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Postgrad. Med. J. 2008;84;354-360
doi:10.1136/pgmj.2007.064915

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Approach to critical illness polyneuropathy and myopathy

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Received 28 September 2007
Accepted 8 June 2008

ABSTRACT

A newly acquired neuromuscular cause of weakness has been found in 25–85% of critically ill patients. Three distinct entities have been identified: (1) critical illness polyneuropathy (CIP); (2) acute myopathy of intensive care (itself with three subtypes); and (3) a syndrome with features of both 1 and 2 (called critical illness myopathy and/or neuropathy or CRIMYNE). CIP is primarily a distal axonopathy involving both sensory and motor nerves. Electroneurography and electromyography (ENG–EMG) is the gold standard for diagnosis. CIM is a proximal as well as distal muscle weakness affecting both types of muscle fibres. It is associated with high use of non-depolarising muscle blockers and corticosteroids. Avoidance of systemic inflammatory response syndrome (SIRS) is the most effective way to reduce the likelihood of developing CIP or CIM. Outcome is variable and depends largely on the underlying illness. Detailed history, careful physical examination, review of medication chart and analysis of initial investigations provides invaluable clues towards the diagnosis.

Acquired neuromuscular disorders (NMD) with flaccid weakness in critically ill patients have gained interest among neurologists, internists and critical care specialists alike, since its first modern description in 1977.¹ It has been estimated that about 46% of patients admitted with sepsis, multi-organ failure or on prolonged mechanical ventilation go on to develop NMD.² Neuromuscular weakness delays recovery and often causes prolonged ventilator dependence, and is an economic concern, apart from increased morbidity and mortality and prolonged hospital stay. NMD comprises three related yet distinct entities: (1) critical illness polyneuropathy (CIP); (2) acute myopathy of intensive care or critical illness myopathy (CIM); (3) a syndrome with features of both 1 and 2 (called critical illness myopathy and/or neuropathy or CRIMYNE). The acronym CRIMYNE was coined to describe the entity in which both CIP and CIM co-exist.³ Evaluating the extent of these neuromuscular diseases in the intensive care unit (ICU) is a challenging task which requires careful neurological assessment, review of administered medications, and investigational studies. Here we review the current understanding of critical illness polyneuropathy and critical illness myopathy and then propose a systematic approach to evaluating neuromuscular weakness in the intensive care unit.

HOW COMMON ARE THESE NEUROMUSCULAR DISORDERS IN CRITICAL CARE?

CIP and CIM are the most likely causes of weakness after ICU admission.⁴ An estimated

25–85% of critically ill patients will acquire a new onset neuromuscular cause of weakness⁵ of which CIP (an acute axonal polyneuropathy) is most prevalent. Although the exact incidence of it is unknown due to wide variation in diagnostic criteria and patients case mix, available data regarding the incidence of CIP in critically ill patients are rather impressive: 58% in patients with a prolonged (>1 week) ICU stay,⁶ 63% in patients with sepsis and >10 day ICU stay,⁷ 70% in patients with multiple organ failure,⁸ 76% in patients with septic shock,⁹ and 82% in patients with sepsis and multiple organ failure.¹⁰ Prolonged respiratory support, difficulty weaning from mechanical ventilatory support, and sepsis have been shown to be important risk factors for the development of CIP.¹¹ While CIM is being increasingly reported in the critical care setting following more frequent use of biopsy and neurophysiological studies, data on its incidence are lacking. However there is general agreement that CIM is at least as frequent as CIP. It needs to be emphasised that many patients who are diagnosed with CIP will show co-existing signs of myopathy on a muscle biopsy.¹²

The theoretical mechanisms of dysfunction in CIP and CIM are shown in fig 1.

