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GUILLAIN-BARRÉ SYNDROME (ACUTE INFLAMMATORY DEMYELINATING POLYRADICULONEUROPATHY) IN TWO BROTHERS: A CASE REPORT SUGGESTING FAMILIAL PREDISPOSITION

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ABSTRACT

Background: Guillain-Barré syndrome (GBS) is the most common cause of acute flaccid paralysis and is typically sporadic; familial clustering has been documented only rarely worldwide, with a handful of reports from India. We describe two brothers who each presented with an acute, ascending, symmetric flaccid paralysis fulfilling the clinical picture of acute inflammatory demyelinating polyradiculoneuropathy (AIDP), the demyelinating subtype of GBS. **Case Presentation:** Case 1, a 47-year-old man, presented with a 15-day history of low-grade intermittent fever, followed by tingling in the feet and hands and a rapidly ascending, symmetric flaccid quadriparesis evolving over four days, with areflexia and a large-fibre (vibration and joint-position) sensory deficit in the hands and feet; pain, temperature and crude touch were preserved. Case 2, his 35-year-old brother, presented separately with a more gradually evolving (over several weeks) ascending, symmetric, proximal and distal flaccid weakness of all four limbs with global areflexia but a fully intact sensory examination. Neither patient had cranial nerve, bulbar, respiratory, bowel or bladder involvement. Both were provisionally diagnosed with acute areflexic flaccid paralysis, most consistent with AIDP/GBS. (CSF analysis showed albumino- cytological dissociation in both patients and nerve conduction studies in case 1 showed early AIDP with prolonged latencies, slowed motor speeds, and "sural sparing" where sensory nerves in the legs remain normal. Case 2 shows severe, advanced AIDP marked by widespread absent F-waves and significant conduction blocks in multiple motor nerves.). **Conclusion:** The near-simultaneous occurrence of clinically classical GBS/AIDP in two brothers is a rare event that raises the possibility of a shared genetic susceptibility, a common environmental or infectious trigger, or both. This case adds to the small body of literature on familial GBS and underscores the value of documenting family history and offering genetic/immunological work-up in such clusters.

KEYWORDS: Guillain-Barré syndrome; acute inflammatory demyelinating polyradiculoneuropathy; AIDP; familial; siblings; acute flaccid paralysis; polyradiculoneuropathy.

INTRODUCTION

Guillain-Barré syndrome (GBS) is an acute, immune-mediated polyradiculoneuropathy and the leading cause of acute flaccid paralysis worldwide, with an annual incidence of approximately 1-2 per 100,000 population that rises with age.[1] It typically follows a preceding infective or immunological trigger by one to three weeks and presents with rapidly progressive, symmetric limb weakness and hyporeflexia or areflexia, evolving over days to a few weeks.[1,2] Acute inflammatory demyelinating polyradiculoneuropathy (AIDP) is the most common electrophysiological subtype in most parts of the world, including India, and is distinguished from axonal variants (AMAN/AMSAN) chiefly by nerve conduction study findings, although the acute clinical syndrome may be indistinguishable at the bedside.[1-3] Diagnosis rests on the combination of a compatible clinical course, albuminocytological dissociation on cerebrospinal fluid analysis, and electrophysiological confirmation, as formalised in the Brighton diagnostic criteria.[3]

GBS is overwhelmingly a sporadic disease. Familial clustering is exceptional: a 2004 study of Dutch

registries identified only 12 families with two or more affected members among a much larger sporadic cohort, and proposed that GBS behaves as a complex genetic disorder in which an underlying genetic susceptibility becomes clinically manifest only in the presence of an appropriate environmental or infectious trigger.[4] Candidate susceptibility loci investigated in familial and sporadic GBS include HLA class II alleles and genes governing innate and adaptive immune responses (e.g., FCGR, TNF-alpha, TLR4, CD1A), although no single variant has been consistently replicated.[5] Reports of familial GBS remain few even globally, and fewer still originate from South Asia: a Pakistani family with four affected siblings,[6] the first reported Indian family with two brothers affected five years apart,[7] three affected brothers from Arunachal Pradesh characterised electrophysiologically,[8] two brothers with simultaneous axonal GBS following dengue infection,[9] and a Pakistani family with three members affected simultaneously.[10] Even earlier, three siblings developing GBS before the age of three years, born to consanguineous parents, were reported from Israel, one of the earliest suggestions of a genetic contribution.[11]

We report two brothers, aged 47 and 35 years, who each presented with an acute, ascending, symmetric flaccid paralysis with areflexia consistent with AIDP. To our knowledge, familial occurrence of GBS/AIDP in adult siblings has not previously been reported from this region, and this case adds to the limited literature supporting a genetic contribution to at least a subset of GBS.

