

GBS-Like Presentation of Hereditary Neuropathy

Kamala Kant Bhoi¹, Piyush Kumar Anshu², Anshuman Sahu³

¹Associate Professor, Department of Neurology, Shri Balaji Institute of Medical Sciences, Mowa, Chhattisgarh, India

²Associate Professor, Department of Neurology, Shri Balaji Institute of Medical Sciences, Mowa, Chhattisgarh, India

³MD Resident, Department of Medicine, Shri Balaji Institute of Medical Sciences, Mowa, Chhattisgarh, India

Abstract—Background: Charcot–Marie–Tooth disease (CMT) comprises a genetically heterogeneous group of inherited peripheral neuropathies. PLEKHG5-related disease is a rare autosomal-recessive disorder associated with lower motor neuron disease and intermediate CMT phenotypes. A hereditary neuropathy may be misdiagnosed as Guillain–Barré syndrome (GBS) when weakness progresses over a relatively short period. **Case Presentation:** A 34-year-old man presented with a GBS-like clinical picture of progressive weakness involving all four limbs. Detailed history revealed an insidious, slowly progressive course, with lower limbs more affected than upper limbs. Neurological examination showed Medical Research Council power of 4/5 in all limbs, distal sensory loss in the legs, areflexia in all limbs, bilateral pes cavus and mild intellectual disability. Cranial nerves were normal, with no fasciculations, muscle wasting or respiratory involvement. Nerve conduction studies demonstrated an axonal sensorimotor neuropathy involving all four limbs, predominantly the lower limbs. Electromyography showed neuropathic changes. Whole exome sequencing identified a heterozygous PLEKHG5 c.1535T>A (p.Val512Asp) missense variant with a variant allele fraction of 50.49%. The laboratory classified the variant as a Variant of Uncertain Significance (VUS), with ACMG evidence PM2 and PP3. No second significant PLEKHG5 variant was detected. **Discussion:** The clinical and electrophysiological findings support a chronic hereditary sensorimotor neuropathy rather than a straightforward acute inflammatory neuropathy. However, PLEKHG5-related disease is autosomal recessive, and a single heterozygous VUS does not establish a molecular diagnosis. The phenotype should therefore be interpreted cautiously, with consideration of confirmation, segregation analysis, assessment for a second PLEKHG5 allele and alternative inherited-neuropathy genes. **Conclusion:** In patients with a GBS-like presentation, an insidious progressive course, pes cavus, areflexia and axonal sensorimotor neuropathy should prompt evaluation for hereditary neuropathy. A heterozygous PLEKHG5 VUS is a potential genetic clue but should not be considered diagnostic without additional evidence.

Keywords— Charcot–Marie–Tooth disease; PLEKHG5; intermediate CMT; hereditary neuropathy; whole exome sequencing; variant of uncertain significance; quadriplegia; pes cavus; India; Chhattisgarh.

I. INTRODUCTION

Charcot–Marie–Tooth disease (CMT), also termed hereditary motor and sensory neuropathy, represents a broad group of inherited peripheral neuropathies characterized by progressive distal weakness, muscle wasting, sensory impairment, reduced or absent reflexes and skeletal deformities such as pes cavus. The disorder is genetically heterogeneous and may be inherited in autosomal-dominant, autosomal-recessive or X-linked patterns.[1-4]

CMT and related inherited neuropathies are among the most common inherited neurological disorders. A recent comprehensive review estimates that CMT and related neuropathies affect approximately 1 in 2,500 people globally, while epidemiological studies show substantial variation among populations.[1] A systematic review found reported CMT prevalence ranging from 9.7 to 82.3 per 100,000 population, reflecting differences in ascertainment and methodology.[5]

The clinical spectrum may be difficult to recognize in adults with acute or subacute deterioration, particularly when a patient is initially diagnosed with an acquired inflammatory neuropathy. The presence of pes cavus may provide an important clue to an underlying hereditary neuropathy, although it is not diagnostic by itself.[1-4]

PLEKHG5 encodes a pleckstrin-homology-domain-containing Rho guanine nucleotide exchange factor. Biallelic pathogenic variants have been associated with autosomal-recessive lower motor neuron disease and, subsequently, with

recessive intermediate CMT. The reported spectrum ranges from predominantly motor disease to intermediate sensorimotor neuropathy.[6-8]

We report an adult male with quadriplegia, pes cavus and mild intellectual disability in whom whole exome sequencing identified a heterozygous PLEKHG5 c.1535T>A (p.Val512Asp) VUS. The case is discussed in the context of global and Indian CMT epidemiology, the limited epidemiological information from Chhattisgarh, and the diagnostic significance and limitations of a single PLEKHG5 VUS.