CRITICAL ILLNESS POLYNEUROPATHY (CIP): PRESENTATION, PATHOLOGY AND TREATMENT

CIP is a sensorimotor polyneuropathy which was first described by Bolton and colleagues in 1984. CIP is usually observed in the ICU, especially in the setting of sepsis and multi-organ failure.¹³ However, it should be noted that, in most cases, a diagnosis of CIP is not easy, given that several factors make it difficult to diagnose: the severity of the underlying illness, frequently associated encephalopathy, and the uncanny use of non-depolarising neuromuscular blocking agents for sedation and paralysis. CIP can occur as early as 2–5 days in the presence of sepsis, or as late as 1 week post-mechanical ventilation.

Clinical features

CIP is a flaccid quadriparesis, often predominant distally, accompanied with loss of tendon reflexes.¹⁴ Failure to wean from mechanical ventilation may be the first recognised manifestation. Sensory loss can be present but is usually difficult to demonstrate in patients who are encephalopathic or under sedation. The presence of cranial nerve pathology suggests the possibility of alternative diagnosis (see below for a list of differential diagnosis), since they are normally spared in CIP.¹⁵

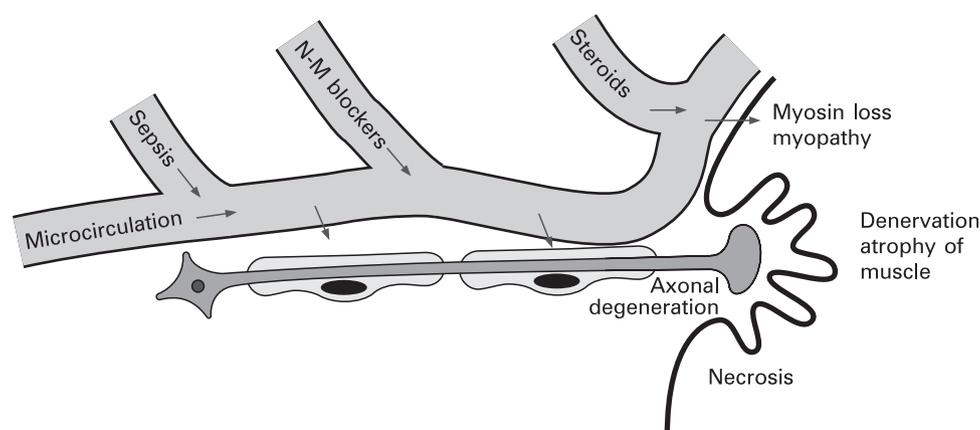


Figure 1 Theoretical mechanisms of dysfunction in critical illness polyneuropathy and myopathy. Sepsis with disturbance of microcirculation is a common underlying factor. Neuromuscular (N-M) blocking agents enhance denervation, and steroids subsequently induce a myopathy with thick filament myosin loss. Varying pathological changes in muscle may result. In the early stages of sepsis there may be a purely functional loss with no structural change in nerve or muscle. Adapted with permission from Bolton.²⁹

Pathology

CIP is primarily a distal axonopathy.¹⁶ This means that the affected nerves have a reduced number of axonal fibres and have relatively intact myelin sheaths. Usually both sensory and motor nerves are involved; however, pure motor and pure sensory forms have also been described.¹⁷ Despite our current advances in understanding CIP, the pathogenesis remains speculative. The current view of axonal degeneration relates to humoral and cellular responses generated in systemic inflammatory response syndrome (SIRS). It is postulated that SIRS leads to loss of vascular autoregulation and increased microvascular permeability, thereby resulting in endoneural oedema, hypoxia and capillary occlusion.¹⁸ This view is further supported by the fact that critically ill patients with high Acute Physiology and Chronic Health Evaluation III score and SIRS are most prone to develop CIP.¹⁹

Diagnostic electrophysiology

Electroneurography and electromyography (ENG-EMG) is the gold standard for diagnosis.²⁰ Nerve conduction studies show reduction or absence of both compound muscle and sensory nerve action potentials. Needle electromyography shows fibrillation potentials and positive sharp waves in resting muscle. Voluntary muscle activation shows recruitment of motor unit potentials with an increased recruitment ratio—a feature consistent with acute axonal loss. Significant slowing of nerve conduction or nerve conduction blocks would suggest alternative diagnostic possibilities such as the demyelinating form of Guillain-Barré syndrome (GBS).

