Original Article

Post-Infective Polyneuritis: A Record Study (2005 - 2009)

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ABSTRACT

Background: Post-infective polyneuritis, also known as Guillain-Barre syndrome (GBS), is a rare and rapidly progressive immune-mediated disorder that consists of inflammation of nerves and usually causes the muscle weakness, sometimes progressing to complete paralysis.

Objective: This retrospective study was undertaken to assess the incidence, management and outcome of post-infective polyneuritis in District Faisalabad, Pakistan.

Methods: We reviewed all the 67 cases, 47 males and 20 females, who were admitted to the ICU of the Allied Hospital, Faisalabad during 2005-2009, and diagnosed as post-infective polyneuritis. The management comprised of treatment of the complications of the disease, and the specific therapy with plasmapheresis or high-dose immunoglobulin. Six paediatric cases were also admitted; two of them were found to be suffering from poliomyelitis. Thirteen cases of muscular weakness due to hypokalaemia mimicked the diagnosis of GBS.

Results: The specific therapy involving plasmapheresis or immunoglobulin resulted in (i) fewer patients needing mechanical ventilation, (ii) the decreased duration of mechanical ventilation, and (iii) reduction in the time needed for motor recovery and walking without assistance.

Conclusion: Early referral to ICU, management of complications, good nursing care and specific therapy with plasmaphresis or immunoglobulin within seven days of onset of the symptoms improve prognosis.

Keywords: Post-infective polyneuritis, IPPV, pneumothorax, hypokalaemia, poliomyelitis, plasmapheresis, immunoglobulin, autonomic crisis.

INTRODUCTION

Guillain-Barre syndrome or acute inflammatory demyelinating polyradiculo-neuropathy is typically a motor and to a lesser extent sensory peripheral neuropathy with sub-acute onset [1].

This inflammatory disease occurs in all parts of the world and in all seasons. It affects people of all ages and of both sexes [2]. The age range of 67 patients was 6 – 65 years with attack rate the highest in persons between 10 – 30 years.

Although the precise etiology is unknown, Guillain-Barre syndrome is immune-mediated and related to antibodies directed against peripheral nerve components. The association with antecedent infection suggests that certain agents may elicit immune responses involving antibodies that cross-react with peripheral nerve gangliosides [3]. In particular, the development of ganglioside antibodies has been observed in Guillain-Barre syndrome after campylobacter – jejuni infections, such as GM_1 antibodies in axonal forms of Guillain-Barre syndrome and GQ_{1b} antibodies in the Miller-Fisher variant of Guillain-Barre syndrome[4].

The major clinical manifestation is progressive weakness, usually most marked in the legs, associated with sensory complaints but without objective signs of sensory dysfunction [5]. Deep tendon reflexes are often significantly reduced or absent at presentation, though this finding may take several days to develop. In addition to lower extremities, the upper extremities, the trunk, intercostal and neck muscles are affected later, and the cranial muscles at last. The weakness can progress to total paralysis. Sensory loss occurs up to a variable degree and in some cases there is no sensory loss at all. Variants of clinical picture are frequent.

Diagnosis is established on clinical grounds and is confirmed by nerve conduction studies [6], showing slowing of conduction or conduction block. CSF proteins are frequently raised to 1 – 3 gm/liter.

The management of the patients with severe and protracted Guillain-Barre syndrome provides a major challenge, as the prognosis is generally excellent if complications can be treated early or avoided. These complications may be life-threatening, affect any of the major organs, systems, or result in permanent disability and can be prevented only by early diagnosis and intensive management. [6].

The specific therapy of GBS is plasma exchange (plasmapheresis) or immunoglobulin therapy which is as effective as plasmapheresis. Supportive therapy is the management of ventilatory failure [7], autonomic dysfunction, psychological and nutritional support and prevention of DVT and meticulous nursing care.

MATERIAL AND METHODS

Total number of patients included in this study was those admitted to ICU from Jan 2005 to Dec 2009. A total of 67 patients were included (Table-1).