II. CASE REPORT

2.1 Patient information

A 34-year-old male presented with a GBS-like clinical picture characterized by progressive weakness involving all four limbs. On detailed review, the onset was insidious and slowly progressive, with the lower limbs affected more than the upper limbs. The available clinical history documented a previous clinical diagnosis of GBS, with hereditary sensory motor neuropathy considered as an alternative diagnosis. Examination documented bilateral pes cavus and mild intellectual disability. The patient had a previous history of acute kidney injury associated with the illness diagnosed as GBS. There was no family history of a similar illness.

The phenotype terms used for whole exome sequencing analysis were: pes cavus, acute kidney injury, tetraplegia and generalized weakness of limb muscles.

2.2 Available clinical phenotype

Clinical feature	Available information
Age/sex	34-year-old male
Presenting syndrome	GBS-like presentation; insidious, slowly progressive weakness of all four limbs, lower limbs > upper limbs
Duration	Approximately 2 months
Previous clinical diagnosis	Guillain-Barré syndrome
Alternative consideration	Hereditary sensory motor neuropathy
Skeletal finding	Bilateral pes cavus
Cognitive finding	Mild intellectual disability
Previous systemic event	Acute kidney injury associated with the illness diagnosed as GBS
Family history	No similar illness reported

2.3 Differential diagnosis

The principal clinical considerations included GBS, hereditary motor and sensory neuropathy/CMT, hereditary motor neuropathy and other inherited neuropathies. The presence of pes cavus supports consideration of a longstanding hereditary neuropathy, whereas the relatively rapid two-month progression of quadriplegia is atypical for many classical CMT phenotypes and requires independent evaluation.

III. INVESTIGATIONS

3.1 Whole exome sequencing

Whole exome sequencing was performed on EDTA whole blood. The laboratory report states that the assay evaluates coding regions and splice junctions and can detect single-nucleotide variants, small insertions/deletions and copy-number changes within the exome. The report used a mean sequencing depth of >80–100× and an Illumina paired-end sequencing workflow with downstream GATK-based analysis aligned to GRCh38.

Parameter	Result
Mean depth	157.647086×
Target bases covered ≥1×	99.16%
Target bases covered ≥20×	98.71%
Total data generated	12.92 Gb
Reads >Q30	96.03%
Total filter-pass reads	100%
Total aligned reads	100%

3.2 Genetic finding

Parameter	Finding
Gene	PLEKHG5
Transcript	NM_020631.6
Exon	Exon 14 / Exon 21
Variant	c.1535T>A
Protein change	p.Val512Asp
Variant type	Missense
Zygosity	Heterozygous
VAF	50.49%
ACMG classification	Variant of Uncertain Significance
ACMG evidence	PM2, PP3
Reported phenotype association	Autosomal-recessive intermediate CMT

The report states that the p.Val512Asp substitution changes the amino acid at position 512 from valine to aspartic acid and may alter protein composition and physicochemical properties. The variant was reported in ClinVar as uncertain significance and was observed in 90 of 85,148 individuals of

South Asian ancestry in gnomAD. The laboratory therefore classified the variant as a VUS.

3.3 Electrophysiological findings

Nerve conduction studies demonstrated an axonal sensorimotor peripheral neuropathy involving all four limbs, predominantly affecting the lower limbs. Electromyography showed neuropathic changes without fasciculations. These findings support a chronic axonal sensorimotor neuropathy and, in the context of insidious progression and bilateral pes cavus, strengthen the clinical suspicion of an underlying hereditary peripheral neuropathy.

