VOLUME - 13, ISSUE - 10, OCTOBER - 2024 • PRINT ISSN No. 2277 - 8160 • DOI : 10.36106/gjra

Original Research Paper | Internal Medicine

GUILLAIN-BARRE SYNDROME (GBS) WITH ANTECEDENT CHIKUNGUNYA INFECTION WITH PRONOUNCED CARDIAC INVOLVEMENT-AN UNUSUAL CASE.

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ABSTRACT

Guillain-Barre Syndrome (GBS) is a post infectious immune mediated disease which is potentiated by the host's immune system. About 30% patients with GBS have had some illness 1-3 weeks prior to presentation. Usual associations are with C.Jejuni, CMV, COVID-19, EBV etc1, 2, and 3. It commonly manifests as acute inflammatory demyelinating polyneuropathy. (AIDP). Other forms are AMAN (acute motor axonal neuropathy), ASMAN-(acute motor sensory axonal neuropathy) and Miller Fisher syndrome. If neglected, GBS may lead to significant morbidity and mortality. Most common manifestations are ascending weakness with demyelinating neuropathy. But there are lots of variations and a huge range of clinical presentations and courses. So, some authorities proclaim GBS to be more of a syndrome . Chikungunya, an arboviral infection that usually causes a self-limiting acute febrile illness can rarely lead to GBS, as has been presented here in this case.4 Herein, we describe a case of 76 year old male who presented acutely with weakness of bilateral lower limbs. Clinical evaluation strongly pointed towards GBS and IvIg was promptly initiated. Because of myalgia, CPK was asked for which was found to be raised. That led to full work up which revealed that he was positive for Chikun Gunya (in serum AND in CSF). Complicating the matters was his severely derailed cardiac markers. The patient gradually improved and was discharged with no sequelae. This case is being presented because the rare association of GBS with CG with cardiac involvement, thrombocytopenia and severe deficiencies of B12 and D3. To the best of our knowledge, hardly 1-2 such cases have been reported in medical literature.

KEYWORDS : Guillain-Barre Syndrome, Chikungunya, cardiac involvement

INTRODUCTION

A 76 Year old hypertensive male presented with complaints of bilateral lower limbs & knee pains which rapidly progressed to weakness involving B/L lower limbs with shortness of breath.

On initial examination, Pulse-86/min, Bp-140/90 mmhg in supine position, spo2 98 % on RA, RBS 201 mg/dl. Motor power was initially 3/5 which rapidly reduced to 2/5 over 4-5 hours. All reflexes were hypo. The single breath holding count was alarmingly low at only 12. No crackles/wheezes were heard and there was no cardiac gallop.

Neurological Examination:
Cognition intact
Pupils BERL 2 CM,
Cranial Nerves: 3, 4, 6, facial -NAD
Motor Examination:
Power

	U/l	L/l
Right	3/5	2/5
Left	3/5	2/5

Reflexes:
ANKLE: B/L Absent, Truncal weakness + +
Plantar: B/L down ward
Knee: B/L + +
Biceps: B/L + +
Neck holding present, no neck rigidity
Sensory Examination: No Sensory Deficit

Clinical Impression and logical explanation: Clinically the diagnosis was GBS.

Table of Investigation:

Hb	11.6
WBC	7.37 (90/6/00/04)
Platelet	207K 168 1119174 82
Creatinine	2.03
Na/k	140/4.8
TROP-I	105
NT-PRO BNP	6285
CRP	52
PCT	0.43
HbA1C	5.7
Vit B12	113
Vit-D	14.5
Serum Chikungunya PCR	DETECTED
SGOT/SGPT	22/21
Lepto /Dengue/Malaria	Negative
AChR Ab	Detected (0.93)

Csf Study – Albumino Cytological Dissociation + (protein 53, Wbc 00). Pcr Csf Chikun Gunya Detected.

Mri- Brain Angio With Wss – Normal

Emg Ncv – Normal.

2 D Echo- Mod Concentric Lvh Hypertrophy.
lvef=60% G1dd.no As/ar.mild Mr.trivial Tr

Final Diagnosis: Guillain-Barre syndrome (GBS) with antecedent chikungunya infection (PCR positive in serum AND CSF) with cardiac involvement.

Course: (pls mention details)

Patient was started on weight based IVIG regime over 5 days. His weight was 70 kg ,so as per standard treatment regimen i.e IVIG 2 gm/kg ,total dose of 140 g ,was divided over 5 days as mentioned below, INJ.IVIG 30 GMS IV x 4 DAYS ,INJ.IVIG 20 GMS IV x 1 DAY ,first vial was given slowly over 4 hours ,remaining vials were given over 2 hours each ,with close

cardiac monitoring and blood sugar monitoring.
INJ.IVIG 10 gm X 3 vials – day 1
INJ.IVIG 10 gm X 3 vials – day 2
INJ.IVIG 10 gm X 3 vials – day 3
INJ.IVIG 10 gm X 3 vials – day 4
INJ.IVIG 10 gm X 2 vials – day 5

,His vit-b12 was 113,b-12 deficiency was corrected with iv B12 supplements , vit-d was 14.5 started on cap.Geo-D3 60 k once a week .

