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RESEARCH ARTICLE

SEVERE PLASMODIUM FALCIPARUM MALARIA COMPLICATED BY ACUTE MYOCARDITIS(CASE REPORT)

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Abstract

Although there are a variety of tools and modalities for diagnosing and treating malaria, it is still regarded as one of the most common diseases with high mortality and morbidity1. Different unusual presentations can be seen in the form of ARDS, myocarditis, and hemophagocytosis2, and a high suspicion should be raised to prompt early aggressive antimalarial therapy and reduce complications.

Introduction:-

Till 2020 Malaria consider one of the most severe public health diseases.lt is a leading case of morbidity and mortality (Abdul H. Mohsen, 2001)[3]. It is a treatable disease with symptoms range from mild to life threatening complication if left untreated[4]. It is a disease that can be classified as complicated/uncomplicated.complicated Malaria is characterized by the following symptoms altered behavior, impaired consciousness, coma, severe anemia, hypotension, hypoglycemia, DIC, ARDS, AKI and Parasitemia level of more than 5%.

Cardiac involvement is a controversy, Previous study (PaolaCostenaro et al, 2011)[6] discussed very rare cardiac involvement.

On the other hand,a recent one (Ray H. N., 2017)states that cardiaccomplications can be common especially with Plasmodium Falciparum[7].

Case presentation:

We are presenting a case of 21 years old male who presentsto the ED with fever associated with jaundice and rigors for 14 days. Other unremarkable history, 3 weeks prior to patient presentation, he was in endemic area of Malaria without taking any chemoprophylaxis against Malaria.His clinical examination showed a fully conscious, alert, and oriented male. However, looks severely jaundiced, not in respiratory distress, hemodynamically unstable bp: 77/40 pulse:97 oxygen saturation maintained on room air, not tachypnea.

Investigations showed hemoglobin: 13.5 units, Platelets 38 -WBC: 6.8 units, Liver function test showsunits mainly conjugated with moderate raise in transaminase.

Coagulation show impending DIC,Peripheral blood film came positive for plasmodium falciparum Malaria with Parasitemia level of 5%, RFT within normal, (Laboratory results in table: 1)

Chest x-ray did not show specific abnormality

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Patient was kept in the critical area section forclose observation due to hypotension. He wasresuscitatedinitiallyby IV fluid. However, with unsatisfactory response and required to start norepinephrine.After few hours,the patient started to complainof chest discomfort and shortness of breath. Upon changing in clinical condition, electrocardiogramwas performed and showedischemicST –T changes, along with signs of lung congestion on the repeated chest x-ray.

Cardiology team was consulted, and echocardiography showed global hyperkinesia, reduced ejection fraction 30-35%, and positive troponin. Diagnosis of acute myocarditis was suspected, and most likely induced by the Malaria Falciparum Parasitemia, and recommendations for conservative management along with antimalarial medications.

Patient was admittedto intensive care for 4 days for close observation, started on prophylaxis anticoagulation and anti-failure medications then transferred to general ward for 3 days and discharge home on anti-failure medication.

Follow up Echocardiography, after about 3 months, that showed normalization of the myocardium.

	Normal range	Day 1	Day 7
WBC	3.5–11.0	6.8	5.3
RBC	4.1–5.65	5.1	4.4
HAEMOGLOBIN(G/DL)	12.5–16.9	15.3	12.5
PLT	110–330	38	308
CREATININE	<1.3	1.08	0.46
Sodium (mmol/L)	135–145	124	138
Calcium (mg/dl)	8.5–10.5	6.6	7.67
Potassium(mmol/L)	3.5-5.1	3.9	4.5
Chloride(mmol/L)	101-109	91	108
Lactic dehydrogenase (IUIL)	200–420	573	376
AST (IUIL)	<37	99	55
ALT (IUIL)	<53	172	105
Total bilirubin (mg/dL)	0.3–1.5	17.3	3.7
Direct bilirubin (mg/Dl)	0.1–0.6	13.6	1.39
Creatine phosphokinase (IUIL)	<200	51	30
Troponin I (NG/ML)	0-0.07	0.224	
PT (seconds)	11.0–13.5	16.7	13.3
PTT (seconds)	25–38	53	37
INR	0.7–1.2	1.31	1
CK-MB	3 - 5%	56.6	18.6
ALBUMIN	3.4 - 5.4	2.36	3.19

