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Case Report

Case report
Guillain-Barré syndrome in pregnancy

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Abstract

Background : Guillain-Barré Syndrome (GBS) is an acute demyelinating polyadiculopathy that usually present as progressive and symmetrical muscle weakness accompanied by absence or loss of deep tendon reflexes. This has been associated with various infectious agents, such as Campylobacter jejuni and usually occurs after 2–4 weeks after respiratory or gastrointestinal diseases. Estimated general incidence in population was 0.75–2: 100,000. Pregnancy can increase risk of GBS. The diagnostic criteria of GBS consist of clinical, laboratory and electrophysiological tests. Developing treatments such as plasmapheresis and intravenous immunoglobulin (IVIG) are relatively safe in pregnancy. Time and methode of delivery are based on obstetric indications and depend on maternal and fetal status.
Purpose : The condition is rare in pregnancy and only few cases have been reported in literature. Appropiate management of pregnant patients with GBS is needed.
Case report : We presented the case of a 20-year-old woman, with a 20–week pregnancy. She had experienced various complications from her GBS such as syndrome of inapropriate antidiuretic hormone secretion (SIADH), dysphagia, type 2 respiratory failure, and infectious during hospital treatment. The termination of pregnancy was carried out at 34 weeks with consideration of maternal and fetal conditions. She delivered a healthy baby.
Conclusion : GBS in pregnancy must be handled by a multidisciplinary team involving neurologists, obstetricians, internist, and anesthetists.

Keywords : Gullain Barre syndrome, pregnancy, intensive care management, SIADH, Intraveva Immunoglobulin , plasmapharesis

INTRODUCTION

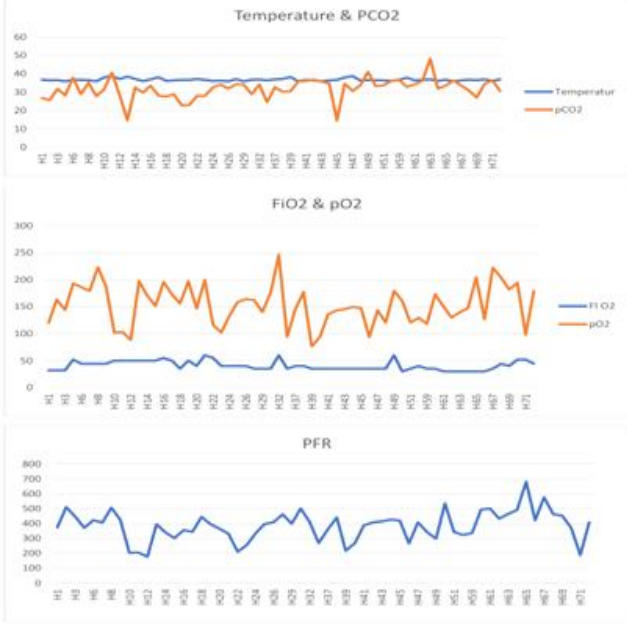
Guillain-Barre syndrome (GBS) also known as Acute Inflammatory Demyelinating Polyneuropathy (AIDP) is characterized by progressive weakness, increases paralysis and areflexia with or without abnormal sensory function. Common symptoms that occur are flaccid weakness at any age.[2,4] Although this case is rare, it increases the need for pregnant women with GBS to support ventilation and a higher mortality rate (10%).[1] About one third of patients with GBS will require mechanical ventilation and most GBS–related deaths occur as a result of respiratory failure. In developing countries, GBS has been shown to be an important cause of flaccid paralysis. Population-based surveys documented annual incidence of GBS had been carried out in various countries around the world and generally occur at rates of 1–3 per 100,000 individuals per year. In a cohort study, age–adjusted relative risk showed the risk for GBS was lower during pregnancy and increased after delivery. The condition usually relapse during consecutive pregnancy.[4]

Occurrence of GBS in the third trimester of pregnancy carries the risk of respiratory complications and prematurity. There is no specific therapy for GBS; Corticosteroids when used alone show minimal therapeutic effect. Therapeutic Plasma Excange was the first therapeutic modality. An international RCT compared therapy with TPE, IVIG or TPE followed by IVIG in 383 adult patients with severe GBS resulting in all three is equivalent. There were no significant differences for all the treatment groups in disability improvement nor the time recovery (TPE group 49 days, IVIG group 51 days, and TPE/IVIG group 40 days). In primary treatment TPE procedure is recommended Grade 1A, category I. After IVIG, TPE is recomended for grade 2C category III.[11] There is no increased rate in spontaneous abortion related with TPE and IVIG treatment. The increased incidence of respiratory complications is mostly caused by the gravid uterus. Mechanical ventilation due to GBS: 33% in pregnancy vs 16% in non pregnancy. Mortality due to GB syndrome multiplies when suffering in the third trimester.[5]

