Neuromuscular Complications of Critical Illness

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2013

Learning Objectives

- Become familiar with the Differential Diagnosis of weakness in the ICU
- Review some of the clinical, pathological and electrodiagnostic characteristics of neuropathy and myopathy in the ICU
- Review the electrodiagnostic approach of assessing weakness in the ICU setting
- Briefly, respectfully critique the confusing literature in CIM/CIPN

Critical Illness Neuromuscular Disorders

- Common
- Poorly understood
- Serious functional consequences

Neuromuscular Complications of Critical Illness

- **History**
  - Muscular wasting as a consequence of sepsis - **Sir William Osler - 1892**
  - Unexplained neuropathic complications in critically ill patients - **Olsen - 1956**
  - Coma-polyneuropathies - described by **Mertens - 1961**
  - Polyneuropathy in burn patients - **Henderson - 1971**
  - Polyneuropathy in septic patients - Bischoff - 1977
  - Development of the term *Critical Illness Polyneuropathy* – **1984 (Bolton)**
  - Polyneuropathy in a patient with an anoxic brain injury - **Erbsloh and Abel - 1989**
  - Polyneuropathy associated with the use of non-depolarizing NMBA
  - Myopathy in patients with asthma who were treated with NMBA and corticosteroids
  - CIP is the most common acute polyneuropathy in the critically ill patient -**2003**

Neuromuscular Complications of Critical Illness

Case: 51 year old male with COPD + respiratory failure

- h/o alcohol abuse and depression
- Became critically ill
- Placed on IV methylprednisolone for 27 days
  - Then to prednisone
- Developed weakness that included facial muscles
- Absent tendon reflexes, CSF normal

Scully RE, et.al., Case Records of the Massachusetts General Hospital Case 11-1997

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NCV Results

Nerve and Site of Stimulation	Latency	Amplitude	Duration	Velocity
	msec	μV	msec	m/sec
Motor nerve				
Median				
Wrist	3.5	1480	9.4	
Elbow	7.9	1430	10.3	52
Ulnar				
Wrist	2.0	1190	10.0	
Below elbow	5.2	1060	10.5	56
Above elbow	7.4	1130	10.6	68
Tibial				
Ankle	4.4	3120	6.8	
Knee	13.0	2960	7.1	44
Sensory nerve				
Median	2.3	12.4		57
Ulnar	2.0	7.0		50
Sural	2.8	7.3		46

Scully 1997

- 34th day post onset
- No conduction block
- Needle EMG
  - No fibs or PSW
  - MUPs ↑ polys
    - NI to low amp
    - Shorter duration
  - Recruitment WNL
- F-wave lat WNL
  - Low amp
- Rep stim WNL

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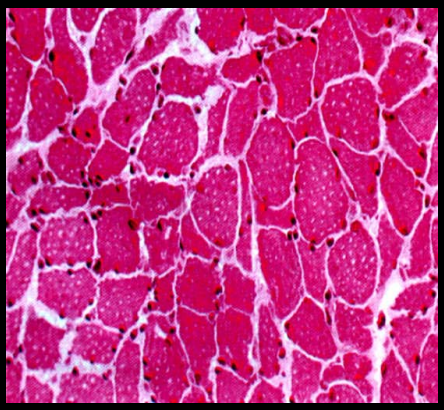
Muscle Biopsy

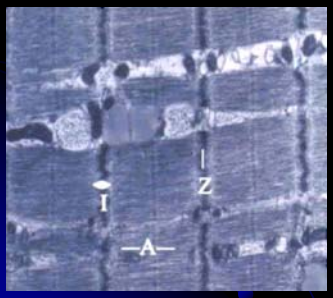
- Right deltoid
- Atrophy of both Type 1&2 fibers
- No inflammation
- No fiber-type grouping



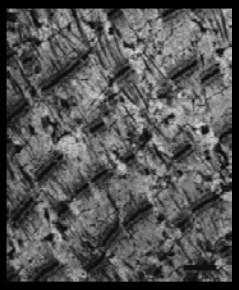
Muscle Biopsy

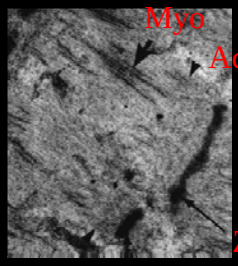
Electron microscopy

- Myofibrillar disruption
- Loss of myosin
- Normal Mitochondria



Normal





Prospective study of neuromuscular abnormalities in critical illness

- 23 patients, ICU stay > 7 days
- Neurophysiological abnormalities in all but 1 of 10 patients
  - 8 primary axonal neuropathy
  - 1 motor abnormalities only
- Abnormal muscle histology in 22 patients
  - diffuse atrophy 8 cases
  - type II fibre atrophy 2 cases
  - myopathy 8 cases (necrosis in 2)
  - neurogenic atrophy 4 cases