IV. INTERPRETATION OF THE PLEKHG5 VARIANT

4.1 Established PLEKHG5 disease

PLEKHG5 was initially identified as a gene responsible for an autosomal-recessive lower motor neuron disease with childhood onset. The original study identified a homozygous missense variant and provided functional evidence implicating impaired NF-κB signaling.[6]

Subsequent work demonstrated that biallelic PLEKHG5 mutations can cause an intermediate form of autosomal-recessive CMT. Affected individuals showed distal weakness and atrophy, foot deformities and intermediate nerve conduction abnormalities, establishing a broader PLEKHG5-related neurological spectrum.[7,8]

4.2 Significance of the present VUS

The present patient has a single heterozygous PLEKHG5 c.1535T>A (p.Val512Asp) variant. The laboratory classified it as a VUS and specifically noted that a second significant heterozygous PLEKHG5 variant was not detected. Because the established PLEKHG5-related CMT phenotype is autosomal recessive, a single heterozygous VUS is insufficient to establish the molecular diagnosis.[7,8]

The American College of Medical Genetics and Genomics/Association for Molecular Pathology framework emphasizes that a VUS should not be used as the basis for definitive clinical decision-making.[9]

V. EPIDEMIOLOGY

5.1 Global epidemiology

CMT is one of the most common inherited neurological disorders. Contemporary reviews describe a global burden of approximately 1 in 2,500 people, although prevalence estimates vary substantially among countries and study designs.[1,4] A systematic review of epidemiological studies found reported prevalence ranging from 9.7 per 100,000 in Serbia to 82.3 per 100,000 in Norway.[5] The broad range emphasizes differences in case ascertainment, diagnostic criteria, access to specialist assessment and historical differences in molecular classification.

The clinical phenotype is also variable. Typical manifestations include length-dependent weakness and wasting, cavovarus/pes cavus foot deformity, sensory abnormalities and reduced reflexes. Age at onset and severity may vary considerably, even among members of the same family.[1-4]

5.2 Epidemiology in India

Reliable population-based prevalence data for CMT in India remain limited. Much of the available evidence comes from tertiary neurological and genetic centres rather than nationwide population-based surveillance.

A Western Indian study of 22 patients demonstrated substantial clinical and genetic heterogeneity, with abnormalities involving genes including PMP22, GJB1, SH3TC2, HSPB1, SPTLC2, MPZ, AARS and NEFH.[10]

A larger Indian cohort of 55 probands with suspected inherited neuropathy used whole exome sequencing, clinical exome sequencing or targeted testing. Sixty-two variants were identified in 37 genes; 34 were classified as VUS and 45 were novel. A molecular diagnosis was established in 87% of the cohort, highlighting the considerable genetic diversity of inherited neuropathies in India.[11]

These observations indicate that Indian CMT is genetically heterogeneous and that rare variants may be encountered frequently in clinical sequencing. Consequently, interpretation of an isolated VUS requires particular attention to phenotype, inheritance and segregation rather than relying on gene-disease association alone.[11]

5.3 PLEKHG5 in India

There is no reliable population-based prevalence estimate for PLEKHG5-related neuropathy in India. A published Indian case described an individual with intermediate CMT and a homozygous PLEKHG5 p.Arg97Gln missense variant, supporting the occurrence of PLEKHG5-related disease in the Indian population.[12]

However, an isolated case does not provide a population prevalence estimate. The reported Indian patient had a homozygous variant, in contrast to the heterozygous VUS identified in the present case.[12]

5.4 Chhattisgarh

At present, robust population-based epidemiological data establishing the prevalence or incidence of CMT specifically in Chhattisgarh are not available in the material reviewed for this manuscript. Accordingly, a numerical prevalence estimate for the state should not be quoted without a population-based study or a validated genetic registry.

A scientifically appropriate statement is: "In Chhattisgarh, as in much of India, the true burden of Charcot-Marie-Tooth disease remains uncertain because population-based epidemiological and genetic registry data are limited. Available Indian studies demonstrate considerable clinical and molecular heterogeneity of inherited neuropathies, while PLEKHG5-related intermediate CMT appears to be exceptionally rare."

VI. REVIEW OF PLEKHG5-RELATED DISEASE

6.1 Genetic and biological basis

PLEKHG5 participates in intracellular signaling pathways including NF- κ B activation. The initial disease report demonstrated that a disease-associated PLEKHG5 mutation altered protein stability and localization and impaired NF- κ B signaling in experimental systems.[6]

6.2 Intermediate CMT phenotype

The identification of biallelic PLEKHG5 mutations in families with recessive intermediate CMT expanded the phenotypic spectrum of PLEKHG5 disease. Affected individuals had distal weakness and atrophy, foot deformities, sensory abnormalities and nerve conduction velocities in an intermediate range.[7,8]

