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Management of Myasthenic Crisis in the Intensive Care Unit

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ABSTRACT

Introduction: Myasthenia gravis (MG) is an autoimmune neuromuscular disorder characterized by muscle weakness and fatigability due to impaired neuromuscular transmission. Approximately 15–20% of patients develop myasthenic crisis requiring endotracheal intubation and mechanical ventilation. Management is challenging in the presence of comorbidities, necessitating careful selection of immunomodulatory therapy.
Case Description: A 39-year-old woman was admitted to the General Intensive Care Unit of Dr. Hasan Sadikin Hospital with progressive dyspnea and generalized weakness. She was diagnosed with myasthenic crisis complicated by bradyarrhythmia, hypercoagulable state (elevated D-dimer, prolonged PT/INR/APTT), and electrolyte imbalance. Intravenous immunoglobulin (IVIG) was chosen over plasma exchange due to its non-invasive administration, avoidance of large-bore vascular access, more favorable hemodynamic profile, and lower risk of arrhythmia or hypotension. Potential arrhythmogenic effects from fluid shifts and hypotension associated with plasma exchange (reported in ~3% of cases) were considered contraindications in this patient. IVIG was administered at 0.4 g/kg/day for 5 consecutive days. Significant clinical improvement was observed, allowing successful extubation on day 8 and transfer to the High Care Unit on day 9.
Conclusion: This case demonstrates the efficacy and safety of IVIG as first-line immunomodulatory therapy in myasthenic crisis with complex comorbidities. A comprehensive multidisciplinary approach combined with appropriate selection of IVIG resulted in rapid clinical recovery and favorable outcome.

Keywords

Myasthenia Gravis, Myasthenic Crisis, Intravenous Immunoglobulin, Plasma Exchange, Intensive Care Unit.

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INTRODUCTION

Myasthenia gravis (MG) is a chronic autoimmune disorder characterised by the production of autoantibodies directed against components of the postsynaptic neuromuscular junction, most commonly the acetylcholine receptor (AChR) [1,2]. The hallmark clinical feature is fluctuating muscle weakness that worsens with sustained activity and improves at rest. In its most severe form, MG may precipitate a myasthenic crisis, defined as acute respiratory failure secondary to weakness of the respiratory and bulbar muscles, requiring mechanical ventilatory support [3,4]. Myasthenic crisis occurs in 15–20% of patients with MG during the course of their disease and remains a neurologic emergency with significant morbidity and mortality if not managed promptly in an intensive care setting [5,6].

The cornerstone of acute immunomodulatory therapy for myasthenic crisis consists of either therapeutic plasma exchange (TPE) or intravenous immunoglobulin (IVIG) therapy. Multiple randomised trials and meta-analyses have demonstrated comparable efficacy between these two modalities in achieving rapid clinical improvement and reducing the duration of mechanical ventilation [7,8]. However, the choice between TPE

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and IVIG must be individualised based on patient-specific factors, including haemodynamic stability, vascular access feasibility, coagulation profile, cardiac comorbidities, and institutional resources [9,10].

IVIG exerts its therapeutic effects through multiple mechanisms, including the neutralisation of pathogenic autoantibodies, inhibition of complement activation, modulation of Fc-receptor function, suppression of proinflammatory cytokines, and downregulation of autoreactive T- and B-cell responses [11,12]. In contrast to TPE, IVIG administration is noninvasive, does not require central venous catheterisation or specialised apheresis equipment, and is associated with a more stable haemodynamic profile [13]. These characteristics make IVIG particularly advantageous in patients with coagulopathy, bradyarrhythmia, advanced age, or limited vascular access, conditions that significantly increase the risk of TPE-related complications such as hypotension, arrhythmia, bleeding, thrombosis, and catheter-associated infection [14,15].This case report describes the successful intensive care management of a patient presenting with myasthenic crisis complicated by a hypercoagulable state and bradyarrhythmia, highlighting the clinical rationale for preferring IVIG over TPE and underscoring the importance of tailored immunomodulatory therapy in critically ill patients with complex comorbidities.