Coakley et al, 1993

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Patterns of neurophysiological abnormality in 44 long stay critically ill patients

Group	*n* (%)	CMAP	SAP	Muscle biopsy
I	7 (16)	Normal	Normal	Normal (2) Diffuse fibre atrophy (1)
II	4 (9)	Normal	Reduced	Diffuse fibre atrophy (2) Type II fibre atrophy (1)
III	11 (25)	Reduced	Normal	Diffuse fibre atrophy (3) Neurogenic atrophy (2) Myopathy (3)
IV	19 (43)	Reduced	Reduced	Diffuse fibre atrophy (6) Neurogenic atrophy (1) Myopathy (3)
V	3 (7)	Not classified	Not classified	Myopathy (2)

Data from Coakley *et al.* [28•]. CMAP, compound muscle action potential; SAP, sensory action potential.

Coakley et al, 1998

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Characteristics of 38 patients with and without critical illness polyneuropathy (CIP) on mechanical ventilation for more than 7 days

	NO CIP	CIP	p
Number	20	18	
Mean age (years)	54.7±17.3	58.5 ±17.0	0.49
Mean number of ventilation days	20.3 ±10.9	33.6 ±19.8	0.02
Mean APACHE II in first 24 h	23.1 ±6.8	21.9 ±9.1	0.66
Mean maximal Muti Organ Dysfunction Syndrome (MODS) score	3.6 ±1.5	5.3 ±1.8	0.003
Median number (range) of days to reach maximum MODS score	1 (1-2)	4.5 (2-9)	0.002
Median number (range) of different organs involved (max 6)	2 (1-4)	4 (3-5)	0.009
Sepsis syndrome	8 (40%)	10 (56%)	0.34
Number of deaths in the ICU	4 (20%)	8 (44%)	0.11

Leijten et al, Intensive Care Med 1996;22:856-861

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Persistent neuromuscular and neurophysiologic abnormalities in long-term survivors of prolonged critical illness*

Simon N. Fletcher, FRCA; Daniel D. Kennedy, FRCA; Indrajit R. Ghosh, MRCP; Vijay P. Misra, MRCP; Kevin Kiff, FRCA; John H. Coakley, MRCP; Charles J. Hinds, FRCP, FRCA

- In 21 of 22 patients remarkably consistent neurophysiological findings indicative of denervation and subsequent reinnervation up to **5 years after discharge.**
- 1 patient who had developed a necrotizing myopathy had no persisting weakness and no evidence of denervation.

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Neuromuscular Complications Diagnostic Approach

Think broadly!

- Long differential diagnosis, depending on the clinical context

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Neuromuscular Complications Diagnostic Approach

- **History**

M edication (intravenous administration of corticosteroids - panucuronium, vecuronium, metronidazole, amiodarone,

U ndiagnosed neuromuscular disorder (PM, DM, ALS, GBS, MG, LEMS, acid maltase deficiency, mitochondrial myopathy, musc. dystrophy)

S pinal cord damage (ischemic, compressive hematoma, trauma)

C ritical illness neuromuscular disorder

L oss of muscle mass (disuse atrophy, rhabdomyolysis, catabolic state)

E lectrolyte disorders (hypokalemia, hypermagnesemia, hypophosphatemia)

S ystemic illness (acute prophyria, AIDS, vasculitis neuropathy, endocrine myopathies)

(Wijdicks E.F.M., et.al, 1995)

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Diagnostic Approach

Examine the patient! Confirm weakness!

- Unexpected lack of ventilatory weaning
- Accelerated peripheral muscle atrophy
- Inability to hold head/limb off bed

Diagnostic Clues

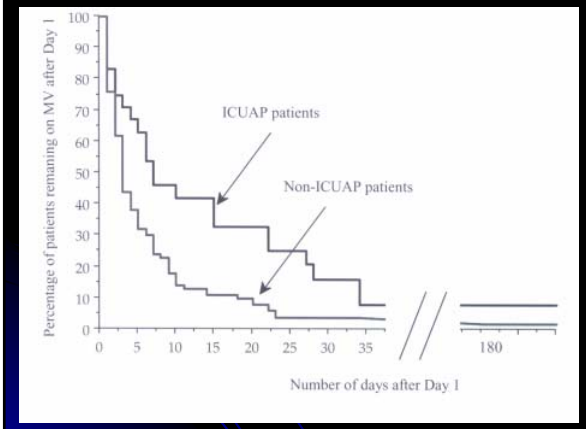
- Mental status - not affected
- Pattern of weakness
  - symmetric, facial sparing
  - CN weakness - think GBS, MG, stroke
- MSR - usually decreased
  - if increased, suggests central lesion

THE DEVELOPMENT OF CRITICAL ILLNESS RELATED WEAKNESS INFLUENCES SHORT TERM OUTCOME

Important to Know Because:

