline as 'Forensic Pathology'!

Conflict of Interest: None

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Written informed Consent: Taken

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