6.3 Indian case report

The Indian case reported by Beijer and colleagues involved a homozygous N-terminal PLEKHG5 p.Arg97Gln variant and an intermediate neuropathy with scapular winging. The authors considered the phenotype and homozygous molecular finding supportive of a causative relationship.[12]

VII. COMPARISON OF PRESENT CASE WITH ESTABLISHED PLEKHG5 DISEASE

Feature	Present case	Established PLEKHG5-related disease
Age	34 years	Variable
Weakness	Quadruplegia/generalized limb weakness	Distal $\pm$ proximal motor weakness
Pes cavus	Present	Reported
Family history	No similar illness reported	Often compatible with recessive inheritance
PLEKHG5 result	c.1535T>A; p.Val512Asp	Biallelic disease-causing variants reported
Zygosity	Heterozygous	Biallelic
Classification	VUS	Pathogenic/likely pathogenic variants in established disease
Second PLEKHG5 allele	Not detected	Expected for recessive molecular diagnosis
Molecular diagnosis	Not established	Established when phenotype and biallelic variants are concordant

VIII. RECOMMENDED FURTHER EVALUATION

8.1 Nerve conduction studies and electromyography

Nerve conduction studies demonstrated an axonal sensorimotor neuropathy involving all four limbs, predominantly affecting the lower limbs. Electromyography demonstrated neuropathic changes without fasciculations.

8.2 Sanger confirmation and segregation

The laboratory recommends confirmation of the PLEKHG5 variant by Sanger sequencing and segregation analysis in affected and unaffected family members. These steps are appropriate because the current result is a VUS and because the inheritance pattern under consideration is recessive.

8.3 Search for a second allele

If clinical and electrophysiological findings strongly support PLEKHG5-related disease, additional testing may be considered for exon-level deletions/duplications, copy-number abnormalities, splice-region variants or other changes that may not be adequately detected by routine WES. The laboratory report specifically notes limitations of NGS for large deletions and duplications.

8.4 Alternative genetic diagnoses

If the PLEKHG5 finding remains non-diagnostic, reanalysis of the exome or broader genomic testing should be considered according to the phenotype. This is particularly relevant because Indian inherited-neuropathy cohorts demonstrate considerable molecular heterogeneity.[11]

IX. DISCUSSION

This case illustrates a clinically important diagnostic dilemma: a phenotype suggestive of hereditary neuropathy coexisting with a rare genetic variant that is biologically plausible but currently insufficient to establish causality. The presence of bilateral pes cavus is an important clue toward hereditary neuropathy, whereas the relatively rapid progression to quadriplegia over approximately two months is not typical of the usual slowly progressive course of classical CMT and should be evaluated independently.

The previous diagnosis of GBS introduces an additional challenge. A hereditary neuropathy may be overlooked when a patient develops acute or subacute deterioration, but the identification of a VUS should not automatically replace an acquired neurological diagnosis. The clinical syndrome should therefore be assessed using examination, electrophysiology and appropriate laboratory/imaging investigations.

The genetic result should be interpreted conservatively. The identified PLEKHG5 variant is heterozygous and classified as a VUS, and the laboratory did not identify a second significant PLEKHG5 variant. Thus, the molecular evidence does not currently meet the standard required to diagnose PLEKHG5-related recessive CMT.[7-9]

The case is nevertheless valuable because it demonstrates how WES can identify a potentially relevant rare variant in an adult with an atypical neurological phenotype while simultaneously illustrating the limitations of genomic testing. It also emphasizes the importance of avoiding premature diagnostic closure when a VUS is identified.

X. MANAGEMENT CONSIDERATIONS

Because a definitive molecular diagnosis has not been established, management should be based on the patient's clinical syndrome and functional impairment rather than the PLEKHG5 VUS. For confirmed CMT and related hereditary neuropathies, multidisciplinary care may include physiotherapy, gait and balance training, stretching, orthotic assessment, occupational therapy and prevention of falls and secondary complications.[13,14]

Any acute or subacute component of quadriplegia should be evaluated on its own merits, including consideration of inflammatory, infectious, metabolic, structural and other neurological causes when clinically indicated.