- Time on ventilator
- Time in intensive care
- Time in hospital
- Mortality
- Morbidity

ICU acquired paresis is an independent predictor of prolonged weaning



- **Multivariate analysis**
  ICUAP independent predictors of prolonged weaning

De Jonghe et al Intensive Care Med. 2004;30:1117-1121

Neuromuscular Complications of Critical Illness

SIRS

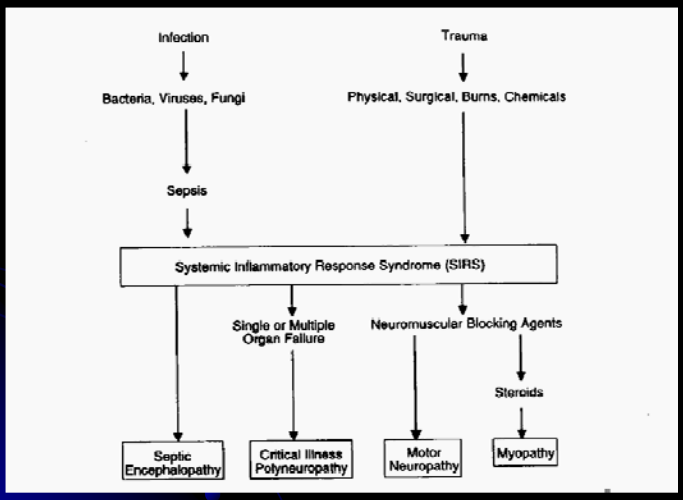
- Systemic Inflammatory Response Syndrome (***SIRS***) - The Society of Critical Care Medicine and The American College of Chest Physicians consensus conference 1992
- The severe systemic response that occurs in critically ill patients
- Etiology varied: infection (sepsis), trauma, burns etc.
- Septic Shock is a term reserved for individuals with organ dysfunction and hypoperfusion

Neuromuscular Complications of Critical Illness

Physiology of SIRS

- Activation of humoral responses primarily involving the cytokines (interleukins, tumor necrosis factor, arachidonic acid, free oxygen radicals and proteases)
- Activation of cellular responses primarily involving lymphocytes, monocytes and neutrophils

Neuromuscular Complications of Critical Illness



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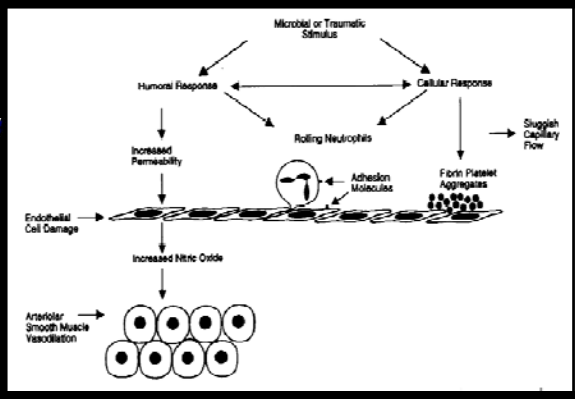
Neuromuscular Complications of Critical Illness

Physiology of SIRS

- Activation increases capillary permeability
- Inadequate nutrient delivery
- Oxygen debt within the tissue
- Vulnerable nervous system



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Myopathy in Critically Ill Individuals

Acute corticosteroid myopathy

Acute quadriplegic myopathy (AQM)

Acute myopathy in Status Asthmaticus

(Acute) (necrotizing) myopathy of intensive care

Critical care myopathy

ICU myopathy

Myopathy with thick filament (myosin) loss

Neuromuscular Complications of Critical Illness*

Neuropathy

- *Critical Illness Polyneuropathy*
- *Acute Motor Neuropathy Associated with Non-Depolarizing Neuromuscular Blocking Agents*

Critical Illness Myopathy

- Thick Filament Myopathy
- Acute Necrotizing Myopathy of Intensive Care
- Catabolic Myopathy

Neuromuscular Junction Abnormalities

Neuromyopathy

*Studies in the pediatric population find a similar pattern of distribution

Critical Illness Polyneuropathy

Neuromuscular Complications of Critical Illness

I. Neuropathy

Critical Illness Polyneuropathy

- Acute, diffuse, mainly motor neuropathy due to axonal dysfunction
- Patients with sepsis or multiple organ dysfunction
- Incidence in this patient population ranges from 50-75%
- Severity proportional to length of time in the ICU

Neuromuscular Complications of Critical Illness

I. Neuropathy

Critical Illness Polyneuropathy

- The earliest sign - difficulty weaning from the ventilator
- Can present with tetraplegia, absent deep tendon reflexes
- Blood CPK levels are normal

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Neuromuscular Complications of Critical Illness

I. Neuropathy

Critical Illness Polyneuropathy

- Septic Encephalopathy usually precedes the diagnosis of Critical Illness Polyneuropathy by two weeks
- Impaired attention, concentration, orientation and writing
- CSF studies are normal or show mild elevations in protein