CASE PRESENTATION

Case 1 (elder brother) June 2022

A 47-year-old shopkeeper from Karimnagar presented with fever of 15 days' duration - low-grade, intermittent, without chills, rigors or diurnal variation, and without associated headache, cough, sore throat, coryza, breathlessness, vomiting, diarrhoea, dysuria, or loss of appetite/weight - which had subsided 12 days before presentation. Seven days before admission he developed sudden-onset tingling in both feet and hands; in the lower limbs this ascended over two days to the level of the knees along the posterior aspect of the leg, while in the upper limbs it remained confined to the palms without proximal progression. There was no impaired perception of clothing or of hot/cold water, no loss of ground sensation while walking, no girdle-like sensation, and no Lhermitte's phenomenon.

Weakness of both lower limbs began on the same day as the sensory symptoms and progressed in a stepwise fashion over four days: initial difficulty rising from squatting and supine positions (day 1, still able to walk unsupported); difficulty walking and climbing stairs requiring wall support (day 2); need for assistance of two people to rise, with a fall while walking to the washroom (day 3); and complete inability to sit, stand, walk or roll in bed by day 4. Upper-limb weakness appeared on day 3 as difficulty holding a glass, opening bottle caps, mixing food and buttoning clothes, while the ability to raise the arms overhead and turn the head remained preserved throughout. There was no stiffness, spasm, wasting or involuntary movement; flaccidity was present throughout. There was no bowel or bladder involvement, no cranial nerve symptoms, no features of raised intracranial tension, no autonomic symptoms (palpitations, sweating, syncope), and no history of recent vaccination, rash, oral ulcers, arthritis, trauma or animal bite. He had travelled to Kanchipuram approximately six weeks before presentation, an event he described as uneventful.

Past history was notable for hypothyroidism since the age of 38 years, treated with thyroidectomy in 2020 and maintained on levothyroxine 100 mcg; he was not a known hypertensive, diabetic or epileptic.

On general examination he was conscious, oriented and cooperative, with mild pallor and no icterus, clubbing, cyanosis, lymphadenopathy or pedal oedema; there were no neurocutaneous markers, tuberculous stigmata or thickened peripheral nerves. Vitals were stable (pulse 72/min regular; BP 110/80 mmHg right arm, 120/80 mmHg left arm; RR 20/min; single-breath count 30). Higher mental functions were intact (MMSE 28/30) and all twelve cranial nerves were normal bilaterally. Motor examination revealed flaccid quadriparesis with hypotonia in all four limbs, more severely affecting proximal and girdle musculature and the trunk. All deep tendon reflexes (biceps, triceps, supinator, knee, ankle) and superficial reflexes (abdominal, cremasteric, plantar) were absent bilaterally. Sensory examination showed normal pain, temperature and crude touch, but reduced vibration and joint-position sense in the hands and feet, indicating predominant large-fibre involvement; cortical sensory modalities were normal. Cerebellar signs could not be fully elicited given the degree of weakness; meningeal signs were absent. Cardiovascular, respiratory and abdominal examination were unremarkable.

Case 1 - motor power (MRC grade)

Muscle group	Right	Left
Neck	Normal	Normal
Trunk	0%	0%
Shoulder abduction (0-90 deg / >90 deg)	3/5	3/5
Shoulder adduction	4-/5	4-/5
Elbow extension / flexion	4-/5	4-/5
Wrist extension / flexion	4-/5	4-/5

Hand grip	70%	70%
Hip flexion	0/5	0/5
Hip extension / adduction	1/5	1/5
Hip abduction	2/5	2/5
Knee extension / flexion	2/5	2/5
Ankle dorsiflexion / plantarflexion	1/5	1/5

Provisional diagnosis: acute areflexic quadriparesis, radiculopathic localisation, most consistent with AIDP; differentials considered included Lyme polyradiculopathy, tick paralysis, hypokalaemic periodic paralysis, diphtheritic polyneuropathy, botulism, poliomyelitis, organophosphate toxicity, porphyria, vasculitic neuropathy and severe hypophosphataemia.