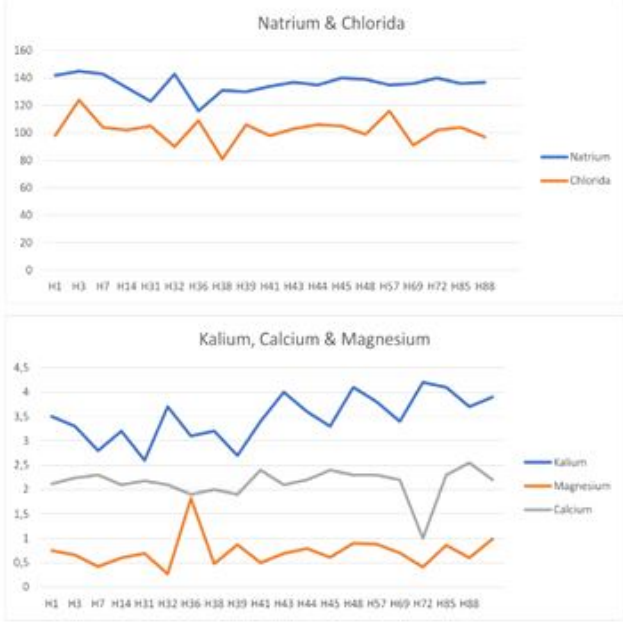
CASE REPORT

A 19-year-old woman in her first pregnancy, at 20 weeks gestation reported to the emergency ward becauseof numbness and weakness in all of her limbs for past 7 days and difficulty in breathing for last 2 hours. She was in acuterespiratory distress and a neurological examination revealed decreased power in all limbs, disphagia, disfonia, N IX–X paralysis with decrease swalowing, absent deep tendon reflexes, and sensory loss in her limb. The size of her uterus history corresponded and ultrasound showed a 20 week single active fetus with no anomalies.

From blood examination we found mild aenemia, increased coagulation study quantitative D-dimer 1540, fibrinogen titer 455.8, Hb: 9.4; Ht: 27.6; Erytrocyt 3.17; Leucosyt 11.2; Trombocyte 294; blood sugar 112; acid lactat 1.8; SGOT 54; SGPT46; Ureum 13; creatinin 0.6; Mg:

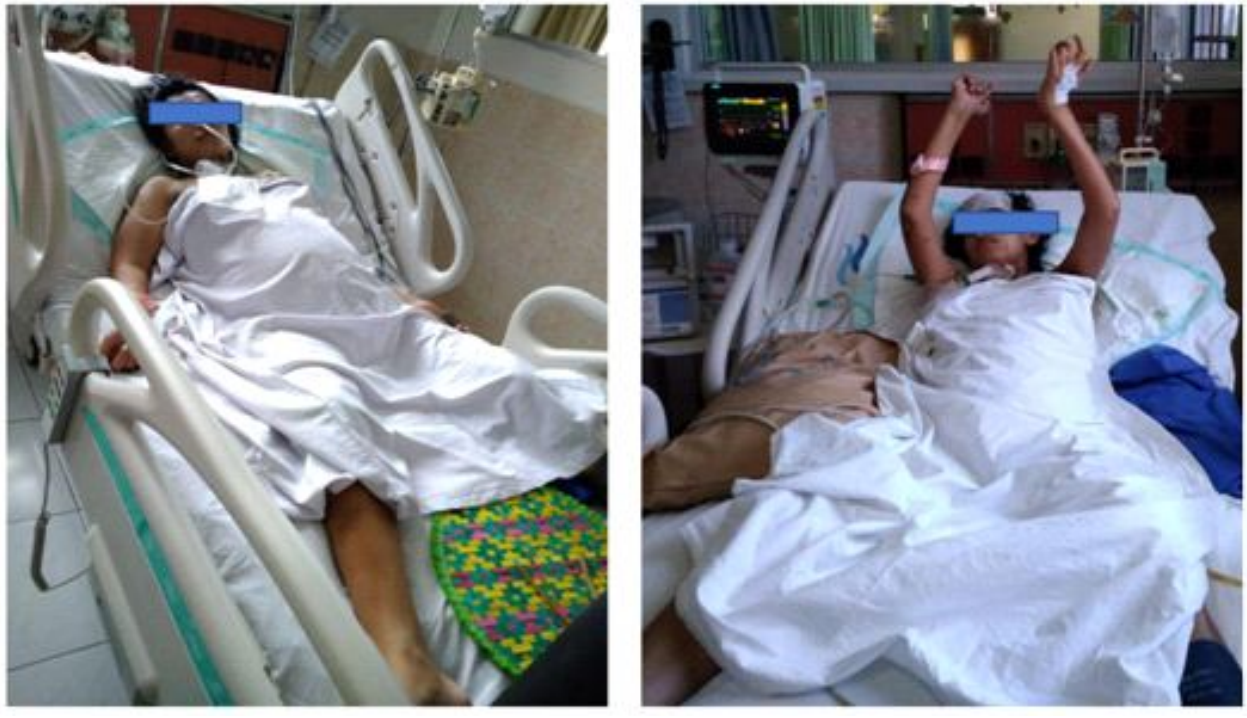


(a)



(b)

Figure 1. Serial Result : (a) Blood Gas Analysis (b) electrolyt



(a) (b)

Figure 2. (a) 30 week pregnancy **(b)** 2 days after delivery

0.75; Ca 2.34; Na: 142; K 3.5; Cl: 98 albumin 3.2). Blood gas analysys in normal value. From EMG we found bilateral severe degree of polycadiculopaty, with severe bilateral sensorimotor polyneuropathy that supports the SGB.

She was moved to Intensive Care Unit (ICU) with a probable diagnosis of GBS, with acut physiology and chronic health evaluation (APACHE) score 5, predicted death rate 5.8% and PFR 374. Investigations included blood biochemistry, X–Ray chest, ECG with oxygen in nassal canul 3 lpm. In CSF examination we found protein, glucose, pmn, mn, ecytrocyt cell in normal value. There was not albuminocytologic dissociation because the onset was below 7 days.

She received therapheutic plasma exchange 5 times frecuently on day 2nd, 4th, 7th, 9th and 13th. On 9th day her condition deteriorated as her tidal and minute volume had decreased, she was ventilated on controlled mandatory ventilation (CMV) mode. She got fever with temperatur 38.0°C, leucocyte 21.500, she underwent thorax X–ray with the result of pneumonia and right pleural effusion. HIV screening was non reactif. From urine culturegrowth they found that growing bacteria was eschericia coli (100.000CFU/ml) and. From the sputum cultur, the bacteria that we found was pseudomonas aeruginosa. She was diagnosed urinary track infection and pneumonia.It decided to be treated with amikacin for 7 days.

On day 16th she got very thirsty, without fever, there was pitting edema in lower limb, ANA test was result 2.1 (negatif). The other finding hypoalbumin (1.7), hyponatremia (133), with polyuria (dyuresis 2.7 cc/kgBB/h). Electrolyt within urine had been checked and the result were: natrium 205 (N), kalium 14.2 (low), chloride urine 176 (N). We corected hypoalbumin an hyponatremia. She was diagnosed SIADH.

On Day17th, she got fever again, from result of bronchial swab culture found Escherichia coli in bronchus. She was diagnosed with Extended spectrum beta–lactamases (ESBL), so She transfered to singgle room to prevent droplet infectionand treated with tygecyclin 100 mg in 1stday and 50 mg in 2nd–7th day, the sensitive antibiotic of the culture result.

One day later she got diarhea, from feces examination we found protein, fat and glucose malabsorption. She was treated with parenteral nutrition, aminofusin, smoflipid and enteral nutrition: proten.

On 22th day, she got IvIg with therapeutic dose 0.4 gr/kgBB/24 hours for 5 days because she was not being better with TPE.

On 35th day, she got fever, temperature 38.5°C, with hyponatremia (116), polyuria. Hypokalemia 3.2, the results of procalcitonin 9.3 and increased into 18.76 on 37th, the result from culture examination we found Acinetobacter baumannii in urine, staphylococcus haemolyticus from blood and pseudomonas aeruginosa from sputum. She got vancomycin 1 gr/12 h for 7 days afterwards due to sensitivity result. Electrolyte imbalance had been corrected.

On 44th, fever (-), from culture reexamination there was no bacteria growing, with pro calcitonin 3.96.

On 56th day the patient complained of itching between the fingers, especially at night it. From dermatologist examination she was diagnosed with scabies, she receive permethrin for 8 hour for three times procedure, continued with 2 time a day.

On 62th day she got fever again, and hematuria, from the result of routine urine examination we found bacteriurua dan from urine culture results, obtained Acinetobacter baumannii. She was treated with meropenem 1 gram/8 hour due to sensitivity test of culture for 10 days.