Treatment

In a majority of cases physical therapy is the only effective rehabilitation treatment available.^{21 22} Where applicable appropriate treatment relies on identification of the cause (table 1). However, prevention of SIRS is the most effective way to reduce the likelihood of developing CIP.²³ Adherence to aseptic technique, avoidance of surgery, and cautious use of neuromuscular blocking agents is advised. Nutritional treatments including dietary therapies remain controversial. While some argue that certain dietary factors may contribute to neurogenic recovery, others argue that it may lead to

neuronal damage. However, there are only limited data in this area.²⁴ In a recent trial, intensive insulin therapy in critical illness was found to reduce substantially the incidence of CIP.²⁵ Other treatments that have been shown not to be effective in small studies include intravenous immunoglobulin (IVIg),²⁶ plasma exchange,¹⁸ anti-tumour necrosis factor (anti-TNF) antibodies,²⁷ interleukin 1 (IL1) receptor antagonists,²⁷ and N-acetylcysteine.^{28 29}

CRITICAL ILLNESS MYOPATHY (CIM): PRESENTATION, PATHOLOGY AND TREATMENT

William Osler, the great 19th century physician, was perhaps the first to describe a “rapid loss of flesh” in patients with prolonged sepsis.³⁰ However, it was not until the second half of the 20th century that critical illness myopathy was described as a distinct pathological entity in modern medical terms.¹ A definitive diagnosis of CIM requires a muscle biopsy. Based on muscle biopsies, three histopathological subtypes have been identified, especially in ICU patients: (1) a diffuse non-necrotising cachectic myopathy (critical illness myopathy or CIM); (2) myopathy with selective loss of thick (myosin) filaments (thick filament myopathy); and (3) the acute necrotising myopathy of intensive care.³¹ While many refer to them collectively as “CIM”, others prefer to subclassify them into subtypes. Some authors believe that the prognostic outcome of CIM is poor in the third subtype—that is, the necrotising variant—and relatively better in other subtypes. Key features of each subtype are listed in table 2.

Clinical features

There is proximal as well as distal muscle weakness. Sensation is spared but often cannot be evaluated. Reflexes are decreased in parallel with the decrease in strength. Ptosis and ophthalmoparesis can occur with prolonged neuromuscular blockade or myasthenia gravis (MG) but are rare with CIM.³²

Pathology

This is multifactorial and often represents a hypercatabolic complication of sepsis. It is associated with high use of non-depolarising muscle blockers and corticosteroids. Creatinine kinase (CK) may be elevated in up to 76% of patients.³³

Table 1 Risk factors, treatment and prevention of critical illness polyneuropathy and myopathy

Risk factors	CIP: sepsis, SIRS CIM: high dose corticosteroids and/or neuromuscular blocking agents, sepsis, SIRS, aminoglycosides, liver and lung transplants, liver failure, multi-organ failure, acidosis
Treatments	No specific pharmacological treatment available Early recognition often improves management Physical therapy is the only effective rehabilitation therapy available Cautious use of neuromuscular blocking agents (NMBA), especially in patients on corticosteroids Overuse of NMBA prevented by daily administration and using peripheral nerve stimulator or periodic return of muscle function When drug degradation/elimination is impaired, monitor for development of myopathy by serial CK measurement and repeated electrodiagnosis Aggressive medical management of sepsis Intensive insulin therapy in ICU patients may reduce likelihood of developing CIP/CIM

CK, creatinine kinase; CIM, critical illness myopathy; CIP, critical illness polyneuropathy; ICU, intensive care unit; NMBA, neuromuscular blocking agents; SIRS, systemic inflammatory response syndrome.