CASE DESCRIPTION

A 39-year-old woman (body weight 50 kg, height 156 cm) with a two-year history of acetylcholine receptor antibody-positive myasthenia gravis, previously treated with azathioprine 50 mg daily, presented to the emergency department of Dr. Hasan Sadikin Central Hospital with progressive symptoms. Seven days prior to admission, she developed difficulty walking, dysphagia requiring a soft/liquid diet, and bilateral ptosis. Three days before admission, profound limb weakness precluded lifting of the arms or legs, and two days prior, she became excessively somnolent. Over the final 24 h, the dyspnoea markedly worsened.

On arrival, the patient was fully conscious but in respiratory distress. Vital signs revealed blood pressure of 117/87 mmHg, heart rate of 56 beats/min (sinus bradycardia), respiratory rate of 40–46 breaths/min, and SpO2 of 90–93% on a non-rebreather mask at 15 L/min. Due to impending respiratory failure, she was intubated in the emergency department and transferred to the general intensive care unit (GICU) for further management. Initial ICU assessment showed Glasgow Coma Scale E3M5VETT (intubated), blood pressure 122/82 mmHg, heart rate 64 beats/min, and stable oxygenation on pressure-synchronised intermittent mandatory ventilation (P-SIMV) with pressure control of 10 cmH_2O, pressure support of 10 cmH_2O, PEEP of 5 cmH_2O, and FiO_2 of 60%. The pertinent laboratory findings on admission are summarised in Table 1.

Table 1. Key laboratory results on ICU admission

Parameter	Value	Reference Range
Hemoglobin	14.3 g/dL	12–15 g/dL
Hematocrit	43.2%	36–46%
Leukocyte count	6,930/µL	4,000–10,000/µL
Platelet count	222,000/µL	150,000–400,000/µL
Random glucose	90 mg/dL	<200 mg/dL
Urea	36.9 mg/dL	15–40 mg/dL
Creatinine	1.20 mg/dL	0.6–1.1 mg/dL
Sodium	122.6 mmol/L	135–145 mmol/L
Potassium	2.0 mmol/L	3.5–5.0 mmol/L
D-dimer	713 ng/mL	<500 ng/mL

The patient was diagnosed with a myasthenic crisis complicated by sinus bradyarrhythmia, a hypercoagulable state (elevated D-dimer with prolonged prothrombin time, INR, and activated partial thromboplastin time), severe hyponatraemia, and hypokalaemia. These comorbidities, together with haemodynamic considerations, guided the subsequent therapeutic strategies.

Table 2. Laboratory findings on intensive care unit admission

Parameter	Result	Reference Range	Units
Hemoglobin	14.3	12.0–15.0	g/dL
Hematocrit	43.2	36–46	%
Leukocyte count	6,930	4,000–10,000	/μL
Platelet count	222,000	150,000–400,000	/μL
Random blood glucose	90	<200	mg/dL
Blood urea nitrogen	36.9	15–40	mg/dL
Serum creatinine	1.20	0.6–1.1	mg/dL
Sodium	122.6	135–145	mmol/L
Potassium	2.0	3.5–5.0	mmol/L
D-dimer	713	<500	ng/mL FEU
Prothrombin time (PT)	Prolonged	11.0–13.0	seconds
International normalized ratio (INR)	Prolonged	0.8–1.2	–
Activated partial thromboplastin time (aPTT)	Prolonged	25–35	seconds

DISCUSSION

Myasthenia gravis (MG) is an antibody-mediated autoimmune disorder targeting the postsynaptic neuromuscular junction, with acetylcholine receptor (AChR) antibodies present in approximately 85% of generalised cases, whereas MuSK and LRP4 antibodies account for most of the remainder. A small seronegative subgroup exists, despite the use of sensitive assays [1,2]. The clinical hallmark is fatigable muscle weakness, often beginning with ocular symptoms and progressing to generalised disease in most patients. Myasthenic crisis, defined as acute exacerbation causing respiratory failure necessitating mechanical ventilation, complicates 15–20% of cases and remains associated with substantial morbidity if not aggressively managed in an intensive care setting [3,4].

Rapid immunomodulatory therapy is the cornerstone of the treatment. High-quality evidence from randomised controlled trials and systematic reviews confirms that intravenous immunoglobulin (IVIG) and therapeutic plasma exchange (TPE) are equally efficacious in achieving early clinical improvement, reducing the duration of mechanical ventilation, and shortening the ICU stay [5–7]. However, TPE is limited by its invasive nature, requirement for large-bore central venous access, citrate-induced hypocalcaemia, haemodynamic fluctuations, and an increased risk of catheter-related infection or thrombosis [8,9]. These complications are particularly undesirable in patients with coagulopathy, bradyarrhythmia, and limited vascular access.

