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Coexisting Acute Respiratory Distress Syndrome, Guillain-Barré Syndrome, and Falciparum Malaria in A 16-Year-Old Female: A Diagnostic and Therapeutic Challenge

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Abstract
This study highlights the complexities of overlapping life-threatening conditions—acute respiratory distress syndrome (ARDS), Guillain-Barré syndrome (GBS), and falciparum malaria—that must be diagnosed and managed in resource-poor settings. A 16-year old young girl presented with 7day history of fever, diarrhea, lower limb weakness, and rectal bleeding. Initial screening confirmed Plasmodium falciparum malaria. She subsequently developed ARDS and GBS, evidenced by albuminocytologic dissociation of cerebrospinal fluid. In spite of mechanical ventilation, intravenous artesunate, and corticosteroids, the patient deteriorated and died on day 8. The case points to the necessity of early diagnosis of GBS in malaria-endemic regions and advocates for multidisciplinary approaches in resource-constrained environments.

Keywords: ARDS, Guillain-Barré syndrome, coagulopathy, resource-limited settings.

Introduction

Severe Plasmodium falciparum malaria continues to be a major global health problem, especially in endemic areas, commonly resulting in life-threatening conditions such as cerebral malaria, acute respiratory distress syndrome (ARDS), and multi-organ failure [1]. ARDS, is an extreme hypoxemic respiratory failure, is caused by pulmonary inflammation induced by cytokines during severe malaria [2]. Simultaneously, neurological conditions such as Guillain-Barré syndrome (GBS), although rare, are being reported more often in malaria-endemic countries, making patient management more complex and challenging [3].

GBS is immune-mediated polyneuropathy mostly triggered by an infection with Campylobacter jejuni or viral pathogens, although other evidence reveals a causative relationship with parasitic infections when observing, for example, malaria [4]. The Pathology occurs because of molecular mimicry where the immune system is induced to attack components of peripheral nerves with subsequent demyelination or axonal injury [5]. This complicates the diagnosis of GBS in malaria patients with superimposing presentations, as well as limited availability of cutting-edge diagnostic machinery in resource-restricted settings [6].

This case report presents a young female patient with fever, diarrhea, and worsening weakness, ultimately diagnosed with severe malaria complicated by gastroenteritis. Rather, her rapid clinical decompensation with respiratory failure and neurologic involvement revealed a more complex interaction of ARDS, GBS, and falciparum malaria. This case highlights the need for early recognition of malaria-related neurological complications and a multidisciplinary approach to address them in resource-limited environments.

Case Presentation
A 16-year-old girl possessing no history of any disease, was admitted with fever, lose stool, and progressive lower limb weakness for seven days however, dyspnea and rectal bleeding developed on day 4.

Clinical Findings
- Vital signs: Temperature 100^0F, heart rate 120/min, respiratory rate 38/min, SpO_2 85% on room air.
- Neurological: Flaccid paraparesis (power 2/5 in lower limbs), absent deep tendon reflexes.
- Abdominal: Mild tenderness; no organomegaly.

Diagnostic Assessment
- Laboratory: Hemoglobin 7.6 g/dL, platelets 160,000/mm^3, leukopenia (3.8×10^9/L).
- Malaria: Positive peripheral smear and rapid diagnostic test for P. falciparum [1].
- Imaging: Bilateral diffuse infiltrates on chest X-ray [2].
- Lumbar Puncture: Raised protein (90 mg/dL) with normal cell count, in keeping with albuminocytologic dissociation [3].
- ABG: (low O2: 44–54 mmHg, high pH: >7.45, low CO2: 20–28 mmHg, low K^+: 2.5–2.9 mmol/L, low Ca^{2+}: 0.52–0.97 mmol/L, mildly elevated lactate: 1.7 mmol/L) (Type II respiratory failure).
- PT: 17 sec, APTT: 30 sec.
- Ultrasound-guided ascitic tap: postponed due to risk of injury.

Therapeutic Intervention
- Malaria: Intravenous artesunate (2.4 mg/kg/dose) according to WHO guidelines [1].
- RDS: Mechanical ventilation (ARDSnet protocol with low tidal volume) [2].
- GBS: Intravenous methylprednisolone (1 g/day) as IV immunoglobulin was not available [4].
- Supportive: Tranexamic acid for bleeding per rectum, empiric antibiotics, and fluid resuscitation [5].

Follow-up and Outcome
The patient developed septic shock and refractory hypoxemia. She succumb on day 8 despite maximal supportive care.

Discussion
This case illustrates the diagnostic and therapeutic dilemmas of treating overlapping critical illnesses in malaria-endemic, resource-constrained environments.The patient's progressive weakness and areflexia raised suspicion of GBS, aided by albuminocytologic dissociation in CSF [3]. However, nerve conduction studies were unavailable, reflecting the use of clinical criteria in resource-constrained environments [6]. Diarrhea-induced hypokalemia was ruled out due to normal electrolyte levels.GBS Management: IV immunoglobulin (IVIG) or plasmapheresis is preferred treatment for GBS [4], but corticosteroids were utilized due to resource constraints. Research in low-resource settings reveals analogous results with corticosteroids when IVIG is unavailable [7].ARDS Management: Lung-protective ventilation and prone positioning were prioritized [2], but concomitant gastroenteritis made fluid management challenging [5].Coagulopathy: Rectal hemorrhage, caused by malaria-associated coagulopathy, was treated with tranexamic acid, although thrombosis hazards remain controversial [5].

Implications for Practice

- Diagnose of GBS in malaria patients presenting with ascending paralysis at early stage [3].
- Optimize the management of ARDS and GBS through Multidisciplinary team approach to [6].
- Advocate Stockpiling IVIG in malaria endemic regions to improve treatment availability [4].

Recommendations

1. Early GBS Diagnosis: Consider GBS in malaria patients with progressive weakness and areflexia; perform a lumbar puncture to assess albuminocytologic dissociation if nerve conduction studies are unavailable [3].
2. Multidisciplinary Care: Involve neurologists, pulmonologists, and intensivists early for synchronous ARDS and GBS management [6].
3. ARDS Management: Utilize lung-protective ventilation, prone positioning, and conservative fluid therapy [2].
4. Medication strategy: Treat malaria with artesunate, GBS with IVIG/plasmapheresis (corticosteroids if not available), and coagulopathy with cautious use of tranexamic acid [1,4,5].
5. Supportive Care: Provide nutritional therapy, withhold acute-phase iron, and manage neuropathic pain [1,4].
6. Resource Optimization: Improve access to diagnostic equipment, IVIG, and clinician training in resource limited settings.[6].

Conclusion

This case highlights the deadly synergy of falciparum malaria, ARDS, and GBS in a resource-constrained environment. Early clinical suspicion of GBS, protocolized management of ARDS, and multidisciplinary care are essential to improve patient outcomes. Clinicians working in malaria-endemic areas need to be aware of neurological complications and implement context-appropriate treatment strategies. More research into the pathophysiology of the Malaria-GBS association and the formulation of guidelines for the management of overlapping critical illnesses is needed to decrease mortality in such complicated cases.

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