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Case Report

Complete heart block - A rare complication of severe malaria

Anuradha Sanadhya[1], Priya Bhardwaj[1]*, Aishwarya Sindhur[1], Sakshi Setia[1], Akanksha Sharma[1]

[1]*Dept. of Pediatrics, RNT Medical College, Udaipur, Rajasthan, India*

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ABSTRACT

Tumors, parasitic infections, pyogenic and granulomatous infections may involve the conducting system and cause complete heart block. These are however very rare causes of CHB and maybe regarded as clinical curiosities. Very few such cases have been reported in literature. We are reporting a case of 14-year-old male who presented after an episode of syncope with history of fever for the last 10 days. At presentation, patient had bradycardia and ECG was suggestive of complete heart block. His labs were suggestive of multiorgan dysfunction and card test for malaria came positive for P. vivax and P. falciparum.

1. Introduction

Malaria is a protozoan disease transmitted by the bite of infected female anopheles' mosquito. The most important of the parasitic diseases of humans, malaria causes ~ 1200 deaths each day. Almost all deaths due to malaria are caused by falciparum malaria which causes severe illness.Severe malaria is a critical illness where many organs may be affected simultaneously or sequentially. However, it is not clear yet, as of which organ will be failing and when.[1]

Severe malaria is manifested by one or multiple of the following manifestations-Unarousable coma/cerebral malaria, acidemia/acidosis, severe anemia, renal failure, non-cardiogenic pulmonary edema, hypoglycemia, bleeding/DIC and convulsions.[1]

Hypotension, shock, circulatory collapse, with impaired hemodynamic functions have been observed occasionally in severe malaria patients. Severe malaria is more common in P. falciparum because of high density parasitemia, excessive proinflammatory cytokines production.[2] Very few studies have been carried out regarding the cardiac function in severe malaria. The pathophysiology of cardiac dysfunction in severe malaria has not received due attention. Here, we report a case of PV/PF positive malaria presenting with Stokes Adams syncopal attack.

2. Case Report

14-year-old male child, born out of non-consanguineous marriage, first in birth order, was brought to Pediatric ICU in altered state of consciousness. H/o fever since 10 days-intermittent type of fever, high grade, associated with chills and rigors, no diurnal variations present. Fever was relieved on medication; no exacerbating factors present. This was associated with diffuse abdominal pain in the last one week- dull, boring type of pain, no relieving or exacerbating factors. History of nausea and vomiting in the past 1 week- non bilious, non-projectile type of vomiting, present occasionally, relieved on taking Domperidone which was prescribed at a local hospital.

Attendants also complain of patient having chest pain for the last 2 days- constricting type of chest pain, diffusely present all over precordium, no radiation of pain, associated with shortness of breath (increased on minimal activity,

* *Corresponding author*.
E-mail address: bhardwaj.priya697@gmail.com (P. Bhardwaj).

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relieved on rest), excessive perspiration and a feeling of impending doom. There was history of 1 episode of loss of consciousness just before reaching hospital, which was associated with abnormal jerky body movements, lasting for a few seconds, resolved by itself. This was followed by post ictal confusion in which state patient presented to PICU. Patient was not a known case of any cardiac disorder or Seizure disorder. Past. History and family history was not significant.

On examination, child was thin built and moderately nourished, disoriented, with a GCS of 12/15 E4V4M4 . Patient was afebrile, HR was 50 / minute, regular rhythm, feeble volume pulse. BP was 70/50mm of Hg. Respiratory rate- 44 per minute. Pallor present, icterus present. No evidence of cyanosis, clubbing, lymphadenopathy, edema or dehydration.

On systemic examination, no evidence focal neurological deficit present, no signs of meningeal irritation or raised ICT present. Abdomen was mildly distended with hepatomegaly ~ 2cm from MCL, no e/o ascites. Tachypnea present with normal vesicular breath sounds present in all lung fields, no adventitious sounds present.