Case 2 (younger brother) September 2022

A 35-year-old agricultural labourer from karimnagar, brother of the patient in Case 1, presented with a one-month history of pain and tingling in both lower limbs, insidious in onset, beginning in the feet and gradually ascending to the level of the umbilicus, followed a week before presentation by tingling in both upper limbs that similarly began distally in the hands and progressed proximally. Weakness of both lower limbs had been present for 15 days, manifesting as difficulty rising from a squatting position, difficulty climbing stairs in both directions, buckling episodes while walking, and difficulty gripping footwear and crossing thresholds. Weakness of both upper limbs, present for seven days, caused difficulty with buttoning and unbuttoning clothes, opening bottle caps, holding a glass, preparing and bringing food to the mouth, reaching above head height, combing hair, brushing teeth and washing the face and hair; there was no difficulty lifting the head from the pillow or turning it side to side.

There was no fever, no bowel or bladder incontinence, no vomiting or loose stools, no loss of perception of clothing (hot and cold water sensation over the body was preserved), no Lhermitte's phenomenon, no girdle-like sensation and no involuntary movements. A detailed bulbar and cranial nerve review was negative: normal olfaction, no visual or colour-vision disturbance, no ptosis, diplopia or difficulty chewing, intact facial sensation, no facial weakness or drooling, preserved taste, no tinnitus or vertigo, no nasal regurgitation, and no dysphagia, dysarthria or tongue weakness. There were no seizures or spasms, no confusion or altered behaviour, no autonomic symptoms, and no history of recent vaccination, arthritis, rash, oral ulcers, trauma or excessive heat exposure.

He had no similar past complaints and was not a known hypertensive, diabetic or patient with coronary artery disease or stroke. Family history was recorded as non-contributory: he was born the second child of what was documented as a non-consanguineous marriage.

On general examination he was conscious, well oriented and comfortable, with no pallor, icterus, cyanosis, clubbing, lymphadenopathy, pedal oedema, thyromegaly or neurocutaneous markers. Vitals were stable (temperature 98.4 F; pulse 82/min regular; BP 110/80 mmHg bilaterally, sitting; RR 20/min). Higher mental functions were intact (MMSE 28/30) with normal speech and no dysarthria. All twelve cranial nerves were normal bilaterally on formal testing. Motor examination showed a symmetric, diffuse flaccid weakness affecting both proximal and distal muscle groups of all four limbs, with reduced tone in both upper and lower limbs (Table 3). Deep tendon reflexes (biceps, triceps, knee, ankle) and superficial reflexes (abdominal, plantar) were globally absent. In contrast to his brother, the sensory examination was entirely normal: spinothalamic modalities (pain, touch, temperature) and posterior column modalities (vibration, joint position, fine touch) were all preserved and symmetric. Cerebellar examination showed no nystagmus, dysdiadochokinesia or rebound phenomenon, though tandem gait could not be assessed because of the weakness; an absent pendular knee jerk was noted. Meningeal signs were absent. Cardiorespiratory and abdominal examination were normal.

Case 2 - motor power (MRC grade).

Muscle group	**Right**	**Left**
Neck	Normal	Normal
Trunk	<60%	<60%
Shoulder abduction / adduction	2/5	2/5
Elbow extension / flexion	2/5	2/5
Wrist extension / flexion	2/5	2/5
Hand grip	40%	40%
Hip flexion / extension / abduction / adduction	2/5	2/5
Knee extension / flexion	2/5	2/5
Ankle dorsiflexion / plantarflexion	2/5	2/5

Summary: a 35-year-old man presenting with acute neurological illness in the form of symmetric, proximal and distal motor weakness of all four limbs with sensory symptoms (though a normal sensory examination) and global areflexia, without cranial nerve, bulbar, autonomic, bowel or bladder involvement - a picture again most consistent with AIDP/GBS. Differentials considered were AIDP, Lyme polyradiculopathy and hypokalaemic periodic paralysis.