On 72th day, she was extubated and transfered to HCU for delivery program at 34th week of pregnancy.

On 86th day, she trough *sectio caesaria* program to deliver of her baby with general aenestecy. The birth weight of her baby is 1.600 gram and immedietly being transfered to unit for underweight newborn ward.

On 88th the TORCH results was negative IgM, on toxoplasam IgG 43 (+), rubella 216 (+) IgG, HbSAg (-), anti DS DNA 2.1 (-), procalcitonin 0.20, anti HAV IgM 0, 0 (-). She was tranfered for regular ward and could go home 5 days later.

DISCUSSION

TPE was the first therapeutic modality. An international RCT compared TPE, IVIG, and TPE followed by IVIG in 383 adult patients with severe AIDP and found all three modalities to be equivalent. There were no differences in the three treatment groups in mean disability improvement at 4 weeks nor the time to be able to walk without assistance (TPE group 49 days, IVIG group 51 days, and TPE/IVIG group 40 days).[11]

Respiratory failure requiring mechanical ventilation is a common complication of Guillain–Barré syndrome. Its etiology is clearly multifactorial but seems primarily due to diaphragmatic weakness. Early prediction of decline in respiratory function and progression to mechanical ventilation can be obtained by a combination of clinical variables, including neck muscle weakness, single breath count, and bulbar weakness. Electrophysiological studies at the early stage of Guillain-Barré syndrome have documented prolonged phrenic nerve latency. In addition, diaphragmatic compound muscle action potential latencies, amplitude, and duration are significantly different between Guillain–Barré syndrome patients with and without respiratory failure. Although phrenic nerve conduction time improves over time (often several weeks), this electrophysiological recording cannot be used to predict weaning from mechanical ventilation, as full clinical recovery usually precedes electrophysiological recovery. It has to be emphasized that phrenic nerve electrophysiology has several limitations. It may not be feasible technically in each patient and requires an experienced electrophysiologist. Another drawback is that phrenic nerve electrophysiology explores only diaphragmatic weakness, but not the activity of intercostal muscles that are also affected in patients with Guillain–Barré syndrome who require mechanical ventilation.[15]

SIADH may develop during or after maximum motor deficit in 30% of GBS patients and generally 65% of dysautonomy cases. Our patient had tetraparesis flaccid and an absent deep tendon reflexes with initial symptoms of SIADH and dysautonomy. Visceral afferent fibers may be affected together with autonomic dysfunction and parasympathetic and sympathetic fibers, leading to sympathetic and parasympathetic insufficiency and hyperactivity linked to neuropathy. These factors combined with vascular tension receptors affecting peripheral autonomic fibers causing abnormal ADH secretion from the neurohypophysis reduce the effects of vagal inhibition. However, without dysautonomy, the relationship between GBS and SIADH has not been clearly explained.The pathogenesis of GBS-related SIADH is uncertain. Among the hypotheses, pathogenesis may be linked to changed osmoreceptor responses due to new lower threshold values in the osmoregulatory system, vasopressinincreased tubular sensitivity or ADH secretion affecting cardiac volume and afferent peripheral autonomic neuropathy of osmolarity receptors. Recently, publications have proposed that a multifunctional cytokine, interleukin 6 (IL-6), may play a central role in the immunopathogenesis of SIADH linked to GBS.[7] Osmoreceptor dysregulation may contribute to polyuria, SIADH, and renal salt wasting related to AD leading to hyponatremia.[8]

Extended–spectrum gram–negative bacteria are emerging pathogens. Extended spectrum beta–lactamases (ESBLs) are the enzymes that have the ability to hydrolyze and cause resistance to various types of newer β–lactam antibiotics, including the expandedspectrum (or third generation) cephalosporins (eg. cefotaxime, ceftriaxone, ceftazidime) and monobactams (eg. aztreonam), but not the cephamycins (eg. Cefoxitin and carbapenems (eg. imipenem, meropenem and etrapenem). These enzymes are sensitive to β–lactamase inhibitors (sulbactam, clavulanic acid, and tazobactam). Major risk factors for colonization or infection with ESBL producing organisms are long term antibiotic exposure, prolonged intensive care unit (ICU) stay, nursing home residency, severe illness, residence in an institution with high rates of ceftazidime and other third generation cephalosporin use and instrumentation or catheterisation. E. coli, that can produce ESBLs, has arisen and disseminated worldwide as an important cause of both nosocomial and community infections and nowadays represents a major

threat.[10]