On cardio vascular system examination , heart sounds were normal, bradycardia present, no murmurs appreciated.Continuous ECG monitoring was done and was suggestive of bradycardia with complete atrio ventricular dissociation, idioventricular rhythm with regular PP and RR interval, implying a grade III AV block or complete heart block. (Figure 1)

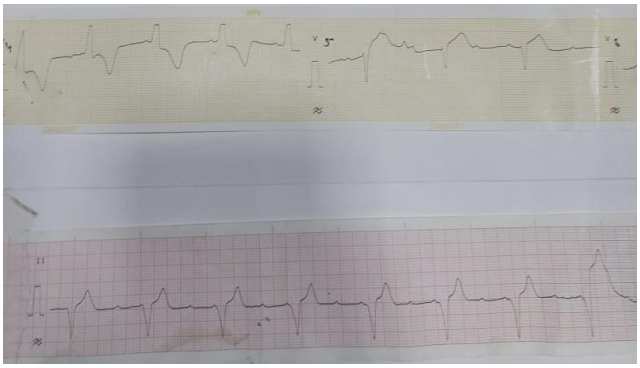

Figure 1: ECG at presentation, suggestive of a complete heart block.

Cardiology opinion was taken and the patient was subjected to transvenous temporary cardiac pacing to stabilize the rhythm. (Figure 2)

The hematologic and biochemical investigation revealed- Hb 10.5g%, WBCs 16K with granulocytic predominance. PLT 88K.Urea 155, Creatinine 1.49, Total Bilirubin/Direct Bilirubin 1/0.4, SGOT/SGPT 12460/6760, AlkPhosphatase 420, Total Protein/ Albumin 6.4/3.3. Calcium 9.8, Sodium 127, Potassium 6.0, Chloride – 94.Random Blood Sugar was 139mg/dl.

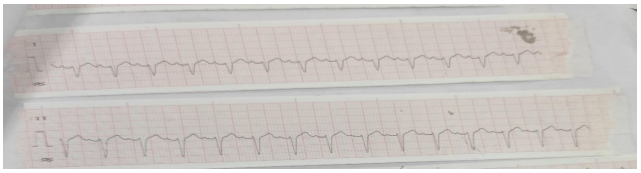

Figure 2: Establishment of sinus rhythm after pacemaker insertion.

Pyrexia Profile revealed Widal negative, Dengue ELISA for IgM equivocal, MP card positive for- P. vivax and P. falciparum ABG at admission was s/o metabolic acidosis. Blood and Urine cultures came sterile.CXR was. suggestive of normal cardiac silhouette, normal lung fields. USG abdomen s/o altered echo texture of liver with moderate hepatomegaly, mild ascites, RPD grade 1, mild pleural and pericardial effusion. All above mentioned investigations were suggestive of complicated malaria.

The child was resuscitated with IV fluids, atropine, temporary cardiac pacing, which eventually lead to an improvement in consciousness as well. The patient was also given broad spectrum IV antibiotics, IV artesunate and corrective measures pertaining to AKI.

Sinus rhythm was established and pacemaker removed after 1 week. Repeat ECG (Figure 3) was suggestive of residual Right Bundle Branch Block associated with Right axis deviation. 2D echo was done, s/o normal biventricular function, intact septae, mild TR and no RWMA.

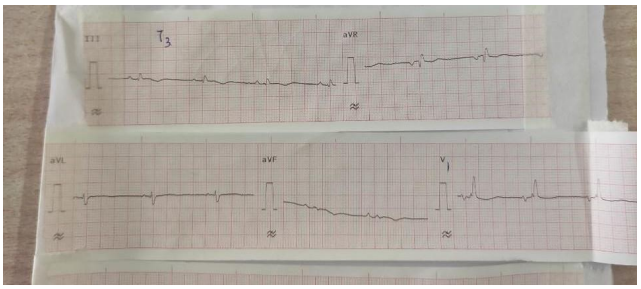

Figure 3: Repeat ECG suggestive of a residual right bundle branch block.

Repeat haematological and biochemical investigations done after 7 days of anti malaria therapy were within normal limits. Patient was discharged after a further observation of 1 week.