Diagnosing CIM

Electrophysiology

CIM is a difficult diagnosis to make. Nerve conduction studies show reduced compound muscle action potential amplitudes, preserved sensory responses, and normal repetitive nerve stimulation studies.³⁴ The EMG usually shows abnormal spontaneous activity in the form of fibrillation potentials and positive sharp waves of varying degrees which can have a neural origin. With voluntary muscle activation, small amplitude short duration motor unit potentials with early full recruitment can be seen. However, many patients with CIM cannot voluntarily contract their muscles either due to profound encephalopathy or sedation. It is important to emphasise that if the patient fails to volitionally activate his/her muscles, the ensuing ENG–EMG results will meet the criteria for diagnosis of CIP even if CIM is in play. This means that standard ENG–EMG cannot always differentiate between axonal and muscle dysfunction. The exception to this rule is with sensory CIP where electrophysiological identification of reduced sensory nerve action potentials cannot be a misinterpretation of primary muscle weakness.³⁵ In order to overcome this limitation it has been suggested that direct muscle stimulation can be used as an alternative test to diagnose CIM in acutely ill or uncooperative patients.³⁶ In this

investigation, a compound muscle action potential is obtained through direct stimulation of muscle fibres (dmCMAP), so that both the motor nerve and the neuromuscular junction are circumvented. When combined with a standard ENG–EMG, a differential diagnosis between myopathy (the muscle is inexcitable at both ENG–EMG and direct muscle stimulation) and neuropathy (the muscle is inexcitable at ENG–EMG, but excitable upon direct muscle stimulation) can be made. Myopathy, meanwhile, is more confidently diagnosed as an absolute reduction in dmCMAP below cut-off values.

Muscle biopsy

Muscle biopsy is the diagnostic method of choice for detection of structural abnormalities, but it is invasive and cannot reasonably be repeated in the same patient. Additionally, acute myopathy in intensive care has a continuum of pathological presentations: at one end of the spectrum there are forms with massive muscle necrosis (acute necrotising myopathy), and at the other end there are forms in which the muscle is structurally intact despite being electrically inexcitable. Therefore, routine muscle biopsy to diagnose CIM does not always exhibit clear pathology and may be questionable.³⁷ Non-invasive and easily repeatable neurophysiological investigations such as direct muscle stimulation may be superior to histological investigations in demonstrating CIM in acutely ill or uncooperative patients. However, this is not an accepted standard among neuromuscular specialists globally.

Treatment

There is no specific pharmacological treatment for CIM. Early recognition, avoidance of risk factors (table 1), and aggressive treatment of sepsis may partly reduce the incidence of CIM. Several studies have sought to evaluate the roles of different nutritional factors in the management of CIM, but none have found any measured treatment regimen to cause a statistically significant improvement in survival. A recent meta-analysis has shown that high dose parenteral glutamine supplementation reduces complications from infection and shortens the length of hospital stay.³⁸ Although this meta-analysis did not explore the effects of glutamine supplement directly on CIM, one could infer that patients with a shorter length of stay might have a lower incidence of ICU acquired myopathy. Special attention should be paid to patients with chronic alcohol abuse as they are predisposed to development of more severe and clinically

Table 2 Salient features of different subtypes of acute myopathy in intensive care

Critical illness myopathy	Thick filament myopathy	Necrotising myopathy of intensive care
Changes are often small and accompany CIP. Affects both types of muscle fibres, occasionally may be restricted to type II myofibres	Associated with selective loss of myosin filaments or "thick filaments"	Prominent myonecrosis along with vacuolisation and phagocytosis of muscle fibres
Histological changes include abnormal variation of muscle fibre size, fibre atrophy, angulated fibres, internalised nuclei, rimmed vacuoles, fatty degeneration of muscle fibres, fibrosis, and single fibre necrosis	Histological changes include altered staining of atrophic fibres with adenosine triphosphatase staining. Neurogenic changes are usually absent. Electron microscopy reveals focal or diffuse loss of myosin filaments. Progression from selective thick filament loss to diffuse myonecrosis is possible	Histological changes include panfascicular necrosis
Inflammatory changes are conspicuously absent, as in CIP	CK may be elevated	May progress to frank rhabdomyolysis
CK values are often normal	Often associated with high dose corticosteroids and neuromuscular blocking agents	CK is frequently elevated
May represent a hypercatabolic complication of sepsis and SIRS	Prognosis better than necrotising myopathy	Often history of being non-septic and placed on high dose corticosteroids, neuromuscular blocking agents, or both
Prognosis is good		Prognosis is poorer than the other two subtypes

