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Case report

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A case of Guillain-Barré Syndrome following a bout of diarrhoea in advanced pregnancy

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Abstract

Guillain-Barre Syndrome is a rare acute inflammatory demyelinating polyradiculoneuropathy, affecting 1.2-1.9 per 100000 women in pregnancy. Whilst the condition is rare, delays in diagnosis and management are associated with poor maternal and perinatal outcomes. We present and discuss a case of 33-year old woman who presented at Harare Central Hospital Maternity Unit with acute flaccid paralysis in late pregnancy following a bout of diarrhoea. We discuss the diagnosis and management of pregnant women with the condition, as well as maternal outcomes.

Introduction

Guillain-Barre Syndrome (GBS) is a rare inflammatory demyelinating polyradiculoneuropathy that results in motor weakness [1-3]. The most common presentation of GBS is acute flaccid paralysis, which is ascending, beginning in the peripheries until it reaches the trunk [2-4]. Involvement of the diaphragm results in respiratory failure and ultimately death [4]. With the control of poliomyelitis, GBS is now the commonest cause of acute flaccid paralysis globally. The pathogenesis of GBS is multifactorial; it most commonly presents post bacterial or viral infections. *Campylobacter jejuni, Escherechia coli, Mycoplasma pneumonia*, Cytomegalovirus, the Human Immunodeficiency Virus (HIV) and other infectious organisms are implicated [1,2,5,6]. More recently, cases preceded by the Zika virus infection have been reported [7]. About 67% of patients with GBS report an antecedent infectious illness 2-4 weeks prior to the onset of symptoms [1,3]. Cases have also been reported post influenza vaccine administration [6,8,9]. Molecular mimicry between antigenic substances from pathogens and the nerve sheath is postulated to trigger an autoimmune response, with resultant demyelination and neurological manifestations [3,4,6,8,10]. Variants of GBS include acute inflammatory demyelinating polyneuropathy, acute motor axonal neuropathy, acute motor-sensory axonal neuropathy, the Miller-Fisher syndrome, acute panautonomic neuropathy, pure sensory GBS as well as other variants [1,3,10,11]. GBS is rare in the general population, and even more so in the pregnant population [5,12-14]. Estimated incidence in pregnancy is 1.2-1.9 per 100000 people annually, with high maternal risk [5,12]. Thus, attending clinicians need a high index of suspicion to avoid missing cases and institute appropriate therapy timeously. We report a case of GBS we managed at Harare Central Hospital Maternity Unit.

Patient and observation

History: a 33-year-old patient, in her second pregnancy, was referred from the local clinic for diarrhoea and generalised body weakness at 34 completed weeks of gestation. She had booked her pregnancy late at 29 weeks due to financial constraints, and had made only one antenatal care (ANC) contact. She was HIV negative and had no prior comorbidities. In her previous pregnancy, she had obstructed labour, and delays in delivery had resulted in development of a vesico-vaginal fistula (VVF) and neonatal death. The fistula was repaired successfully. She was in her second marriage. She stayed in a socially disadvantaged neighbourhood in Harare with poor water and sanitation facilities. When she presented to hospital she had two weeks´ history of profuse watery diarrhoea without fever or other associated symptoms. She also had generalised body weakness for the prior 2 days in the upper and lower limbs, as well as reduced sensation. She fell once at home, prompting the relatives to seek medical attention.

Physical examination: on examination, she was fully alert, with normal vital observations, and normal respiratory and cardiovascular examinations. The height of fundus on obstetric examination was 34 cm in keeping with gestational age, however the fetal heart could not be perceived by Pinard auscultation and there was no facility for emergency obstetric ultrasound scanning for fetal well-being. The neurologic examination was

remarkable, with reduced power, 1/5 in the upper and lower limbs, hypotonia, absent sensation to light touch and pinprick as well as absent deep tendon reflexes and ankle clonus. However, the neurologic findings did not extend to the abdomen, chest and the face. Working diagnoses of acute gastroenteritis and acute flaccid paralysis in pregnancy were made.