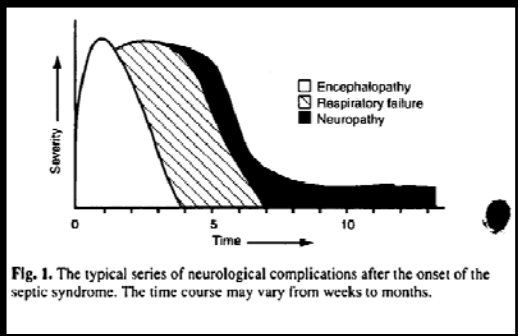

Fig. 1. The typical series of neurological complications after the onset of the septic syndrome. The time course may vary from weeks to months.

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Neuromuscular Complications of Critical Illness

I. Neuropathy

Critical Illness Polyneuropathy

- Development associated with hyperglycemia
- Development associated with hypoalbuminemia
- Neuropathy occurs more often in patients getting parenteral nutrition

Neuromuscular Complications of Critical Illness

I. Neuropathy

Critical Illness Polyneuropathy

- Mechanisms proposed for nutritional axonal injury are:
  - Glucose-induced depletion of intracellular phosphate stores with subsequent depletion of high-energy phosphate compounds
  - Oxidative modification of dietary lipid

Neuromuscular Complications of Critical Illness

I. Neuropathy

Critical Illness Polyneuropathy

- Prognosis depends on the underlying disease process
- Recovery usually occurs over weeks to months depending neuropathy severity

Neuromuscular Complications of Critical Illness

I. Neuropathy

Electrodiagnostic Findings in Critical Illness Polyneuropathy

- Nerve conduction velocities and distal latencies are normal
- Reduction in the CMAP and SNAP amplitude
- No evidence of conduction block
- Repetitive nerve stimulation does not show a defect

Neuromuscular Complications of Critical Illness

I. Neuropathy

Critical Illness Polyneuropathy

- **Severe**: absent SNAP, fibrillation potentials in all muscle groups, and multiple CMAP amplitudes less than 1mv amplitude
- **Moderate**: SNAP amplitudes <5uv, multiple CMAP amplitudes between 1 and 3 mV, fibrillation potentials and positive sharp waves in distal muscles and occasionally present in more proximal muscles
- **Mild**: SNAP amplitudes >5uv, CMAP amplitudes >3mv, occasional positive sharp waves in distal muscles

Spitzer AR, Giancarlo T, Maher L, et al. 1992

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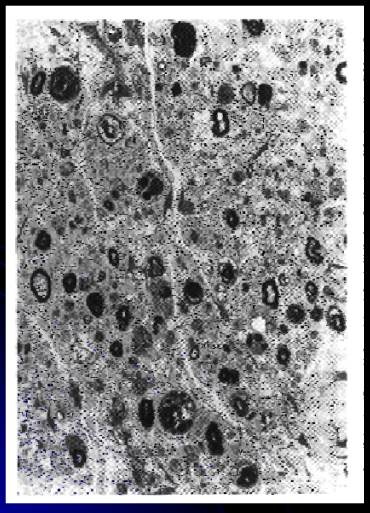
Neuromuscular Complications of Critical Illness

I. Neuropathy



Nerve Biopsy

- Severe primary axonal degeneration of motor and sensory fibers
- Affects distal segments primarily
- No evidence of inflammation

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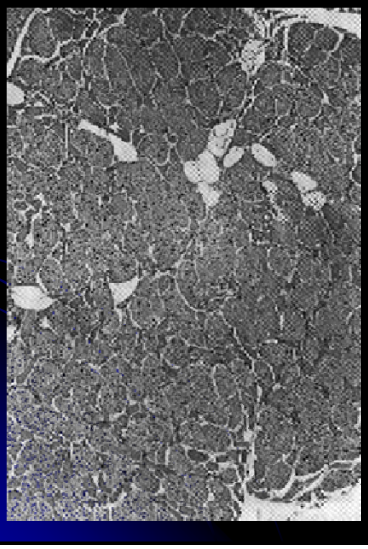
Neuromuscular Complications of Critical Illness

I. Neuropathy



Muscle Biopsy

- Scattered atrophic fibers in the acute phase

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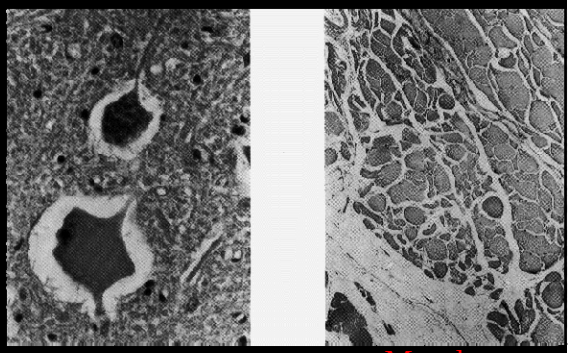
Neuromuscular Complications of Critical Illness