In the present case, the laboratory findings at admission revealed a hypercoagulable state (D-dimer, 713 ng/mL, with prolonged PT, INR, and aPTT) alongside persistent sinus bradyarrhythmia (heart rate, 56–64 beats/min). These comorbidities render TPE relatively contraindicated because of the unacceptable risks of bleeding, arrhythmia exacerbation, and haemodynamic instability during rapid volume shifts [10,11]. Therefore, IVIG was administered at a standard dose of 0.4 g/kg/day for five consecutive days (total 2 g/kg body weight). This regimen exerts multiple immunomodulatory effects, including neutralisation of pathogenic autoantibodies, Fc-receptor blockade, complement inhibition, and cytokine modulation, without the procedural hazards of TPE [12,13]. The patient demonstrated marked clinical improvement within days, with progressive recovery of respiratory and bulbar muscle strength, successful extubation on day 8, and transfer to the high-care unit on day 9—outcomes fully consistent with large multicentre registries and recent meta-analyses of IVIG-treated myasthenic crisis [14-16].

Concomitant intensive care management adhered to the contemporary guidelines. Severe hyponatraemia (122.6 mmol/L) and hypokalaemia (2.0 mmol/L) were cautiously corrected to prevent central pontine myelinolysis. Early enteral nutrition was initiated within 48 h after confirmation of high nutritional risk by the NRS-2002 and modified NUTRIC scores, targeting 25–30 kcal/kg/day and 1.6–2.0 g/kg/day protein according to ASPEN/SCCM recommendations [17]. Pain and sedation were managed using validated behavioural scales (Figure 1).