CIP, critical illness polyneuropathy; CK, creatinine kinase; SIRS, systemic inflammatory response syndrome.

Box 1: Causes of generalised weakness in the intensive care unit (ICU) (*disorders seen after admission to ICU)**Muscle diseases**

- ▶ Acute myopathy of intensive care* (subtypes: critical illness myopathy, thick filament myopathy, acute necrotising myopathy of intensive care)
- ▶ Inflammatory myopathies: polymyositis, dermatomyositis
- ▶ Myopathy secondary to dyselectrolytaemia-like hypokalaemia, hypophosphataemia
- ▶ Rhabdomyolysis*
- ▶ Muscular dystrophy
- ▶ Mitochondrial myopathies
- ▶ Acid maltase deficiency
- ▶ Pyomyositis*
- ▶ Periodic paralysis

Neuromuscular junction disorders

- ▶ Neuromuscular blocking agent induced weakness*
- ▶ Antibiotic induced myasthenia
- ▶ Myasthenia gravis
- ▶ Organophosphorus poisoning
- ▶ Snake envenomation
- ▶ Insect/marine toxins
- ▶ Lambert–Eaton myasthenic syndrome
- ▶ Congenital myasthenic syndromes
- ▶ Hypermagnesaemia
- ▶ Botulism
- ▶ Tick paralysis

Peripheral neuropathies

- ▶ Critical illness polyneuropathy*
- ▶ Guillain–Barré syndrome (acute inflammatory demyelinating polyneuropathy)
- ▶ Chronic idiopathic demyelinating polyneuropathy
- ▶ Phrenic neuropathies*
- ▶ Toxic neuropathy
- ▶ Porphyric neuropathy
- ▶ Vasculitic neuropathy
- ▶ Diphtheria
- ▶ Lymphoma
- ▶ Cytomegalovirus related polyradiculoneuropathy

Anterior horn cell disorders

- ▶ Amyotrophic lateral sclerosis
- ▶ Paraneoplastic motor neuron disease
- ▶ West Nile virus infection–acute flaccid paralysis*
- ▶ Spinal muscular atrophy
- ▶ Acute poliomyelitis

Spinal cord disorders

- ▶ Trauma
- ▶ Haematoma
- ▶ Spinal cord infarction
- ▶ Epidural abscess
- ▶ Demyelinating: multiple sclerosis, Devic's disease, transverse myelitis
- ▶ Infective myelitis: coxsackievirus A, B, cytomegalovirus, legionella
- ▶ Paralytic rabies ("dumb rabies")

Brain pathology

- ▶ Cerebrovascular accident (infarction, haemorrhagic)
- ▶ Demyelinating: multiple sclerosis, acute disseminated encephalomyelitis

deleterious CIM in the presence of sepsis. Earlier nutritional intervention might benefit this group of patients in particular.

OUTCOMES IN PATIENTS WITH CIP AND CIM

A short term outcome of practical and economic importance to note is that duration of weaning from artificial ventilation is two to seven times greater in patients with CIP than in patients without CIP.³⁹ With respect to long term clinical outcomes, Lacomis and colleagues⁴⁰ reported in a prospective case study of ICU patients that about a third of both CIP and CIM patients died in the acute phase; a third were ambulatory within 4 months; and the rest took 4–12 months to recover or remained ventilated. Following discharge, many patients diagnosed with CIP or CIM complain of profound muscle weakness and chronic disability.⁴¹ Persisting mild disabilities that are common, even in patients with complete functional recovery, include reduced or absent deep tendon reflexes, stocking and glove sensory loss, muscle atrophy, painful hyperaesthesia, and foot drop.