XI. LIMITATIONS

The present report has several limitations. Neurological examination demonstrated MRC power of 4/5 in all four limbs, distal sensory loss in the legs, generalized areflexia, and bilateral pes cavus, with no fasciculations or muscle wasting. Cranial nerve examination was normal and there were no

respiratory problems. NCS demonstrated an axonal sensorimotor neuropathy involving all four limbs, predominantly the lower limbs, and EMG showed neuropathic changes. The molecular finding remains a single heterozygous PLEKHG5 VUS and therefore does not establish PLEKHG5-related disease. Family segregation testing and definitive assessment for a second pathogenic allele have not yet been reported. State-specific epidemiological data for Chhattisgarh are also insufficient to support a numerical prevalence estimate.

The molecular finding is a single heterozygous VUS and therefore does not establish PLEKHG5-related disease. Family segregation testing and evaluation for a second allele have not yet been reported. Finally, state-specific epidemiological data for Chhattisgarh are insufficient to support a numerical prevalence estimate.

XII. CONCLUSION

CMT is a clinically and genetically heterogeneous inherited neuropathy with substantial variation in reported prevalence across populations. Indian studies demonstrate considerable molecular diversity, while reliable population-based data from Chhattisgarh remain limited. PLEKHG5-related intermediate CMT is an exceptionally rare autosomal-recessive disorder generally associated with biallelic disease-causing variants.

In the present patient, the heterozygous PLEKHG5 c.1535T>A (p.Val512Asp) variant is classified as a VUS and does not independently establish PLEKHG5-related CMT. Clinical–electrophysiological correlation, Sanger confirmation, segregation analysis and consideration of a second pathogenic allele or an alternative inherited-neuropathy gene are warranted.

XIII. LEARNING OBJECTIVES

- Recognize clinical features suggesting hereditary neuropathy in an adult with a GBS-like presentation, particularly pes cavus and slowly progressive weakness.
- Use neurological examination and NCS/EMG findings to characterize the neuropathy and distinguish axonal from demyelinating patterns.
- Understand that a heterozygous PLEKHG5 VUS does not establish PLEKHG5-related autosomal-recessive CMT.
- Apply appropriate genetic follow-up, including Sanger confirmation, segregation analysis and evaluation for a possible second pathogenic allele or alternative gene.
- Avoid premature diagnostic closure when the clinical phenotype and genetic findings are discordant.
- NCS/EMG is essential for electrophysiological classification.
- Sanger confirmation and family segregation studies are appropriate next steps.
- Discordance between phenotype and genotype should prompt reconsideration of alternative diagnoses or additional genomic testing.

PATIENT CONSENT

Written informed consent for publication of the case and relevant clinical and genetic information was obtained from the patient.

CONFLICT OF INTEREST

The authors declare no conflict of interest, unless otherwise applicable.

FUNDING

No specific funding was received for preparation of this case report, unless otherwise applicable.

