GUILLAIN-BARRE SYNDROME IN ASSOCIATION WITH HEPATITIS A INFECTION AND NEPHRITIC SYNDROME

Imran Khan[1], Aliena Badshah[2]

ABSTRACT

A 17-year-old male developed Guillain-Barre Syndrome (GBS) in association with acute hepatitis A infection followed by nephritic syndrome. Some cases of GBS were reported in association with acute hepatitis or nephrotic syndrome. GBS in association with nephritic syndrome alone or simultaneous nephritic syndrome and hepatitis A infection has never been reported.

Key Words: *Acute hepatitis A, Guillain-Barre Syndrome, Nephritic syndrome*

This case report may be cited as: Khan I, Badshah A. Guillain-Barre Syndrome in association with Hepatitis A infection and Nephritic Syndrome. J Postgrad Med Inst 2012; 26(2): 226-8.

INTRODUCTION

Acute viral hepatitis A is uncommonly associated with various neurological manifestations like encephalitis, meningitis, myelitis, radiculitis, Guillain Barre Syndrome and mononeuritis multiplex. Such neurological involvement may occur as a result of direct cytotoxicity of the virus or immunological reaction to viral particles.

Although hepatitis B infection usually causes a more severe illness than hepatitis A and may cause chronic liver disease, hepatitis A is more common[1] and may occasionally cause other serious complications as well as fulminant hepatic failure. Gullain Barre syndrome has been observed in patients with membranous glomerulonephritis, focal segmental glomerulonephritis, extra membranous glomerulonephropathy or nephrotic syndrome. Few cases of GBS in association with acute hepatitis have been reported[2-4]. To the best of our knowledge it has never been reported with nephritic syndrome alone or in association with concurrent nephritic syndrome and acute hepatitis. We report Guillain-Barre syndrome with serologically confirmed hepatitis A followed by nephritic syndrome.

[1-2]Medical Unit D Khyber Teaching Hospital Peshawar - Pakistan

Address for Correspondence:
Dr. Imran Khan
Medical Unit D,
Khyber Teaching Hospital Peshawar - Pakistan
E-mail: khan114@hotmail.com

Date Received: October 27, 2011
Date Revised: December 25, 2011
Date Accepted: January 10, 2012

CASE REPORT

A 17 year old male patient presented to the medical unit with the chief complaint of jaundice for 7 days accompanied by prodrome symptoms. The symptoms improved over two days when he had rapid onset of bilateral progressive lower limb weakness over 3 days. He denied history of fever, itching, abdominal pain, headache, blurred vision, giddiness or altered sensorium. He had not received any hepatotoxic or neurotoxic drugs or vaccination in the recent past.

Examination revealed mild soft nontender hepatomegaly; there were no signs of chronic liver disease. Power in the upper limbs was 4/5 but a power of 3/5 in both lower limbs. Reflexes were normal in the upper limbs but absent in lower limbs. Perception of position, touch, pinprick, and temperature was normal. The patient had a weak cough reflex.

His peripheral smear, Urine R/E, serum electrolytes and rest of the baseline investigations were normal. The serum liver function tests were abnormal. SGPT (ALT) = 111U/liter. Alkaline phosphatase 198 IU/I (normal 30-110 lU/l); serum bilirubin concentration was 4.4mg/dl, Anti HCV by ELISA, Hepatitis B surface antigen (HBsAg) and antibody (HBsAb), hepatitis B core antigen (HBcAg), and IgM antibody were all absent; serological test results for recent Epstein-Barr virus and cytomegalovirus infections were negative. Hepatitis A IgM antibody measured by radioimmunoassay was detectable. A lumbar puncture was planned. Report of the CSF R/E revealed proteins = 86mg%, glucose = 67mg% and a total cell count = 3/cumm with no neutrophils and a lymphocyte count = 100%. No AFBs or other micro-organisms were seen. Subsequently, the patient underwent nerve conduction studies which showed electrophysiologic evidence of

acute denervation in the upper and lower extremity muscles. Sensory responses were reported as normal. The motor responses showed reduction in amplitude in legs but no evidence of demyelination such as conduction block or temporal dispersion.This pattern of findings in the context of this patient's illness was consistent with acute motor axonal neuropathy (AMAN). A diagnosis of GBS was made and the patient shifted to intensive care unit, where he received 3 cycles of plasmapharesis with which he improved.