Discussion:-

Etiology:-

The heart was thought to be harmed in severe malaria in the past, with up to 14% of fatal cases attributed to a cardiac cause. Recent experience, however, has revealed that cardiac involvement is a rare consequence of malaria. Nearly all recorded cases of malaria with cardiac problems are limited to Plasmodium falciparum infection. Cardiac problems, on the other hand, rarely persist after malaria therapy has been completed (F. Brunel, 2003).

Malaria has been a major selective force on humans, and numerous erythrocyte polymorphisms that confer resistance to severe malaria have emerged. Although the importance of the ABO blood group system to protection against severe malaria has received little attention, Plasmodium falciparum resetting, a parasite virulence characteristic linked with severe malaria, is reduced in blood group O erythrocytes compared to groups A, B, and AB. We hypothesized that blood group O confers resistance to severe falciparum malaria via a decreased resetting mechanism. (Rowe et al., 2007)

Pathology:-
Though the specific pathophysiological link between cardiac injury and malaria is unknown, recent research has suggested some theories. The possible cause of myocardial ischemia is capillary blockage caused by parasites and parasitized red blood cells adhering to capillary endothelium via cytoadherence mediated by strain-specific erythrocyte membrane adhesive protein, and this sequestration of red blood cells may also interfere with the microcirculatory flow of the heart.

However, cytoadherence is more common in P. falciparum malaria than in P. vivax malaria, which could be owing to reduced parasite density. The theory of cytokines - Some research has connected cytokine-mediated endothelial activation to complex P. vivax malaria, which could be a source of temporary myocardial ischemia. This is a better explanation for how lungs and myocardial damage are involved in our instance. In complicated P. vivax malaria, a variety of cytokines (tumor necrosis factor alpha, interleukin-10, intercellular adhesion molecule-1, and vascular cell adhesion molecule-1) as well as vivax-specific "malaria toxins" are released, which are thought to cause organ-specific inflammation, increased alveolar-capillary membrane permeability, capillary leakage, and leukocyte aggregation. (Singh, Dinkar& Singh, 2020)

Applicable to our case: -
It is unknown what is the best way to treat Knowles malaria. Chloroquine and quinine have been used to treat P. Knowles infection satisfactorily, but the therapeutic efficacy of other antimalarial drugs is unknown. Artemisinin-derivative combination therapy is presently the WHO's preferred treatment for uncomplicated falciparum malaria and is becoming more widely recommended for no falciparum malaria; nevertheless, its efficacy in Knowles malaria is uncertain. Similarly, intravenous articulate is now the therapy of choice in adults with severe falciparum malaria, but the therapeutic response to this regimen in severe Knowles malaria is uncertain. We used Artemisinin derivatives to treat simple and severe knowlesimalaria. (William, T., Menon2011)

In the present case, complex Malaria is defined by symptoms such as altered behavior, decreased awareness, coma, severe anemia, hypotension, hypoglycemia, DIC, ARDS, AKI, and a Parcitemia level of more than 5%.

Our findings suggest that the frequency of primary cardiac complications in severe Plasmodium falciparum malaria may be underestimated, particularly in adult patients with cardiovascular risk factors (obesity, smoking, diabetes, hypertension, advanced age), but also in cases of unknown or silent underlying cardiomyopathy.

Conclusion:-
Damage to myocardial cells detected by troponin T level, antimalarial should be provided immediately to reduce furtherscomplications, anti-failuremedications should be given if reduced ejection fraction, follow up recommended with echocardiography

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