Table-1
Distribution of Cases Owing to Sex

Sex	No. of Patients	Percentage
Male	47	70.2%
Female	20	29.8%
	(Total=67)	

Male to female ratio was found to be 2.39 to 1. Looking at yearly distribution, the number of patients admitted to the ICU is variable (Table-2).

Table-2
Yearly Increase of Patients of PIP

Year	No. of Patients	Male	Female
2005	15	11	04
2006	16	08	08
2007	16	14	02
2008	09	05	04
2009	11	09	02
TOTAL	67	47	20

Almost all patients had typical history of loss of muscle tone. There was only motor loss in 52 cases, and both sensory and motor loss in 15 cases (Table-3).

Table-3
Typical Clinical Representation

	Male	Female	Total
Motor loss	37	15	52
Motor & Sensory loss	10	5	15

Forty-eight patients out of sixty-seven needed ventilatory support (Table-4).

Table-4
No. of patients needing IPPV

Male	Female	Total
35	13	48

Yearly distribution of patients needing ventilatory support is shown in Table-5.

Table-5
Yearly Distribution of Patients Treated With IPPV

Year	Total	IPPV
2005	15	12
2006	16	12
2007	16	12
2008	09	07
2009	11	05
Total	67	48

On the whole, the mortality in our ICU is 22.3% (Table-6).

Table-6
Total Mortality

Total	67
Expired	15
Cured	52
Mortality	22.3%22.3%

It is comparable to International standards. The mortality rate in patients who needed ventilatory support was 26.08%, (Table-7).

Table-7
Morality Rate in Patients on IPPV

Total	46
Expired	12
Cured	34
Mortality	25%

Mortality of those patients who did not need ventilatory support is 19.04% (Table-8). Yearly mortality percentage is shown in Table-9.

Table-8
Mortality Rate in Patients Not on IPPV

Total	21
Expired	4
Mortality	19.04%

Table-9
Yearly Distribution (mortality)

Year	Total	Expired	Cured	Mortality
2005	15	4	11	26.66%
2006	16	3	13	18.75%
2007	16	4	14	25%
2008	9	2	7	22.22%
2009	11	2	9	18.18%

Table-10
CSF Proteins

	Normal	Raised
First Day	50	17
24 hours	37	30
48 hours	8	61
72 hours	8	61
96 hours	2	67

Diagnostic criteria which were kept in mind were reliance on typical history and loss of motor power on physical examination. The rise of CSF proteins level was noted to increase with the passage of time. Most of the patients showed high levels of proteins after four days (Table-10). The specific therapy with plasmaphresis led to (i) fewer patients needing mechanical ventilation, (ii) the decreased duration of mechanical ventilation for those who required it, (iii) and reduction in the time needed for motor recovery and walking without assistance. Plasma exchange was the most effective when carried out within seven days of the onset of symptoms (Table-14a). The plasma exchange schedule consisted of three to five exchanges of 1–2 plasma volume, each over 1–2 week(s). Adverse effects were common and were somewhat related to the disease itself. Fresh-frozen plasma had more side-effects than albumin as the replacement fluid. Specific therapy with immunoglobulin was as effective as plasma exchange (Table-14b) but was limited to a few patients because of being expensive.

Table -11
Relationship of IPPV to Duration of Illness

01 - 48 hours	30
48 - 72 hours	18
72 - 96 hours	10

Table-12
Incidence of IPPV to Raised CSF Proteins

CSF Proteins	No	IPPV
In 24 hours	17	17
In 48 hours	61	31
(Earlier the rise of CSF proteins, higher the incidence of IPPV.)		

Table-13
Management

Supportive	CVS Respiratory System Electrolyte Balance Nutritional Support Psychological Support Prevention of DVT Meticulous Nursing Care

Supportive management is cardiovascular, respiratory, fluid, electrolyte and nutritional balance, sedation, analgesia and good nursing care (Table-13). In addition to 67 cases of Guillain-Barre syndrome,

thirteen more cases were admitted to ICU diagnosed as GB Syndrome, indicating vomiting and diarrhea (Table-16).