I. Neuropathy

BIOPSY

- Grouped fiber atrophy in the chronic phase
- Autopsy studies also may reveal central chromatolysis of anterior horn cells



Spinal Cord Muscle

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Acute Motor Neuropathy Associated with Non-Depolarizing Neuromuscular Blocking Agents

Neuromuscular Complications of Critical Illness

I. Neuropathy

Acute Motor Neuropathy Associated with Non-Depolarizing Neuromuscular Blocking Agents

- Associated with the administration of pancuronium or vecuronium
- 3-hydroxy metabolites of pancuronium(3-desacetylpancuronium) and vecuronium (3-desacetylvecuronium) retain neuromuscular blocking activity and accumulate in the body with prolonged use

Neuromuscular Complications of Critical Illness

I. Neuropathy

Acute Motor Neuropathy Associated with Non-Depolarizing Neuromuscular Blocking Agents

- Occurs more readily in patients with renal or hepatobiliary disease
- Neuropathy occurs as a result of a toxic effect of the non-depolarizing agent in a patient with SIRS
- Neuropathy is more likely the longer these agents are used

Neuromuscular Complications of Critical Illness

I. Neuropathy

Acute Motor Neuropathy Associated with Non-Depolarizing Neuromuscular Blocking Agents

- Significant increase in the incidence when these agents are used for >48 hours
- **Presents as difficulty weaning from the ventilator and limb weakness**

Neuromuscular Complications of Critical Illness

I. Neuropathy

Electrodiagnostic Findings in Acute Motor Neuropathy Associated with Non-Depolarizing Neuromuscular Blocking Agents

- Primary axonal degeneration of mostly motor fibers

Neuromuscular Complications of Critical Illness

I. Neuropathy

Muscle Biopsy Finding in Acute Motor Neuropathy Associated with Non-Depolarizing Neuromuscular Blocking Agents

- Varying degrees of denervation, atrophy, and muscle necrosis

Thick Filament Myopathy

Neuromuscular Complications of Critical Illness

II. Myopathy

Thick Filament Myopathy

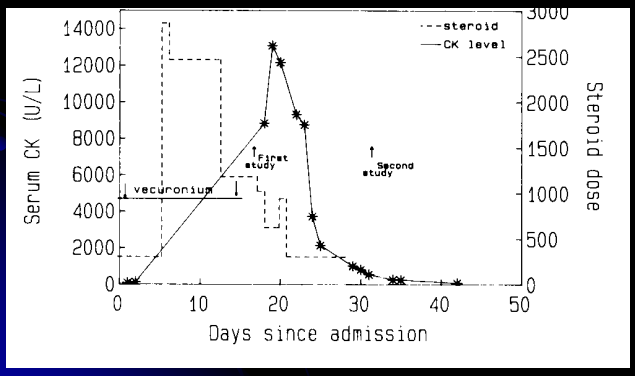
- Occurs in patients with a sudden severe asthma exacerbation
- Patients who receive NMBA and high dose corticosteroids
- Incidence associated with the length of chemical paralysis

Neuromuscular Complications of Critical Illness

II. Myopathy

Thick Filament Myopathy

- Douglass found an elevated CPK in 76% of patients ventilated with severe asthma



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Neuromuscular Complications of Critical Illness

II. Myopathy

Thick Filament Myopathy

- Douglass found an elevated CPK in 76% of patients ventilated with severe asthma
- 36% of these developed a symptomatic myopathy
- Dubois and Almon found the number of glucocorticoid receptors increase to three times normal after surgical interruption of nerve supply
- Suggests that a denervated muscle may be hypersensitive to treatment with corticosteroids

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Neuromuscular Complications of Critical Illness

II. Myopathy

Thick Filament Myopathy

- Patients present with failure to wean and flaccid limb weakness of distal and proximal muscles
- Facial muscles are often affected
- Peripheral sensation is normal
- CPK levels are significantly increased
- Recovery is usually rapid

Neuromuscular Complications of Critical Illness

II. Myopathy

Electrodiagnostic Findings in Thick Filament Myopathy

- Motor nerve conduction velocities are usually normal
- Low amplitude CMAP
- Sensory studies are normal
- Repetitive nerve stimulation is normal
- Needle EMG reveals motor unit action potentials that are of short duration, low amplitude and polyphasic

Neuromuscular Complications of Critical Illness

II. Myopathy

Muscle Biopsy Finding in Thick Filament Myopathy

- Loss of structure centrally that is a result of destruction of the thick myosin filaments
- Pattern of atrophy, necrosis, and regeneration of type I and II muscle fibers
- Little or no associated inflammation