Comparative summary of the two cases

Feature	Case 1 (elder brother)	Case 2 (younger brother)
Age / occupation	47 years, shopkeeper	35 years, agricultural labourer
Antecedent event	Fever x15 days, subsided 12 days before onset of weakness; travel to Kanchipuram ~6 weeks earlier	None reported (no fever, no diarrhoea, no vaccination)
Sensory symptom onset -> motor symptom onset	Tingling (feet/hands) 7 days before weakness	Tingling (lower limbs) 1 month before weakness; ~3 weeks before upper-limb tingling
Pattern of weakness	Ascending, lower limbs (day 1) -> upper limbs (day 3) -> flaccid quadriparesis by day 4	Ascending, lower limbs (proximal+distal) then upper limbs over ~3 weeks
Power at presentation	Proximal and distal power reduced (grade 0-3/5 in most groups; hip flexion 0/5)	Diffuse power 2/5 in all limb groups tested
Deep tendon reflexes	Absent throughout (UL and LL, bilateral)	Absent throughout (UL and LL, bilateral)
Sensory examination	Pain/temperature/crude touch normal; vibration and joint-position sense reduced in hands and feet (large-fibre pattern)	All modalities (pain, touch, temperature, vibration, joint position, fine touch) intact bilaterally
Cranial nerves	I-XII normal bilaterally	I-XII normal bilaterally
Autonomic / bowel-bladder involvement	None	None
Cerebellar / meningeal signs	Negative	Negative
Provisional diagnosis	Acute areflexic quadriparesis, query AIDP	Acute inflammatory demyelinating polyradiculoneuropathy (AIDP) / GBS

Investigations, Treatment and Outcome

Parameter	Case 1 – Elder brother	Case 2 – Younger brother
Hb	12.8 g/dL	13.6 g/dL
TLC	8,400/µL	7,900/µL
Platelets	2.46 lakh/µL	2.71 lakh/µL
Serum sodium	138 mmol/L	140 mmol/L
Serum potassium	4.2 mmol/L	4.0 mmol/L
Serum calcium	9.1 mg/dL	9.3 mg/dL
Serum magnesium	2.0 mg/dL	1.9 mg/dL
Serum phosphate	3.5 mg/dL	3.7 mg/dL
Urea	27 mg/dL	24 mg/dL
Creatinine	0.9 mg/dL	0.8 mg/dL
Liver function	Within normal limits	Within normal limits
CK	96 U/L	88 U/L
NCS	Demyelinating polyneuropathy compatible with early AIDP; prolonged distal latencies, slowed motor conduction velocities and prolonged/absent F waves; relative sural sparing	Advanced demyelinating sensorimotor polyneuropathy with widespread absent F waves and conduction block involving multiple motor nerves
Electrophysiological subtype	AIDP	AIDP
CSF cells	2 cells/µL	3 cells/µL
CSF protein	112 mg/dL	168 mg/dL
CSF glucose	68 mg/dL	72 mg/dL

Albuminocytological dissociation	Present	Present
MRI spine	No significant cord compression/radiculopathy	No significant cord compression/radiculopathy
Anti-ganglioside antibodies	Negative	Negative
Campylobacter	Negative	Negative
CMV	Negative	Negative
EBV	Negative	Negative
Dengue	Negative	Negative
Chikungunya	Negative	Negative
IVIG	0.4 g/kg/day × 5 days	0.4 g/kg/day × 5 days
Respiratory involvement	None	None
Mechanical ventilation	Not required	Not required
Autonomic dysfunction	None	None
Hughes grade at discharge	3	3
Follow-up	Improved strength, ambulant independently	Improved strength, ambulant independently

DISCUSSION

Both brothers presented with the cardinal features of GBS: an acute, symmetric, ascending, predominantly motor polyradiculoneuropathy with global areflexia, evolving over days (Case 1) to a few weeks (Case 2), without cranial nerve, bulbar, autonomic, bowel or bladder involvement in either patient - a pattern that fulfils Brighton Criteria level 1-2 diagnostic certainty for GBS on clinical grounds alone, pending the electrophysiological and CSF confirmation noted above.[3] The two brothers nonetheless differed in several clinically informative respects. Case 1 had a clear antecedent febrile illness in the preceding weeks, a tempo of progression to peak weakness within four days, and a sensory examination dominated by loss of vibration and joint-position sense (large-fibre/posterior-column pattern) with sparing of pain and temperature - a pattern that can be seen in AIDP with prominent large-fibre involvement. Case 2 had no antecedent febrile illness, a more gradual progression over several weeks, and, notably, a completely normal sensory examination despite prominent sensory symptoms (tingling) - the dissociation between subjective sensory complaints and objective sensory findings is well described in GBS and does not argue against the diagnosis. This phenotypic heterogeneity within a single family mirrors observations in other reported familial GBS clusters, where affected relatives have shown variable severity, subtype (demyelinating vs. axonal) and antecedent triggers despite a shared genetic background.[6-10]