Three days later, the patient developed peri-orbital puffiness.A repeat urine R/E showed albumin = +, sugar = nil, pus cells = 3-5/hpf, and RBCs = numerous. The total urinary proteins were 163mg/dl, with 24 hour urinary proteins = 2.36gm and 24 hour urine output of 1.5liters. Urine for culture/sensitivity revealed no growth of micro-organisms. Total serum proteins were 4.1g/dl (6-8g/dl) while the serum albumin was 2.7g/dl (2.5-4.5g/dl). A renal biopsy was planned and patient started empirically on steroids. Report of the renal biopsy showed increased mesangial cellularity, inflammatory infiltrate including neutrophils and narrowing of capillary lumina, all suggestive of post-infectious glomerulonephritis.

DISCUSSION

Our patient had simultaneous onset of Guillain-Barré syndrome, acute hepatitis A followed by nephritic syndrome. There was no history of previous upper respiratory tract infection or any identifiable cause of post infectious glomerulonephritis. The patient was labeled as having Gullain Barre Syndrome and the diagnosis was established on basis of the acute clinical course, nerve conduction studies suggestive of acute motor axonal neuropathy and a finding of albumino-cytologic dissociation in CSF. Many granular casts and RBC casts were noticed in the urine sediment. Renal biopsy specimens showed increased mesangial cellularity, inflammatory infiltrate including neutrophils and narrowing of capillary lumina, all suggestive of post-infectious glomerulonephritis. The presence of IgM antibody against hepatitis A virus identified it as the cause of the acute hepatitis in this patient.

Association of GBS with viral hepatitis is well described. Reported cases are due to acute hepatitis B[4-6] and A[7], only few cases have been reported with non-A, non-B hepatitis. Neurological weakness occurs within 14 days of the onset of jaundice and more than 50% cases occur when the icteric illness is over[4]. Males are predominantly affected and proprioception is commonly involved. In most of the described cases the overall outcome was good, as was our in patient who had a significant improvement with three cycles of plasmapharesis. The diagnosis is essentially clinical and supported by nerve conduction studies[6]. Although the protein content of the CSF is usually high, it may be normal, especially in the early stages, and in some cases remains so[7]. The major danger in Guillain-Barre syndrome is paralysis of the respiratory muscles, but the prognosis is generally good. Approximately 75% of patients recover totally[8].

Guillain-Barre syndrome is often associated with some form of bacterial or viral infection. Respiratory infections are the most common[9]. The association with hepatitis is unusual. In a series of 1100 cases of Guillain-Barre syndrome Leneman[9] reported 11 associated with acute hepatitis. This report[9] and others[10,11] preceded the development of specific serological tests to identify the hepatitis viruses. Subsequently, a few cases of Guillain-Barre syndrome associated with hepatitis B have been reported[2-4] but there has only been one previous report associated with hepatitis A[12]. Guillain Barre syndrome has been observed to be associated with many diseases including many immunopathies which include, hashimoto's thyroiditis[13], temporal arteritis[14], autoimmune Interstitial lung diseases[15], Systemic lupus erythematosis[16], lupus nephritis[17], cutaneous or systemic vasculitis[18], Sjogren syndrome[19], myasthenia Gravis[20], Henoch schonlein purpura[21], Waldenstrom's disease[22], cryoglobinemia, hepatitis C[23], ulcerative colitis and there is also a report of Miller Fisher syndrome (a clinical variant of Guillain-Barre syndrome) and Crohn's disease[24]. MFS has been observed in patients with Adult onset Still's Disease[25]. Atypical GBS in association with acute rhabdomyolysis (causing an elevation of CK) has also been reported[26].

GBS has been observed in patients with Glomerulonephritis[27] (Focal segmental Glomerulonephritis, Extramembraneous Glomerulonephropathy or nephrotic syndrome). Our patient developed Guillain Barre Syndrome 1 week after he had acute hepatitis with nephritic syndrome; whether he developed acute hepatitis-induced immunological reaction as a result of which he developed Guillain Barre syndrome, and the same immunological reaction was responsible for his nephritic syndrome is a subject to discussion and the mechanism remains open to speculation.

REFERENCES

1. Fawaz KA, Matloff DS. Viral hepatitis in homosexual men. Gastroenterology 1981;81:537-8.
2. Ng P, Powell L, Campbell CB. Guillain barre

syndrome during preicteric phase of acute type B viral hepatitis. Aust N Z J Med 1975;5:367-9.