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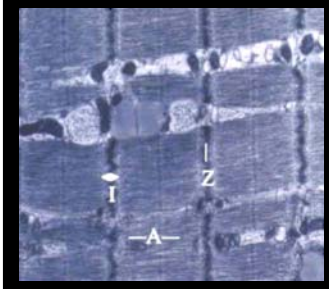
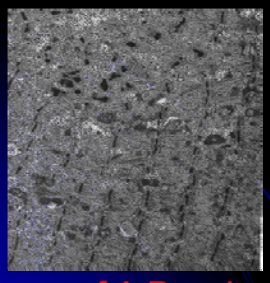
Neuromuscular Complications of Critical Illness

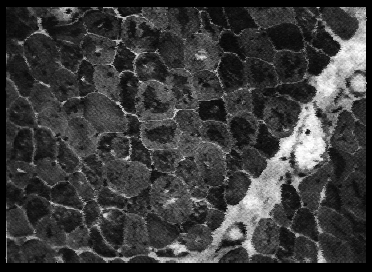
II. Myopathy



Normal



Loss of A Bands (EM)



Central necrosis and atrophy

Enhanced calcium-induced proteolysis

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Acute Necrotizing Myopathy of Intensive Care

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Neuromuscular Complications of Critical Illness

II. Myopathy

Acute Necrotizing Myopathy of Intensive Care

- Occurs in the presence of overwhelming infections, with viruses, Escherichia coli, Leptospirosis, Legionnaires disease, and organisms that cause toxic shock syndrome
- Presents with severe weakness and myalgias, high concentrations of CPK and myoglobinuria

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Neuromuscular Complications of Critical Illness

II. Myopathy

Acute Necrotizing Myopathy of Intensive Care

- Electrodiagnosis is consistent with a severe myopathy
- Muscle biopsy shows severe widespread muscle fiber necrosis
- Prognosis correlates with the severity of the myopathy

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Catabolic Myopathy

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Neuromuscular Complications of Critical Illness

II. Myopathy

Catabolic Myopathy

- Ill-defined
- Believed to result from the action of Interleukin-1 and tumor necrosis factor
- Defects in high-energy metabolites in patients with respiratory failure, cardiogenic shock, severe congestive heart failure, and sepsis

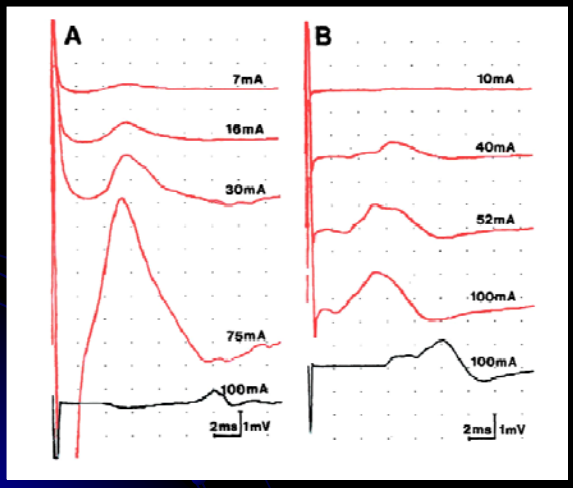
Neuromuscular Complications of Critical Illness

Direct Muscle Stimulation

- Direct Muscle Stimulation can help in distinguishing myopathy from peripheral neuropathy in the critically ill.
- In CIP the denervated muscles should retain electrical excitability and direct muscle stimulated CMAP should be normal. In Critical Illness Myopathy the muscle fibers lose excitability and thus the direct muscle and nerve stimulated CMAP are reduced

Neuromuscular Complications of Critical Illness

Direct Muscle Stimulation



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Neuromuscular Junction Abnormalities

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Neuromuscular Complications of Critical Illness

III. Neuromuscular Junction Abnormalities

- Transient persistent neuromuscular blockade occurs with the use of non-depolarizing NMBA long after the medications are discontinued
- More likely to occur in patients with renal or liver disease
- Repetitive Nerve Stimulation reveals a neuromuscular transmission defect (decremental response)

Neuromuscular Complications of Critical Illness

III. Neuromuscular Junction Abnormalities

- Patients present with failure to wean from the ventilator and flaccid limbs
- Recovery over the course of several weeks or months
- Segredo et. al. **associated metabolic acidosis, elevated plasma concentrations of magnesium and female gender as variables with the presence of prolonged neuromuscular blockade**
- Vecuronium total dose, rate of administration and duration of treatment did not correlate with the presence of prolonged neuromuscular blockade

Neuromyopathy

Neuromuscular Complications of Critical Illness

III. Neuromyopathy

- Refers to the coexistence of neuropathy and myopathy in patients with critical illness
- Critical Illness Polyneuropathy and Acute Necrotizing Myopathy of Intensive Care
- Incidence = 6% of patients with neurologic illness secondary to critical illness
- Nates felt that the presence of one neuromuscular complication of critical illness may provide an environment in which the second process is encouraged