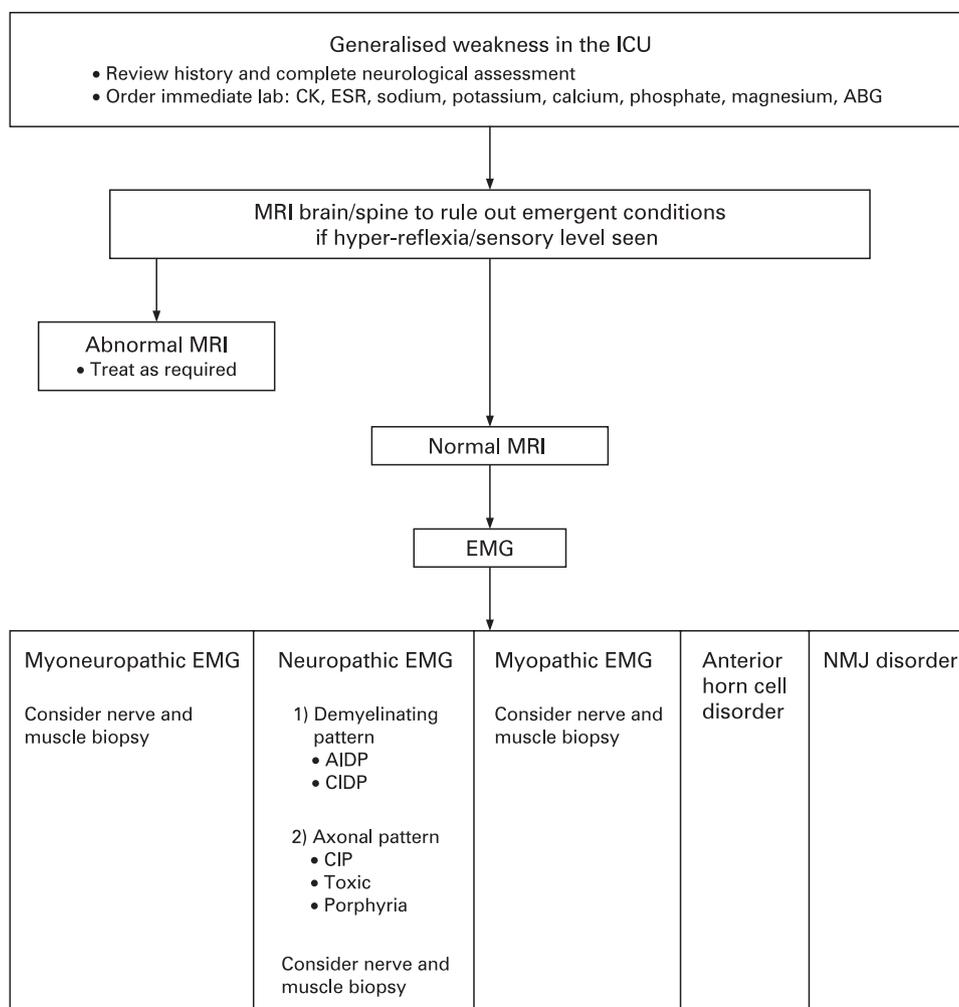
APPROACH TO THE PATIENT WITH NEUROMUSCULAR WEAKNESS IN ICU

Critically ill patients may develop weakness or paralysis due to a number of interacting factors including: sepsis, SIRS, multiple organ dysfunction syndrome, medications, and electrolyte and endocrine disturbances. Evaluating these patients can be difficult, especially when they are confused, sedated or intubated. Communication is difficult in such patients and may obscure the presence of motor weakness or sensory disturbances. Failure to wean from the ventilator or incidental finding of decreased limb movements is often the first point in identifying and requesting for neurological consultation. For simplicity patients with neuromuscular weakness in the ICU can be classified into three groups:

1. Patients with pre-existing neuromuscular disorders that have led to ICU admission. GBS and MG are examples of this type of weakness.
2. New onset or previously undiagnosed neurological disorder which progresses following admission to ICU. Medications exacerbating weakness in a previously undiagnosed MG is an example of this type of weakness.
3. Neuromuscular weakness arising as a complication of non-neuromuscular critical illness during the stay in ICU. CIP/CIM are examples of this type of weakness.