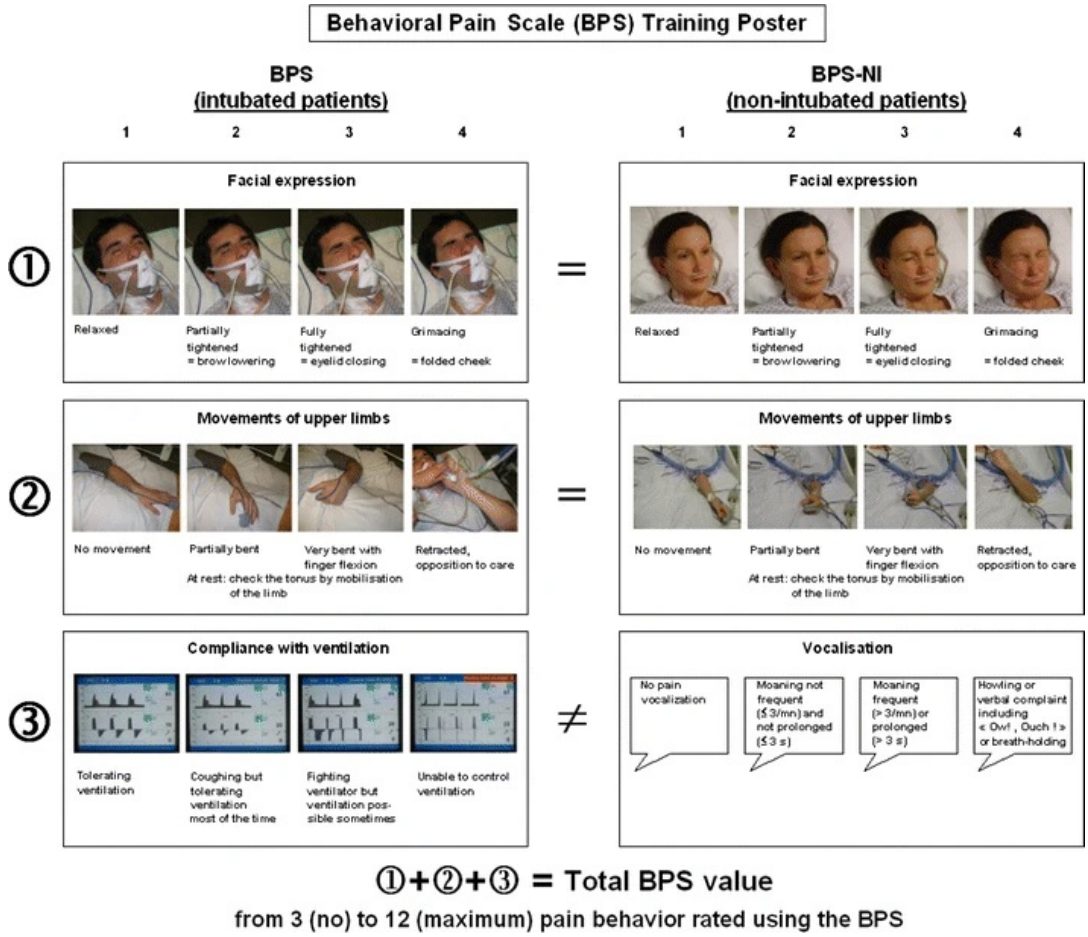

Figure 1. Pain Assessment (Behavioral Pain Scale)

with an early transition from midazolam to dexmedetomidine and low-dose fentanyl to minimise delirium and ventilator days in accordance with the 2018 PADIS guidelines [18]. Pharmacologic thromboprophylaxis with low-molecular-weight heparin was instituted after formal risk stratification using the Padua and IMPROVE scores (Figure 2) [19,20], and standard measures for stress-ulcer and aspiration prevention were applied.

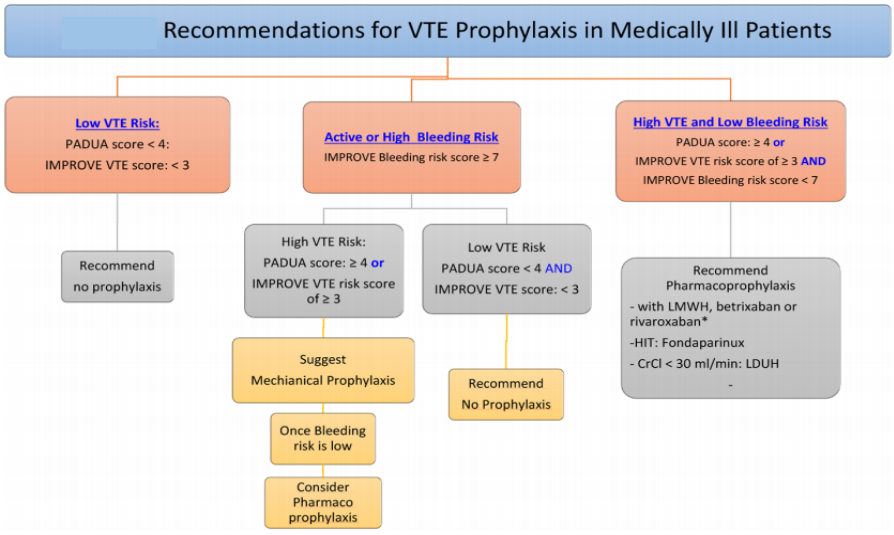

Figure 2. Recommendations for Thromboprophylaxis

This case underscores the critical importance of individualised immunomodulatory treatment in myasthenic crises. When complicated by hypercoagulability and bradyarrhythmia, IVIG is a safer, equally effective, and logistically superior alternative to plasma exchange [21,22]. When integrated within a comprehensive, evidence-based critical care protocol, IVIG reliably produces rapid neurological recovery and favourable short-term outcomes in high-risk populations.

CONCLUSION

This case confirms that intravenous immunoglobulin (IVIG) is a safe and highly effective first-line therapy for myasthenic crisis complicated by hypercoagulability and bradyarrhythmia, in which plasma exchange is relatively contraindicated. Administered at 2 g/kg over five days within a comprehensive, multidisciplinary ICU protocol, IVIG achieved rapid clinical improvement, successful extubation on day 8, and excellent outcomes without complications. IVIG should be prioritised for similar high-risk presentations.

DECLARATIONS

None

CONSENT FOR PUBLICATION

The Authors agree to be published in the Journal of Society Medicine.

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COMPETING INTERESTS

The authors declare no conflicts of interest in this case report.

AUTHORS' CONTRIBUTIONS

All authors made substantial contributions to the case report. APP was responsible for patient management, data collection, and initial drafting of the manuscript. All authors reviewed and approved the final version of the manuscript, ensuring its accuracy and integrity, and are accountable for all aspects of the work.

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