Neuromuscular Complications of Critical Illness

Management

- Prompt and effective treatment of underlying disease, in particular sepsis, and prevention of MODS
- "Tight glycemic control" (reduced incidence CIP from 52% to 29% !)
- Avoid contributory factors
  - steroids
  - NMBAs
  - Hypoalbuminaemia
- Involve Rehabilitation Team

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Critical Illness Polyneuropathy: A 2-Year Follow-Up Study in 19 Severe Cases

Case	Day	Sensory findings	Follow up
1	12	Distal sensory loss	2 years:paraparesis
2	10	n.a.	Complete recovery within 3 m
3	30	Distal sensory loss	2 years:quadriplegia
4	20	Distal sensory loss	Death at day 65 without recovery
5	20	Normal	Complete recovery within 3 m
6	13	Distal sensory loss	Complete recovery within 1y
7	7	n.a.	Complete recovery within 3m
8	30	Distal sensory loss	2 years after: quadriplegia
9	15	Distal sensory loss	Complete recovery within 6 months
10	29	Distal sensory loss	Death at day 168

Seze et al, Eur Neurol 2000;43:61-69

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Critical Illness Polyneuropathy: A 2-Year Follow-Up Study in 19 **Severe** Cases

Case	Day	Sensory findings	Follow up
11	33	Normal	Complete recovery within 3 m
12	19	Normal	Complete recovery within 6 m
13	23	Distal loss	Complete recovery within 1 y
14	30	Distal sensory loss	Complete recovery within 6 m
15	31	n.a.	Complete recovery within 6 m
16	120	Normal	Complete recovery within 1 y
17	10	Distal sensory loss	2 years: quadriparesis
18	29	n.a.	Death at day 72 without recovery
19	25	Distal sensory loss	Death at day 348

Seze et al, Eur Neurol 2000;43:61-69

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Persistent neuromuscular and neurophysiologic abnormalities in long-term survivors of prolonged critical illness*

Simon N. Fletcher, FRCA; Daniel D. Kennedy, FRCA; Indrajit R. Ghosh, MRCP; Vijay P. Misra, MRCP; Kevin Kiff, FRCA; John H. Coakley, MRCP; Charles J. Hinds, FRCP, FRCA

- All 22 patients gave a clear history of prolonged weakness, fatigue and difficulty with mobility
- In some patients weakness was still clinically evident up to 4 yrs after discharge and in 1 was severe

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- Neurological examination
  - sensory deficits in 27%
  - motor weakness in 18%
  - sensory and motor deficits in 14%
  - bilateral peroneal nerve palsies in 2 patients
  - bilateral upper limb weakness in 3 patients

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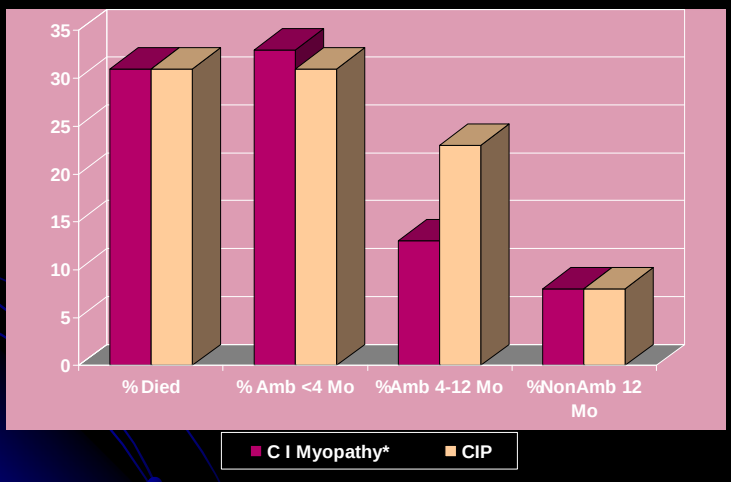
Outcomes