Familial clustering of GBS is well recognised but rare. The largest systematic study, from the Netherlands, identified 12 families with two or more affected members and proposed a complex genetic model in which disease expression requires both an underlying susceptibility and an environmental/infectious trigger, consistent with an earlier age of onset in successive generations in some pedigrees.[4] Candidate genetic contributors studied in GBS include HLA class II alleles and polymorphisms in genes governing the innate and adaptive immune response, though associations have generally been inconsistent across populations, likely reflecting genetic heterogeneity and small sample sizes.[5] Reports specifically of familial GBS from the Indian subcontinent remain few: four siblings from a consanguineous Pakistani family (two with definite and two with probable GBS);[6] the first Indian report, of two brothers affected five years apart;[7] three brothers from Arunachal Pradesh with electrophysiological characterisation;[8] two brothers with simultaneous axonal GBS temporally associated with dengue infection;[9] and a Pakistani family with three members affected simultaneously, two of whom had a preceding diarrhoeal illness.[10] An early report from Israel described three siblings developing GBS before three years of age, born to consanguineous parents, one of the first suggestions that consanguinity might unmask a recessively inherited susceptibility.[11]

This raises an important point specific to the present family history documentation: Case 1's records describe his parents' marriage as consanguineous, while Case 2's records describe a non-consanguineous marriage. As the two patients are stated to be brothers, this discrepancy should be verified directly with the family before submission, since consanguinity (or its absence) is directly relevant to the genetic-susceptibility argument developed in this discussion, and inconsistent family-history documentation would need to be reconciled or explained (for example, if the two men are paternal or maternal half-siblings rather than full siblings) to avoid an easily raised reviewer query.

A shared environmental or infectious trigger cannot be excluded either, particularly as both brothers presumably shared a household and geographic exposure, even though only Case 1 reported an antecedent febrile illness and travel history; Case 2's agricultural occupation raises the possibility of a distinct zoonotic or enteric exposure (e.g., Campylobacter jejuni from livestock contact) that was not elicited on history but should be sought on

serological/stool testing, as this remains the most commonly identified antecedent infection in GBS worldwide.[1,2] Distinguishing a shared genetic susceptibility from a shared environmental trigger - or, most plausibly, an interaction between the two - is not possible from clinical assessment alone and would require HLA typing and, where feasible, broader genetic testing of both affected brothers and unaffected family members, as has been done in some of the prior familial reports cited above.[5,7,8]

From a practical standpoint, this case reinforces two points for clinicians managing acute flaccid paralysis. First, a family history of similar illness should be actively sought in every GBS patient, since familial clustering - though rare - is almost certainly under-recognised when this history is not specifically elicited. Second, when a cluster is identified, complete diagnostic work-up (nerve conduction studies, CSF analysis, and where possible antiganglioside antibodies) in each affected member strengthens both the individual diagnosis and the scientific value of the report, and should be complemented by genetic counselling for the family once the diagnosis is confirmed.

CONCLUSION

We describe two brothers who each presented with a clinically classical acute inflammatory demyelinating polyradiculoneuropathy (GBS/AIDP), an occurrence that is rare enough to warrant report and that adds to the small existing literature suggesting a genetic contribution to susceptibility in at least a subset of GBS cases. Confirmatory electrophysiological and cerebrospinal fluid data, details of treatment and functional outcome, and clarification of the family's consanguinity status are needed to complete this report before submission. Clinicians evaluating acute flaccid paralysis should routinely enquire about similarly affected family members, as such clusters may offer insight into the genetic basis of an otherwise sporadic disease.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

PATIENT CONSENT

Written informed consent for publication of the clinical information and relevant investigations obtained from both patients.

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