Table-14(a)
Effects of Plasma Exchange

Duration from onset	Duration of Controlled ventilation	Motor Recovery
1 – 7 days	3 – 4 days	Within 7 days
7 – 15 days	8 – 10 days	After 10 days

Table-14(b)
Effects of Immunoglobulin Infusion

Duration from onset	Duration of Controlled ventilation	Motor Recovery
1 – 7 days	48 – 73 hours	5 – 7 days
7 – 15 days	10 – 12 days	After 10 days

Table-15
Associated Complications

Autonomic Crises and other complications	No. of cases	%age
Autonomic crises	30	44.73%
Polyurea	30	44.73%
Pneumothorax	2	2.98%
Pneumonia	5	7.46%
Haematemesis	5	7.46%

Table-16
Additional Cases Admitted as G.B. Syndrome

Total	13
Male	8
Female	5

There was no definite pattern of motor loss or sensory loss and CSF proteins were normal. On laboratory analysis, hypokalaemia was found to be the cause of muscle weakness (Table-17). Thirty out of forty-eight patients needed IPPV for 5 – 10 hour, Table-11, and there was no mortality. Five paediatric cases were also admitted (Table-19) as cases of Guillain-Barre syndrome. From clinical history, two out of six patients were found to be suffering from poliomyelitis (Table-19). They needed IPPV and there was no mortality (Table-20).

Table-17

History – Vomiting/Diarrhoea
No definite pattern of motor loss
No sensory loss
No CSF change
Lab Report Hypokalaemia

Table-18

IPPV	48 patients
Male	35
Female	13

Table-19
Paediatric Cases

Total	6 patients
G. B. Syndrome	4
Poliomyelitis	2

Table-20
Clinical Presentation

o Fever
o Weakness of respiratory muscles
o Paresis
o CSF normal
o All needed IPPV

DISCUSSION

For the first three years of the period of study (2005 – 2007), the number of patients admitted to our ICU and diagnosed as post-infective polyneuritis gradually

increased. For the next two years it decreased. This number is quite comparable to the well-established ICUs like the one in Massachusetts General Hospital where the number of cases per year are 10 – 15. [8] The decrease in number of cases coming to our institution could be due to: (i) lack of awareness in population and individual level, (ii) lack of recognition by General Practitioners, and (iii) the hesitancy of referral from medical unit to ICU.

The delayed referral of the patients from the medical wards to the ICU also affected the mortality rate. Most of the patients were referred only when they went into severe respiratory distress [9]. Respiratory infection already contracted in medical wards contributed to higher mortality of 19.04% in those patients who were on ventilator [10, 11]. Four out of twenty-one patients expired although they did not need any respiratory support and the cause of death in these patients was autonomic crisis. We had also noted that patients who developed Guillain-Barre syndrome at a fast rate had higher proportion of respiratory distress.

Thirty patients needed respiratory support within forty-eight hours. This clearly indicates that as soon as the patient is diagnosed, he should be transferred to ICU with all the supportive means at hand. [12, 13] In our cases, autonomic disturbances were noted in 44.73% of the cases, and 44.73% cases developed diabetes inspidus.

The patients who died in spite of respiratory support developed autonomic imbalance and resulted in loss of life. Looking into social setup, sixty-three patients belonged to families living in poor hygienic conditions. Only four cases belonged to the middle class.

The other conditions which can mimic the diagnosis of Guillain-Barre syndrome included thirteen cases of muscular weakness due to hypokalaemia. The shifting of these patients to ICU benefitted them. It is very difficult to differentiate in children between Guillain-Barre syndrome and poliomyelitis at the start of the treatment. In our series, two out of six children were suffering from poliomyelitis.

CONCLUSION

The management of the patient with severe and protracted GBS provides a major challenge, as the prognosis is generally excellent if complications can be treated early or avoided. For specific therapy, plasma exchange (plasmapheresis) is of value in GBS. Though it is expensive, immunoglobulin therapy is as effective as plasmapheresis. The finding is supported by other similar studies [14]. The diagnosis should be based on clinical features and the CSF proteins level. One should keep in mind electrolyte imbalance as a causative factor for muscular weakness. In children, poliomyelitis should be kept in mind as the first cause of muscular weakness as compared to Guillain-Barre syndrome.

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