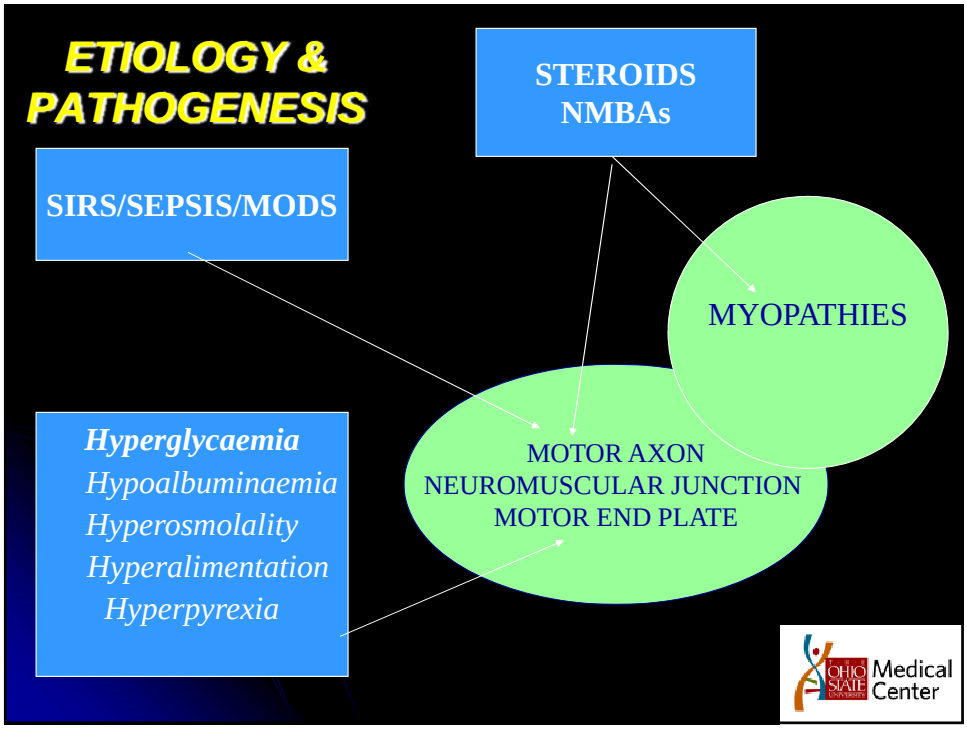


General Outcome Concepts

- Severe weakness, prolonged recovery and residual clinical motor and sensory neurological deficits are extremely common in survivors of protracted critical illness
- In those with mild or moderate CIP recovery is often relatively rapid and complete (weeks or months)

General Outcome Concepts

- In some with more severe ICUAP recovery is delayed for years
- Quality of life is often impaired
- In a few persistent, severe neuromuscular weakness may contribute to late deaths.



Much of Newer Literature Suggests Myopathy More Common

- Acute myopathy 3x as common as polyneuropathy --Lacomis M&N May 1998.

Summary of Literature Review and Clinical Experience

- There is vast disparity in the medical literature.
- Much of the early literature appears unsatisfactory in adequately describing the conditions.
- The vast majority, at our institution, have a primarily myopathic influence.

Deficiencies in the Early Literature Include:

- Initially studied primarily severely tetraparetic patients, resulting in little motor unit evaluation
- Only sporadic use of nerve and muscle biopsies
- No use of direct muscle stimulation
- Over-interpreted absent F-waves

Complicating Factors in ICU Assessment

- Poor mobility
- Frequent cognitive impairments and sedation
- Frequent limb edema
- Often co-morbid factors (eg diabetes mellitus, CRF)

Myopathy not axonopathy

- Described as a “motor predominant” axonopathy because it’s a myopathy!
- Rarely sensory symptoms or signs in communicative patients
- Virtually never neuropathic pain
- Little to no membrane instability

Myopathy not Axonopathy

- Many short duration, small amplitude polyphasic MUAPs
- “Early” Recruitment!
- Little to no improvement with direct muscle stimulation
- Involves proximal and distal muscles equally

Myopathy not Axonopathy

- Recovery is generally too rapid to be attributed to re-innervation of catastrophic axonal loss.

Many Cases Appear to be "Membranopathies"

- Normal histology and rapid recovery
- Presumption: The muscle membrane becomes dysfunctional, inexcitable and leaky
- Resultant loss of myoglobin

CIM

- Others cases similar to disuse atrophy with loss of type II fibers
- Loss of thick myosin filaments more frequent seen in patients treated with corticosteroids and NMJ blocking agents
- Necrotic/muscle necrosis less common

Combination of Neuropathy and Myopathy is not Uncommon

My Opinion

- Electrodiagnosis remains the most practical and comprehensive testing for ICU weakness

Electrodiagnostic Approach

Need to assess for neuropathy, myopathy, primary NMJ disorder, motor neuron disease, central weakness

Electrodiagnostic Approach

- Neuropathy: CIP, DM, Other pre-morbid, AIDP, CIDP, MMN
- Myopathy: CIM, PM, other inflammatory, congenital
- NMJ: MG, MS, Botulinum
- MND
- Central: CVA, Cervical myelopathy
- Co-morbid conditions

Electrodiagnostic Approach

- Motor NCS with F-waves
- Include phrenic studies if indicated
- Sensory studies
- Repetitive Stimulation including fast stimulation or (preferably) post-exercise
- Needle EMG
- If needed, direct muscle stimulation

Note: Will generally sample at least three limbs of the above

Summary

- The constellation of neuromuscular abnormalities in ICU weakness can be complex
- Caution should be used with earlier literature (ie before 1997) or more recent reviews that rely on earlier literature
- Additional studies are needed to further qualify and understand these conditions

Summary

- Comprehensive clinical and electrodiagnostic evaluation can provide an effective evaluation of generalized weakness in the ICU.