Malabsorbsi in tractus gastrointetinal might be result from disbyosis that could lead by antibiotic used. The effects of antibiotics on the gut microbiome are unique to each antibiotic and depend on several factors: the target spectra, the dose and duration, the methode of administration and the pharmacokinetic and pharmacodynamic properties of theagent (incomplete absorption or secretion of intravenous antibiotics by bile or intestinal mucosa may result in higher concentrations in the intestine with greater ecological effects. Disturbances may be assessed (1) qualitatively (emergence of novel bacteria types with resistance genes, plasmids, and transposons) or (2) quantitatively (changes inmicrobiota composition due to antibiotic pressures. Broadspectrum antibiotic treatments affect not only the aberrant pathogenic bacteria but also benefcial members of the gut community, reduce diversity and richness, anddisturb the ecology of the gut. However, it remains a challenge to distinguish antibiotic effects on the gut microbiota from other confounding factors such as stress, diet andhost genetics. The route of administration has implications forantibiotic–mediated disturbances in the gut microbiota because oral, inhaled and intravenous routes result in different concentrations in different sites.[14]

In patients with suspected ventilator associated pneumonia (VAP), It is recommended including coverage for Staphylococcus aureus, Pseudomonas aeruginosa, and other gram–negative bacilli in all empiric regimens. If empiric coverage for *Methicillin–resistant Staphylococcus aureus* (MRSA) is indicated, it is recommended either vancomycin or linezolid. MRSA, *Hospital aquired pneumonia* (HAP)/VAP are recomended be treated with either vancomycin or linezolid rather than other antibiotics or antibiotic combinations. For patients with HAP/VAP due to P. aeruginosa, it is recommend that the choice of an antibiotic for definitive (not empiric) therapy be based upon the results of antimicrobial susceptibility testing. For patients with HAP/VAP due to P.aeruginosa, were commend against aminoglycoside monotherapy (strong recommendation, very low-quality evidence). Routine antimicrobial susceptibility testing should include assessment of the sensitivity of the P. Aeruginosa isolatetopolymyxins (colistinorpolymyxinB) in settings that have a high prevalence of extensively resistant organisms.[12] Procalcitonin is a recently re–discovered biomarker that fulfils many of these requirements especially in comparison to conventional and widely used other biomarkers that have demonstrated superior diagnostic accuracy for a variety of infections, including sepsis.[16]

Scabies is a common parasitic skin disease, caused by the mite Sarcoptes scabiei. It is characterized by intensely pruritic, often widespread, popular, and/or eczematous lesions, but a typical presentations can occur. Complications include secondary bacterial infection and crusted (Norwegian) scabies. Although the prevalence of scabies in pregnancy has not been systematically investigated, observation studies indicate that it accounts for 2–6% of all skin diseases in pregnancy. Its clinical presentation does not differ from that in non–pregnant women. Crusted scabies seems to be rare in pregnancy. Scabies is not associated with adverse pregnancy outcomes; however, it is very important to differentiate scabies from other pruritic skin diseases during pregnancy, in particular specific dermatoses of pregnancy, so that scrapings for the mite, eggs, or fecal pellets and dermatoscopy that shows the jetliner–with–contrail sign at the site of infestation are recommended in suspected cases. The history of intense pruritus in close contact(s) is an important clue. Permethrin 5% cream (Category B) is the treatment of choice in pregnancy. Permethrin is a pyrethroid with </= 2% systemic absorption rate. No evidence of fetal harm or mutagenicity was observed in animal studies with oral permethrin at exposed to permethrin (31 during the 1st trimester), no differences in live births, spontaneous abortions, major malformations, birth weight, and gestational age at delivery were observed when compared to matched controls. This was supported in a study of 196 pregnant women exposed to permethri doses of 200–400 mg/kg/day. Although it may cross the placenta, the risk for fetal exposure is minimal because permethrin is absorbed in only small amounts and is rapidly metabolized.[13]

CONCLUSION

Although GBS is relatively rare medical condition to encounter inpregnancy, the challenge of it requiring a multidisciplinary approach. A prompt evaluation should be undertaken in patients presenting with the typical clinical features. Treatment includes of supportive care with respiratory support and cardiac monitoring that should be followed, when needed, by either plasmapheresis or IVIG, both relatively safe inpregnancy. There are complication in GBS, that pure from GBS such us dysautonomy, SIADH, respiration failure and its from infection and malnutrition that must be treated in a pregnant women with imunocompromise condition.

GBS is not an indication for caesarian delivery. Procedure of delivery should be decided individually, concidering some of obstetric indications. Anesthesia should be performed according to maternal status and the result of anesthesiologist consult.

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