Guillain-Barre' Syndrome: A Case Report



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Abstract

Background and Objectives: Guillain-Barre´ Syndrome (GBS) is an acute-onset, monophasic, immune-mediated polyneuropathy that often follows an antecedent infection. Special nutritional support has an important role in these patients.

Methods: A 6 years old girl with GBS was admitted to NICU of Dr. Sheikh Children Hospital (Tehran, Iran). Chief compliance for hospitalization was inability to walk and having a progressive muscle involvement of the lower extremities upwards. The patient was affected to this condition during just one day before hospitalization. After the patient was stabilized, she was referred to nutritional support team for specialized Guillain-Barre medical nutritional therapy. The patient was discharged after 37 days and had improved medical condition.

Findings: From the 157 obtained samples, 50 (30.9%) were positive cultures. A significant difference in the frequency of positive cultures was identified between four surgical scrub solutions (df = 3, χ 2 = 17.4, P = 0.001). The lowest frequency of positive culture (7.5%) was observed for the solution containing povidone-iodine (10% Betadine Scrub).

Conclusions: GBS is rare; however, it must be considered in the differential diagnosis of any patient, who presents with progressive weakness. It is important to recognize the variety and severity of the neurologic symptoms associated with GBS, especially with the diagnostic difficulties associated with the pediatric population.

Keywords: Diet, Guillain-Barre' Syndrome (GBS), Nutrition support

Background

Guillain-Barre' Syndrome (GBS) is an acute-onset, monophasic, immune-mediated polyneuropathy that often follows an antecedent infection [1]. Controlled epidemiological studies have linked it to infection with *Campylobacter jejuni* in addition to other viruses, including cytomegalovirus and Epstein Barr virus.

The syndrome includes several pathological subtypes, the most common being a multifocal demyelinating disorder of the peripheral nerves in close association with macrophages [2]. The annual incidence of GBS is around 1-3/100 000 population according to the epidemiological studies from the Europe, USA, and Australia. It can occur in any age group. The age specific curve seems to show a bimodal distribution, with peaks in young adults and the elderly [3, 4]. The diagnosis relies heavily on the clinical impression obtained from the history and examination, although cerebro-spinal fluid analysis and electro-diagnostic testing usually provide evidence supportive of the diagnosis. The clinician must also be familiar with mimics and variants to promptly and efficiently reach an accurate diagnosis. Intravenous immunoglobulin and plasma exchange are efficacious treatments. Supportive care during and following hospitalization is also crucial [1].



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Days of hospitalization	Tests and Result
Day 2	ESR=115, CRP=1+
	Urine Analysis: Sugar: negative, Protein: negative, Bilirubin: negative, Urobilinogen:
	negative, Blood: negative, Acetone: positive
Day 3	ESR = 90, CRP = 2+
Day 5	Phosphorus: 3.5, Calcium total: 9.2, Cholesterol: 174, Triglyceride: 169, Sodium: 139,
	Urea: 38, Creatinine: 0.4, T. Billirubin:1.6, D. Billirubin:0.4, Magnesium: 2.1,
	CSF tests: Sugar: 71, Protein: 39, Appearance: clear, WBC: 0, RBC: 0, Direct Gram stain:
	negative
Day 20	T. Billirubin: 0.5, D. Bilirubin: 0.2, CPK total: 19, LDH total: 464, Urea: 36
Day 30	Urea:19, Creatinine: 0.5, Calcium total: 10, Phosphorus:5, Sodium: 139, Potassium: 4.6,
	Chloride: 86, Magnesium: 2.2

Table 1 Laboratory Tests

Case presentation

Background information

A 6-years old girl with GBS was admitted to the NICU of Dr. Sheikh Children Hospital (Tehran, Iran). Chief complaints of the patient for hospitalization were disability and inability to walk, having a progressive muscle involvement of the lower extremities upwards, and speech disorder while she had the ability to swallow. Moreover, DTR was negative and Banski was flat, but GAG reflex was positive. The patient was affected to this condition during just one day before hospitalization. She had diarrhea two weeks before hospitalization that was relieved with no medicine. The patient was the first child of non-consanguineous marriage and had no history of hospitalization. Her birth weight was 3,600 grams, and was 20 Kg on hospital admission. She was intubated at the beginning of hospitalization, and tracheotomy was done. Furthermore, she was placed under EMC NCV due to weakness and paralysis of lower extremities. NCV was reduced and nerve conduction was positive. Also IVIg was received during hospitalization, and Plasmapheresis was done for one time. Tracheostomy/Tracheotomy was done at the day 10 of hospitalization.