REFERENCES

- Burns J, Timmerman V, Laurá M, Yiu EM, D'Antonio M, Mukherjee-Clavin B, et al. Charcot-Marie-Tooth disease and related neuropathies. *Nat Rev Dis Primers*. 2026;12:3.
- Skre H. Genetic and clinical aspects of Charcot-Marie-Tooth's disease. *Clin Genet*. 1974;6(2):98-118.
- Rossor AM, Saporta ASD, Boland-Freitas R, Gonzalez MA, Choi BO, Chung KW, et al. Clinical characteristics of Charcot-Marie-Tooth disease. *J Neurol Neurosurg Psychiatry*. 2018;89(12):124-137.
- Pareyson D, Marchesi C. Diagnosis, natural history, and management of Charcot-Marie-Tooth disease. *Lancet Neurol*. 2009;8(7):654-667.
- Barreto LCLS, Oliveira FS, Nunes PS, Costa IMPF, Garcez CA, Goes GM, et al. Epidemiologic study of Charcot-Marie-Tooth disease: a systematic review. *Neuroepidemiology*. 2016;46(3):157-165.
- Maystadt I, Rezsöházy R, Barkats M, Duque S, Vannuffel P, Remacle S, et al. The nuclear factor κ B-activator gene PLEKHG5 is mutated in a form of autosomal recessive lower motor neuron disease with childhood onset. *Am J Hum Genet*. 2007;81(1):67-76.
- Azzedine H, Zavadakova P, Planté-Bordeneuve V, Vaz Pato M, Pinto N, Bartsaghi L, et al. PLEKHG5 deficiency leads to an intermediate form of autosomal-recessive Charcot-Marie-Tooth disease. *Hum Mol Genet*. 2013;22(20):4224-4232.
- Han BS, et al. Mutations in the PLEKHG5 gene are relevant to autosomal recessive intermediate Charcot-Marie-Tooth disease. *BMC Med Genet*. 2013;14:95.
- Richards S, Aziz N, Bale S, Bick D, Das S, Gastier-Foster J, et al. Standards and guidelines for the interpretation of sequence variants: a joint consensus recommendation of the ACMG and AMP. *Genet Med*. 2015;17(5):405-424.
- Khadilkar SV, Patil ND, Kadam ND, Mansukhani KA, Patel BA. Clinico-electrophysiological and genetic overlaps and magnetic resonance imaging findings in Charcot-Marie-Tooth disease: a pilot study from Western India. *Ann Indian Acad Neurol*. 2017;20(4):425-429.
- Sharma S, Govindaraj P, Chickabasaviah YT, Siram R, Shrotri A, Seshagiri DV, et al. Genetic spectrum of inherited neuropathies in India. *Ann Indian Acad Neurol*. 2022;25(3):407-416.
- Beijer D, Polavarapu K, Preethish-Kumar V, Bardhan M, Dohm MF, Rebelo A, et al. Homozygous N-terminal missense variant in PLEKHG5 associated with intermediate CMT: a case report. *J Neuromuscul Dis*. 2022;9(2):191-198.
- Reilly MM, Shy ME, Laura M, Pareyson D. Charcot-Marie-Tooth disease. In: *GeneReviews®* [Internet]. Seattle (WA): University of Washington, Seattle; updated periodically.
- McCorquodale D, Pucillo EM, Johnson NE. Management of Charcot-Marie-Tooth disease: improving long-term care with a multidisciplinary approach. *J Multidiscip Healthc*. 2016;9:89-99.
- Rossor AM, Polke JM, Houlden H, Reilly MM. Clinical implications of genetic advances in Charcot-Marie-Tooth disease. *Nat Rev Neurol*. 2013;9(10):562-571.
- Saporta ASD, Sottile SL, Miller LJ, Feely SME, Siskind CE, Shy ME. Charcot-Marie-Tooth disease subtypes and genetic testing strategies. *Ann Neurol*. 2011;69(1):22-33.
- Fridman V, Bundy B, Reilly MM, Pareyson D, Bacon C, Burns J, et al. CMT subtypes and disease burden in patients enrolled in the Inherited Neuropathies Consortium natural history study: a cross-sectional analysis. *J Neurol Neurosurg Psychiatry*. 2015;86(8):873-878.
- Bird TD. Charcot-Marie-Tooth hereditary neuropathy overview. In: *GeneReviews®* [Internet]. Seattle (WA): University of Washington, Seattle.
- Landrum MJ, Lee JM, Benson M, Brown GR, Chao C, Chitipiralla S, et al. ClinVar: improving access to variant interpretations and supporting evidence. *Nucleic Acids Res*. 2018;46(D1):D1062-D1067.
- Lek M, Karczewski KJ, Minikel EV, Samocha KE, Banks E, Fennell T, et al. Analysis of protein-coding genetic variation in 60,706 humans. *Nature*. 2016;536(7616):285-291.
- 1000 Genomes Project Consortium. A global reference for human genetic variation. *Nature*. 2015;526(7571):68-74.
- McKenna A, Hanna M, Banks E, Sivachenko A, Cibulskis K, Kernysky A, et al. The Genome Analysis Toolkit: a MapReduce framework for analyzing next-generation DNA sequencing data. *Genome Res*. 2010;20(9):1297-1303.
- Burns J, Ryan MM, Ouvrier RA. Evolution of clinical phenotypes in Charcot-Marie-Tooth disease. *J Child Neurol*. 2009;24(7):849-855.
- Deenen JCW, Verbeek ALM, Verschuuren JJGM, van Engelen BGM, Voermans NC. Prevalence and incidence rates of 17 neuromuscular disorders: an updated review of the literature. *J Neuromuscul Dis*. 2025;12(1):1-25.

Address for Correspondence

Dr. Kamala Kant Bhoi, Associate Professor, Department of Neurology, Shri Balaji Institute of Medical Sciences, Mowa, Raipur-492001, Chhattisgarh, India
E-mail: drkkbhoinero@gmail.com