During the initial assessment, effort should be made to establish the category in which the present weakness falls as it helps to limit the differential diagnosis and investigations. A list of differential diagnosis for generalised weakness in the ICU has been tabulated according to anatomical level in box 1. Disorders which typically can arise after admission to the ICU are marked with an asterisk. Detailed history, careful physical examination, review of medication chart, and analysis of initial investigations provides invaluable clues towards the diagnosis and further investigation. The clinical setting in which the patient is seen/examined also tends to influence the differential diagnosis. In general, patients seen after cardiothoracic procedures (especially aortic repair) tend to have ischaemic myopathies, and patients in medical ICU with critical illness, sepsis, or SIRS tend to have CIP or CIM or both. Detailed electrophysiological investigation, serum creatine kinase values, other laboratory studies, and histological examination of muscle biopsy help in the diagnosis. If a central nervous system (brain or spinal cord) lesion is suspected, neuroimaging studies are required. In addition to conventional nerve conduction and needle electromyography,

Figure 2 Approach to neuromuscular weakness in intensive care. ABG, arterial blood gases; AIDP, acute inflammatory demyelinating polyradiculopathy; CIDP, chronic inflammatory demyelinating polyradiculopathy; CIP, critical illness polyneuropathy; CK, creatine kinase; EMG, electromyography; ESR, erythrocyte sedimentation rate; ICU, intensive care unit; MRI, magnetic resonance imaging; NMJ, neuromuscular junction. Adapted with permission from Maramattom *et al.*⁴²



phrenic nerve conduction, diaphragm electromyography, blink reflex and, recently, the technique of direct muscle stimulation have been employed in determining the cause of acute weakness in the ICU patient. However these studies remain highly specialised and are available only in select centres. A systematic approach to investigations for neuromuscular weakness has been suggested by Maramattom and colleagues⁴² (fig 2).

IMPORTANT RESEARCH QUESTIONS

Diagnostic

A simplified electrophysiological investigation such as refined direct muscle stimulation is necessary to diagnose CIM promptly at the bedside. A recent multi-centre trial has evaluated the use of a simplified electrophysiological test to screen critically ill patients for CRIMYNE.⁴³ The trial concluded that a peroneal compound muscle action potential reduction two standard deviations below normal values accurately identified patients with CRIMYNE. The use of rapid horizontal pore gradient SDS-PAGE for determining the myosin/actin ratio has also been suggested as a diagnostic tool for identifying CIM.⁴⁴ Further development of these tools would greatly aid diagnosis. Although this does not currently greatly affect management it will help in defining the disease and appropriately targeting experimental therapies.

Therapeutic

Early treatment with IVIg at the time of diagnosis of sepsis has been suggested and examined in a few small studies. Whether this can mitigate the development of CIP or CIM remains uncertain but has been suggested as a possibility.⁴⁵ IVIg would seem unlikely to specifically alter the pathogenesis of CIP or CIM since these entities are believed to be primarily caused by toxic insult rather than being immune mediated. Indeed, a Canadian advisory panel has recently reviewed the use of IVIg in neurologic conditions and did not recommend it as a treatment in CIP.⁴⁶ Recombinant activated protein C has been shown to reduce morbidity and mortality in septic patients; while it is tempting to infer that it may improve outcomes in CIP/CIM, its role in CIP/CIM has not been specifically addressed.²⁷