Investigations and management: the patient was considered for multi-disciplinary team management including the anaesthesiologists, neurologists and physiotherapists, and escalation of care to the high-dependency unit (HDU). The physicians/neurologists made a clinical diagnosis of Guillain-Barre Syndrome in pregnancy and attempted to do CSF studies but obtained a dry tap on lumbar puncture (LP). From laboratory investigations patient had normocytic anaemia, hypokalaemia and markedly elevated urea and creatinine, possibly from the profuse diarrhoea with resultant dehydration. Widal test for typhoid was negative; however, stool microscopy, culture and sensitivity results were later noted to be negative for bacterial diarrhoeal pathogens. The neuropathy worsened on day of transfer to the Intensive Care Unit (ICU) with respiratory involvement and patient was intubated for respiratory support. She went into spontaneous labour and had an emergency caesarean section. A grossly macerated foetus was delivered. A diagnosis of intrauterine foetal death had been made but due to very dense fibrosis of the vaginal tract and a history of obstetric fistula an attempt at vaginal delivery was abandoned. Day 1 post caesarean section patient was noted to have worsening kidney injury and thrombocytopenia. The nephrologists were consulted for consideration of haemodialysis. However, they felt the patient was not a suitable candidate at that time given the thrombocytopenia, respiratory compromise and the poor general outlook.

Outcome: the patient demised due to respiratory failure on the first day after the caesarean section.

Discussion

We presented a case of acute flaccid paralysis, clinically suspected to be GBS, which followed a bout of gastroenteritis in a pregnant patient. This represents a typical presentation, where an episode of an otherwise unremarkable infectious illness precedes the onset of neurological symptoms [1,3]. Some cases may present to unsuspecting clinicians, and who may ascribe some of the early symptoms to the pregnancy, with resultant delays in diagnosis and treatment [15]. Such delays have been associated with poorer outcomes. Sometimes patients present at an advanced stage of the disease owing to prohibitive socio-economic circumstances. The diagnosis of GBS is primarily clinical, based on history and examination findings [1,3,15,16]. The Brighton collaboration has developed a set of diagnostic criteria based on descriptive clinical, laboratory and electrodiagnostic parameters [15]. These criteria account for the level of diagnostic certainty based on the findings from level 1 (highest level of diagnostic certainty), to level 4 (reported as GBS, possibly due to insufficient data for further classification). Based on the presence of bilateral flaccid paralysis, decreased tendon reflexes, monophasic course of illness from onset to demise and absence of alternative diagnosis for weakness, the presented patient was level 3. Basic laboratory work-up (full blood count, urea and electrolytes, liver functions tests) and metabolic panel are generally normal in GBS and serve to exclude other aetiologies for the neuropathy. Electromyography (EMG) and nerve conduction studies (NCS) may assist in the diagnosis [1,5]. NCS studies may reveal abnormalities consistent with demyelination, evident in classic cases of GBS. These tests are locally available but at a different hospital, and given that the patient was critically ill at this point, it was deemed unnecessary to move her for the tests at this point. The absence of electrodiagnostic criteria must not preclude a patient from being managed as GBS, as according to the Brighton criteria, there is allowance for clinical diagnosis [15]. Pulmonary function tests aid to

assess respiratory function, as ascending GBS is frequently complicated by respiratory paralysis, and ultimately, failure, leading to patient demise.