3. Discussion

Cardiac involvement in severe malaria has been undermined in literature. Very few studies have been carried regarding the same.

In 2004 and 2005, Ehrhardt et al have demonstrated raised cardiac enzymes in complicated malaria.[3] Similarly, Yacoub et al assessed ejection fraction by echocardiography

which significantly reduced on admission compared with discharge.[4]

P falciparum can cause severe infections and can be complicated by ARDS, acute kidney injury, hypoglycaemia, cerebral malaria and metabolic acidosis. Cardiac complications due to malaria or the drugs used in its treatment have been reported to be infrequent.[5]

Malaria can affect the heart by causing myocarditis[6] and ischaemic heart disease.[7] Cardiac dysfunction has been reported in children with hypovolemia and metabolic acidosis as a consequence of severe malaria.

The central pathogenic mechanism in severe malaria is due to cytoadherence, rosetting, agglutination and decreased deformability of RBCs. This results in sequestration and microcirculatory impediment, lactic acidosis,[8] increased production of proinflammatory markers, capillary leakage, oedema and shock.

Along with this, there is role of direct myocyte damage due to plasmodium toxin- glycosyl phosphatidyl inositol (GPI) which has been shown to augment apoptosis in cardiomyocyte culture. Along with Plasmodial factors, host factors also contribute to myocardial injury. Proinflammatory cytokines like TNF alpha, impair myocardial function via negative ionotropic effects. The anti-malarial medications further complicate the picture.

An already existing cardiac pathology may be worsened by malaria and its management. In our case however, patient did not have any existing cardiac pathology and the conduction block could only have been due to sequestration of red cells leading to downstream ischemia in coronary circulation.

Other Infectious causes of CHB include Chagas disease, Lymes, Syphilis, Tuberculosis, Diphtheria, Toxoplasmosis

4. Conclusion

Involvement of conducting system of heart, although rare, is a known tendency in infections like malaria.

5. Source of Funding

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6. Conflict of Interest

None declared.

References

1. White NJ, Ashley EA, Malaria. Malaria. In: Jameson JL, Fauci A, Kasper D, Hauser S, Longo DL, Loscalzo J, et al., editors. Harrison's Principles of Internal Medicine. 20th edn. India: McGraw Hill / Medical;.
2. Kliegman R, Geme J. Nelson Textbook of Pediatrics, 2-Volume Set, 21st Edn. Philadelphia, PA: Elsevier - Health Sciences Division; 2019.
3. Ehrhardt S, Mockenhaupt FP, Anemana SD, Otehwamah RN, Wichmann D, Cramer JP. High levels of circulating cardiac impairment in African children with severe Plasmodium falciparum malaria. *Microbe Infect*. 2005;7(11-12):1204–10.
4. Yacoub S, Lang HJ, Shebbe M, Timber M, Ohuma E, Tulloh R, et al. Cardiac function and hemodynamics in Kenyan children with severe malaria. *Crit Care Med*. 2010;38(3):940–5.
5. Bethell DB, Phuong PT, Phuong CX, Nosten F, Waller D, Davis TM, et al. Electrocardiographic monitoring in severe falciparum malaria. *Trans R Soc Trop Med Hyg*. 1996;90(3):266–9.
6. Mohsen AH, Green ST, West JN, McKendrick MW. Myocarditis associated with Plasmodium falciparum malaria: a case report and a review of the literature. *J Travel Med*. 2001;8(4):219–20.
7. Jain K, Chakrapani M. Acute myocardial infarction in a hospital cohort of malaria. *J Global Infect*. 2010;2(1):72–3.
8. Mishra S, Behera PK, Satpathi S. Cardiac involvement in malaria: an overlooked important complication. *J Vector Borne Dis*. 2013;50(3):232–5.

Author biography

Anuradha Sanadhya, Professor

Priya Bhardwaj, Junior Resident

Aishwarya Sindhur, Junior Resident

Sakshi Setia, Junior Resident

Akanksha Sharma, Junior Resident

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