3. Niermeijer P, Gips CH. Guillain barre syndrome in acute HBsAg-positive hepatitis. Br Med J 1975;4:732-3.
4. Berger JR, Ayyar R, Shermata WA. Gullain barre syndrome complicating acute hepatitis B. A Case with detailed electrophysiological and immunological studies. Arch Neurol 1981;38:366-8.
5. Penner E, Maida E, Mamoli B, Gangle A. Serum and cerebrospinal fluid immune complexes containing hepatitis B surface antigen in gullain barre syndrome. Gastroenterology 1982;82;576-80.
6. Ray G, Ghosh B, Bhattayacharya R. Acute hepatitis B presenting as gullain barre syndrome. Indian J Gasteroenterol 2003;22:228. 6
7. Ono S, Chida K, Takasu T. Gullain barre syndrome following viral hepatitis A. Intern Med 1994;33:799-801.
8. Macleod WN. Sporadic non-A ,non B hepatitis and epstein barr hepatitis associated with gullain barre syndrome. Arch Neurol 1987;44:438-42.
9. Leneman F. The Gullain barre syndrome. Arch Int Med 1966;118:139-44.
10. Lescher FG. The nervous complications of infective hepatitis. Br Med J 1944;1:544-6.
11. Asbury AK. Hepatic neuropathy. In: Dyck PJ, Thomas PK, Lambert EH, editors. Peripheral Neuropathy. Philadelphia: WB Saunders; 1975. P. 993-8.
12. Johnston CLW, Schwartz M, Wansbrough-Jones MH. Acute inflammatory polyradiculoneuropathy following type: a viral hepatitis. Postgrad Med J 1981;57:647-8.
13. Behar R, Penny R, Powell HC Guillain barre syndromeassociated with hashimoto's thyroiditis. J Neurol 1986;233:233-6.
14. Roca B, Ferrer D, Calvo B. Temporal arteritis and guillain barré syndrome. South Med J 2002;95:1081-2.
15. Kashihara K, Marui K, Takahashi K, Abe K, Tanimoto Y. Inflammatory demyelinating polyradiculoneuropathy associated with interstitial lung disease. Clin Rheumatol 2000;19:235-7.
16. Vaidya S, Jasin HE, Logan J. Systemic lupus erythematosus and guillain-barre syndrome. J Clin Rheumatol 1999;5:349-53.
17. Van Laarhoven HW, Rooyer FA, van Engelen BG, van Dalen R, Berden JH. Guillain barré syndrome as presenting feature in a patient with lupus nephritis, with complete resolution after cyclophosphamide treatment. Nephrol Dial Transplant 2001;16:840-2.
18. Guilevin L, Cohen P, Gayraud M, Lhote F, Jarrousse B, Casassus P. Churg strauss syndrome: clinical study and long-term follow-up of 96 patients. Medicine (Baltimore) 1999;78:26-37.
19. Mochizuki H, Kamakura K, Masaki T, Hirata A, Nakamura R, Motoyoshi K. Motor dominant neuropathy in sjögren's syndrome: report of two cases. Intern Med 2002;41:1426.
20. Kizilay F, Ryan HF, Oh SJ. Mya stheni a gravis and guillain barre syndrome occurring simultaneously in the same patient. Muscle Nerve 2008;37:544-6.
21. Moreau BA, Schuller E, Georges B. Association of Guillain-Barré syndrome and Henoch-Schönlein purpura: is immunoglobulin a responsible for the neurologic syndrome? Am J Med Sci 1988;296:198-201.
22. Taillan B, Pedinielli FJ, Blanc AP. Association of Waldenstrom's disease and Guillain Barré syndrome. Presse Med 1985;14:844.
23. Lacaille F, Zylberberg H, Hagège H, Roualdès B, Meyrignac C, Chousterman M, et al. Hepatitis C associated with guillain-barré syndrome. Liver 1998;18:49-51.
24. Vinals M, Anciones B, Cruz Martinez A, Barreiro P. Sindrome de Miller-Fisher recidivante asociado a neuritis braquial. Neurologia 1989;4:61-5.
25. Desai SS, Allen E, Deodhar A. Miller Fisher syndrome in adult onset Still's disease: case report and review of the literature of other neurological manifestations. Rheumatology (Oxford) 2002;41:216-22.
26. Scott AJ, Duncan R, Henderson L, Jamall GA, Kennedy PGE. Acute rhabdomyolysis associated with atypical guillain barre syndrome. Postgrad Med J 1991;67:73-4.
27. Talamo TS, Borochovitz D. Membranous glomerulonephritis associated with the guillain barre syndrome. Am J Clin Pathol 1982;78:563-6.