Cerebrospinal fluid (CSF) studies are recommended for all cases of GBS. In the acute phase, findings include albuminocytologic dissociation, which is an elevation in CSF protein (>0.55g/L) without a corresponding increase in white blood cells [1,2,5,16]. This is thought to reflect the widespread inflammation of the nerve roots. Where a dry tap is obtained, repeat attempts can be made later to obtain CSF for analysis. However, in this case no later attempts at LP were made because the patient was very sick and the clinical diagnosis of GBS was convincing, and the CSF results would probably not have altered the course of management. Computed tomography and magnetic resonance imaging studies do not assist in diagnosis of GBS per se, but rather in excluding mechanical causes of myelopathy [1,2]. Due to time and resource limitations, we could not carry out these investigations in our patient. We could also not carry out serological assays for some of the implicated organisms. However, these generally do not add value to diagnosis and treatment and may be omitted in resource-limited settings. Patient management was complicated by institutional delays in transferring patient to HDU/ICU for ventilatory support; however, the patient was already seriously compromised with electrolyte abnormalities, metabolic derangements and acute kidney injury from dehydration secondary to gastroenteritis. The thrombocytopaenia noted on day of demise could signify disseminated intravascular coagulation in a patient who delivered a grossly macerated foetus. GBS in pregnancy, like in the non-pregnant patient, is often preceded by an antecedent bacterial or viral respiratory or gastrointestinal infection. The reported patient had a bout of diarrhoea overlapping with the onset of neurological symptoms. Most of the literature regarding GBS in pregnancy is in case reports, as the rarity makes larger observational studies difficult. Cases have been reported across all trimesters; however most cases tend to occur during the third trimester and in the immediate post-partum period [5,12,13,16]. GBS reportedly worsens in the post-partum period, and this is ascribed to an increase in type 4 hypersensitivity reactions [5,13]. The mortality of GBS in the non-pregnant population in good settings is estimated at <5%; however, this goes up to >7% in the pregnant population [12]. Up to 20% of pregnant patients who suffer from GBS will be disabled at 1-year post-delivery [5,12].

Pregnant patients with GBS are managed in the same way as non-pregnant patients. The mainstay of treatment is supportive, manifested as close monitoring of respiratory, cardiovascular and haemodynamic status as well as thrombophylaxis with low molecular weight heparin and thromboembolic deterrent stockings [5,12,13,16,17]. It is critical that pregnant GBS patients be managed in a centre with specialists and ICU facilities. A multi-disciplinary team approach involving anaesthesiologists, physicians, neurologists, obstetricians, physiotherapists and ICU nurses is the best. Plasmapheresis and intravenous immunoglobulin (IVIG), which are often not available in resource-limited settings, result in full recovery in up to 70-80% of the patients [5,12,13,17]. Inamdar *et al.* reported a case of a 23 year old who presented with GBS at 33 weeks of gestation and was managed with IVIG and respiratory support in an ICU setting [17]. The gravid uterus has a splinting effect on the diaphragm, and therefore may worsen respiratory compromise in the pregnant woman with GBS. Without ventilator support, outcomes are poor for patients with involvement of the diaphragm [16]. Respiratory involvement requiring ventilator support has been observed to increase the risk of spontaneous premature labour. The presented patient went into preterm labour, delivered a foetus in late stages of maceration, and eventually succumbed to respiratory failure, though this may have been complicated by the co-existing metabolic, renal and haematological derangements. Recovery periods are variable, ranging from a few weeks to many months. Hukuimwe *et al.* reported a case of 16 year old who developed GBS in the third trimester of

pregnancy [5]. Post-caesarean section she stayed in ICU for 66 days and HDU for 37 days, and even then she was discharged to the referring institution with residual weakness, and demised on the second day post back-referrral. Relapses have been reported in 5.5-6.8% of patients [5,13]. Meenkashi-Sundaram *et al.* reported a case of a 30 year who had an initial episode of GBS at 34 weeks of gestation, which was appropriately managed [13]. Patient experienced a relapse in the post-partum period, which was also appropriately managed, and she recovered completely. However, some patients may remain with permanent residual disability [1,3,13].

***Ethics**:* written informed consent to publish this case was obtained from the patient´s partner. We sought consent from the partner because the decision to publish the case was made when the patient had already demised. The Medical Research Council of Zimbabwe and the Joint Research Ethics Committee, which are the local Ethical Committees, do not require written informed consent for publication of any case-report, provided no patient identifying details are included in the write-up; however, per SAGE requirements we went ahead to obtain it.

Conclusion

We have presented and discussed a case of GBS in late pregnancy. In resource-limited settings, a rapid clinical diagnosis is critical, as there are often limitations in laboratory and neurological investigations to aid diagnosis. A multi-disciplinary team approach in a tertiary facility with ICU facilities confers the best outcomes.