CONCLUSION

Neuromuscular weakness in the critically ill patient is a common problem encountered in the ICU and is associated with high mortality and morbidity. The essential feature of muscle weakness is easily overlooked in these patients who are often unresponsive or have life threatening comorbidities. Clinicians working in this area should be aware of the risk factors for the development of neuromuscular weakness, have a high index of suspicion, and actively seek out any suggestive signs. Either neuropathy or myopathy can exist alone, but there

Learning points

Neuromuscular weakness in the intensive care unit

- ▶ Common
- ▶ High mortality
- ▶ Considerable healthcare costs

Critical illness polyneuropathy

- ▶ Axonal neuropathy
- ▶ Caused by toxins/systemic immune response
- ▶ CK values usually normal
- ▶ Electrophysiology diagnostically useful

Critical illness myopathy

- ▶ Myopathy with spectrum of pathology
- ▶ CK elevated in some types

Risk factors

- ▶ Sepsis
- ▶ Systemic inflammatory response syndrome
- ▶ High dose corticosteroids
- ▶ Use of neuromuscular blockers
- ▶ Aminoglycosides
- ▶ Liver/lung transplant
- ▶ Liver failure
- ▶ Multi-organ failure
- ▶ Acidosis

Treatment

- ▶ No proven specific therapy
 - but intensive insulin therapy may reduce risk
- ▶ Prevention
 - careful use of neuromuscular blockers
 - serial CK measurements
 - repeat electrophysiology
 - aggressive medical management of sepsis

is often an overlap clinically, pathologically and electrophysiologically. CIP is a distal axonopathy probably caused by systemic disturbances generated in SIRS. Diagnosis is clinical and electrophysiology is invaluable in confirming peripheral weakness; however, it may not be able to distinguish between neuropathy and myopathy in the unresponsive patient.

Treatment is supportive with emphasis on prevention. Few specific treatments have proven benefit and mortality may be as high as 50–60%. CIM has a spectrum of pathologies but all cause a similar proximal and distal muscle weakness. Diagnosis is often difficult to make because interpretation of nerve conduction studies is complicated by the unresponsiveness of the patients and muscle biopsy may not show any changes. Direct stimulation of muscle fibres can help overcome some of these difficulties and elevated CK values are also suggestive. Treatment is again focused on prevention and physiological support with no specific therapies currently available.

Acknowledgements: We would like to thank Dr C Bolton, Dr B Maramattom and Dr E Wijidicks for allowing us to use some of the pictures. We would also like to thank the Medical Illustration Department at the John Radcliffe Hospital, Oxford.

CHOOSE THE BEST OF THE FIVE OPTIONS FOR EACH QUESTION (ANSWERS AFTER THE REFERENCES)

1. Critical illness polyneuropathy (CIP) is:

- A. Predominantly a type of motor neuropathy
- B. Primarily a distal axonopathy

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- C. Intravenous immunoglobulin is the treatment of choice
- D. Significant slowing of nerve conduction is the characteristic finding
- E. All of the above

2. In relation to critical illness myopathy (CIM) :

- A. It is a proximal myopathy
- B. Only type II muscle fibres are affected
- C. It is associated with high use of non-depolarising muscle relaxant
- D. Prednisolone is the treatment of choice
- E. Raised creatinine kinase (CK) is the hallmark of this disease

3. Which of the following is not a cause of newly acquired neuromuscular weakness in a patient admitted in intensive care?

- A. West Nile virus infection–acute flaccid paralysis
- B. Guillain–Barré syndrome
- C. Acute myopathy of intensive care
- D. Pyomyositis
- E. Neuromuscular blocking agent induced weakness

4. Which of the following is true for neuromuscular weakness in intensive care unit?

- A. Failure to wean from mechanical ventilation is often the initial presentation
- B. Electroneurography and electromyography (ENG–EMG) is the gold standard for diagnosing CIP
- C. Inflammatory changes are conspicuously absent in CIM
- D. Intensive insulin therapy in critical illness can reduce the incidence of CIP
- E. All of the above

Competing interests: None declared.

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Answers

1. B
2. C
3. B
4. E