Laboratory tests

Table 1 presents laboratory test the patient underwent.

Food/nutrition-related history

TROPLC nutrition was started at the first day of hospitalization. At the day 3 of hospitalization, the patient tolerated liquid every four hours via gavage, as well as 200 cc pediatrics entrameal every 5 hours, gain up advance 200cc/TID , multivitamine 5cc po/QD, Amp Vit B,C, Tab Folic acid , Mg po/BID, Feru sulfate 2.5 cc po/BD, syr zinc sulfate 2.5 cc po/BD, and liquid 300cc/q4h.

Parenteral nutrition was started one week after hospitalization (AA 10% and IL 10% 250cc/IVQD).

The patient was discharged after 37 days. Fluid and soft food were tolerated by oral (600cc/2h) and the tracheostomy was fixed. Moreover, the force of the upper extremities was 2-3 of 5 and the lower extremities 1 of 5.

The goals of nutrition support for the patient with GBS are the same as any other patient in the critical care unit (CCU). They include maintenance of fluid and electrolyte balance, immune competence, and skin integrity. The goals can be applied for prevention of superimposed starvation-induced wasting of the muscles from the effects of metabolic stress [5]. To achieve these goals, feeding with a sufficient amount of nutrients and optimal distribution of non-protein calories are required. This method seems to be the most important during the ventilator weaning, when nutritional repletion is necessary to build up the musculature, and over-feeding could lead to an increased ventilatory load. Minerals and free water must be provided according to the patient's needs, and vitamins should be added in sufficient amounts. Failure to achieve optimal nutritional support will lead to the unwanted effects of any hyper-metabolic and hypercatabolic state [6], including infectious complications and failure to wean from mechanical ventilation. The total daily calorie requirements are usually estimated on a body weight basis or the Harris-Benedict formula, which calculates the basic energy expenditure from the body weight, height, and age [5]. In patients with intensive support, this formula is modified to account for their non-basal status. Despite such a regimen, some patients appear to be hyper-metabolic as evidenced by weight loss in the setting of euvolemia and normal calorie provision. These patients also appear to be hypercatabolic as evidenced by indirect calorimetry, increased urinary nitrogen excretion, and negative nitrogen balance (despite supra-physiologic replacement) [6]. The reasons for these findings remain unclear, but a possible explanation could be stress-induced catabolism with associated increased levels of circulating catacholamines, cytokines, or corticosteroids [7, 8].

Conclusions

GBS is rare; however, it must be considered in the differential diagnosis of any patient, who presents with progressive weakness. It is important to recognize the variety and severity of the neurologic symptoms associated with GBS, especially with the diagnostic difficulties associated with the pediatric population.

Abbreviations

(GBS): Guillain-Barre' Syndrome; (CCU): Critical Care Unit

Competing Interests

The authors declare no competing interests.

Authors' Contributions

The authors had the same contributions in the study.

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References

- Burns TM, editor. Guillain-Barré syndrome. Seminars in neurology. New York. Thieme-Stratton Inc; 2008.
- Winer JB. Guillain Barre syndrome. *Mol Pathol* 2001, 54(6):381-85.
- Govoni V, Granieri E, Casetta I, Tola MR, Paolino E, Fainardi E, *et al.* The incidence of Guillain-Barré syndrome in Ferrara, Italy: Is the disease really increasing? *Journal of the neurological sciences* 1996,137(1):62-8.
- Jiang G, Cheng Q, Link H, de Pedro-Cuesta J. Epidemiological features of Guillain-Barré syndrome in Sweden, 1978-93. *J Neurol Neurosurg Psychiatry* 1997, 62(5):447-53.
- Rogera M, Hanley D.Optimizing nutrition for acute neurologic illness. In Critical Care Report; 1989.
- 6. Roubenoff RA, Borel CO, Hanley DF. Hypermetabolism and hypercatabolism in Guillain-Barré syndrome. *Jpen-Parenter Enter* 1992, 16(5):464-72.
- 7. Durocher A, Servais B, Caridroix M, Chopin C, Wattel F. Au-

tonomic dysfunction in the Guillain-Barre syndrome. Hemodynamic and Neurobiochemical studies. *J Intensive Care Med* 1980, 6(1):3-6.

 Davies AG, Dingle HR. Observations on cardiovascular and neuroendocrine disturbance in the Guillain-Barre syndrome. *J Neurol Nerosurg Psychiatry* 1972, 35:176-179.

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