On day 5 of IVIG therapy patient was able to walk with support, he was discharged on day 8from admission.

Follow Up: When seen in OPD on day 10 after discharge, he reported near complete self-sufficiency, walked without support with NO objective CNS deficits. Single breath holding count had increased to 33.

DISCUSSION:

Guillain-Barre Syndrome (GBS) is an acute and frequently severe and fulminant polyradiculoneuropathy. Several lines of evidence support an autoimmune basis for AIDP. It's likely both cellular and humoral mechanisms contribute to tissue damage in AIDP .T-cell activation is suggested by the finding that elevated levels of cytokines and cytokine receptors are present in serum (interleukin (IL) 2, soluble IL-2 receptor and in CSF (IL-6,tnf-alpha ,interferon gamma)[5]

Circumstantial evidence suggest that GBS results from immune responses to non-self antigens (infectious agents ,vaccines)that misdirect to most nerve tissue through a resemblance of epitope (molecular mimicry) mechanism .The neural targets are likely to be glycoconjugates especially gangliosides.They are typically exposed on plasma membrane of cells ,rendering them susceptible to an antibody mediated attack .Gangliosides and other glycoconjugates are present in large quantity in human nervous tissues and in key sites ,such as nodes of Ranvier . Antiganglioside antibodies, most frequently to GM1, are common in GBS, particularly AMAN & AMSAN, whereas Anti-GQ1b IgG are found in > 90 % of patients with Miller Fisher Syndrome (MFS)[5] Taken together, these observations provide strong evidence that autoantibodies play an important pathogenic role in GBS. In AIDP, an early step in the induction of tissue damage appears to be complement deposition along outer surface of the Schwann cell. Activation of complement initiate a characteristics vesicular disintegration of the myeline sheath and damage to axons.[5]

Likely reason for Cardiovascular involvement in Chikungunya is presence of receptors for chikungunya virus like MXRA8 (low expression) and PBH (medium expression) [6]. Post-mortem myocardial biopsies usually reveal extensive necrosis and cytoplasmic viral inclusions in the cells. This damage induces the innate immune response, including the recruitment of natural killer cells (NKC) and the release of cytokines, gamma interferon and nitric oxide. T-lymphocytes can cause further damage by killing more cardiomyocytes which leads to fibrotic infiltration of the cardiac tissue causing Dilated cardiomyopathy, myocarditis and associated complications like arrhythmia ,myocardial infarction and sudden cardiac death.[7]

CONCLUSION:

In the case discussed above, GBS, itself a rare disease, was compounded by TWO more rarities. (A) Chikun Gunya as the aetiological agent (with muscle/joint involvement) and (B) significant cardiac affection as the other. Thrombocytopenia was the fourth jigsaw puzzle. Hence this report.

REFERENCES:-

1. Jacobs BC, Rothbarth PH, van der Meché FG, Herbrink P, Schmitz PI, de Klerk MA, van Doorn PA. The spectrum of antecedent infections in Guillain-Barré syndrome: A case-control study. Neurology. 1998;51(4):1110–1115. doi: 10.1212/wnl.51.4.1110. [PubMed] [CrossRef] [Google Scholar](1)
2. Guillain–Barré syndrome (2023). https://www.who.int/news-room/fact-sheets/detail/guillain-barré-syndrome. Accessed 08 Aug 2023.(2)
3. Kuwabara S. Guillain-Barré syndrome: Epidemiology, pathophysiology and management. Drugs. 2004;64(6):597–610. doi: 10.2165/00003495-200464060-00003. [PubMed] [CrossRef] [Google Scholar] (3)
4. Guillain-barré syndrome (GBS) with antecedent chikungunya infection: a case report and literature review Sreelakshmi V.,#1 Amrita Pattanaik, corresponding author#1 Srilatha Marate,1 Reeta S Mani,2 Aparna R. Pai,corresponding author3 and Chiranjay Mukhopadhyay1 https://www.ncbi.nlm.nih.gov/pmc/articles/PMC11008014/(**4**)
5. Harrisons Principals of Internal Medicine 21st edition chapter 447 (5)
6. Cardiovascular involvement and manifestations of systemic Chikungunya virus infection: A systematic review by María Fernanda Alvarez,1 Adrián Bolívar-Mejía,2 Alfonso J. Rodriguez-Morales,a,3,4 and Eduardo Ramirez-Vallejo3,4 https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5405794/ (**6**)
7. Cardiomyopathy and Death Following Chikungunya Infection: An Increasingly Common Outcome
8. Elizabeth M. Traverse,1 Hannah K. Hopkins,1 Vedana Vaidhyanathan,2 and Kelli L. Barr https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8293388/(**7**)