Competing interests

The authors declare no competing interests.

Authors' contributions

Grant Murewanhema clerked, admitted and managed the patient, did the caesarean section and prepared the manuscript. Bismarck Mateveke and Munyaradzi Nyakanda are consultants who were actively involved in patient management and manuscript preparation. Francis M Gidiri provided technical expertise in case writing. All the authors have read and agreed to the final manuscript.

References

1. Burns TM. Guillain-Barré syndrome. Semin Neurol. 2008;28(2): 152-67. **Google Scholar**
2. Andary MT. Guillain-Barre Syndrome clinical presentation. Emedicine. 2018;1-6.
3. Wilison HJ, Jacobs BC, van Doorn PA. Guillain-Barré syndrome. The Lancet. 2016;388(10045): 717-727. **Google Scholar**
4. Winer JB. An Update in Guillain-Barré Syndrome. Autoimmune Dis. 2014;2014: 793024. **PubMed**| **Google Scholar**
5. Hukuimwe M, Matsa TT, Gidiri MF. Guillain-Barré syndrome in pregnancy: a case report. Women´s Heal. 2017;13(1): 10-3. **PubMed**| **Google Scholar**
6. Sejvar JJ, Baughman AL, Wise M, Morgan OW. Population incidence of Guillain-Barré syndrome: a systematic review and meta-analysis. Neuroepidemiology. 2011;36(2): 123-33. **PubMed**| **Google Scholar**
7. Leonhard SE, Lant S, Jacobs BC, Wilder-Smith A, Ferreira MLB, Solomon T *et al*. Zika virus infection in the returning traveller: what every neurologist should know. Pract Neurol. 2018;18(4): 271-7. **PubMed**| **Google Scholar**
8. Park YS, Lee KJ, Kim SW, Kim KM, Suh BC. Clinical features of post-vaccination Guillain-Barré syndrome (GBS) in Korea. J Korean Med Sci. 2017;32(7): 1154-9. **PubMed**| **Google Scholar**
9. Campanharo FF, Santana EF, Sarmento SG, Mattar R, Sun SY, Moron AF. Guillain-Barré Syndrome after H1N1 Shot in pregnancy: maternal and fetal care in the third trimester-case report. Case Rep Obstet Gynecol. 2012;2012: 1-2. **PubMed**

10. McGrogan A, Madle GC, Seaman HE, de Vries CS. The Epidemiology of Guillain-Barré Syndrome Worldwide. Neuroepidemiology. 2008;32(2): 150-63. **PubMed**| **Google Scholar**
11. Levin KH. Variants and mimics of Guillain Barré Syndrome. Neurologist. 2004;10(2): 61-74. **PubMed**| **Google Scholar**
12. Zafar MS, Naqash MM, Bhat TA, Malik GM. Guillain-Barré syndrome in pregnancy: an unusual case. J Fam Med Prim Care. 2013;2(1): 90. **PubMed**| **Google Scholar**
13. Meenakshi-Sundaram S, Swaminathan K, Karthik S, et al. Relapsing Guillain-Barre syndrome in pregnancy and postpartum. Ann Indian Acad Neurol. 2014;17(3): 352. **PubMed**| **Google Scholar**
14. Gupta R, Chhabra B. Critical care of a pregnant patient with Guillain-Barre Syndrome. Indian J Anaesth. 2003;47(1): 50-2.
15. Fernando TN, Ambanwala AM, Ranaweera P, Kaluarachchi A. Guillain-Barré syndrome in pregnancy: a conservatively managed case. J Fam Med Prim Care. 2017;5(3): 688. **PubMed**| **Google Scholar**
16. Zilberlicht A, Yonai NB. Gullian-Barre Syndrome in pregnancy: a case report and review of the literature. Gynecol Obstet. 2016;06(01): 1-4. **Google Scholar**
17. Inamdar S, Inamdar A, Chaudhary R, et al. Successful maternal and fetal outcome of Guillain-Barre syndrome complicating pregnancy: a case report. Int J Reprod Contraception, Obstet Gynecol. 2013;2(3): 478-9.