

EXTRACORPOREAL NEPHROLOGY GROUP [ECNG]

RHABDOMYOLYSIS

DR VILESH VALSALAN

CONSULTANT NEPHROLOGIST AND TRANSPLANT PHYSICIAN

ACADEMIC CORDINATOR - ECNG

TERMINOLOGY

Myopathy	General term referring to any disease of muscles Can be acquired or inherited and can occur at birth or later in life
Myalgia	Muscle ache or weakness without creatine kinase elevation
Myositis	Muscle symptoms with creatine kinase elevation
Rhabdomyolysis	Muscle symptoms with marked creatine kinase elevation, typically substantially >11 times the upper limit of normal, a creatinine elevation consistent with pigment nephropathy, and usually with brown urine with myoglobinuria

Reproduced with permission from Elsevier.¹²

DRUGS - RHABDOMYOLYSIS

Statins	Other Antilipid Agents	Psychiatric Agents	Abused Substances	Antihistamines	Other
Lovastatin	Ezetimibe	Amitriptyline	Alcohol	Diphenhydramine	Amphotericin B
Pravastatin	Bezafibrate	Amoxapine	Cocaine	Doxylamine	Arsenic
Simvastatin	Clozafibrate	Doxepin	Heroin/Opiates		Azathioprine
Fluvastatin	Ciprofibrate	Fluoxetine	Amphetamines		Carbon monoxide
Atorvastatin	Clofibrate	Fluphenazine	Methamphetamines		Halothane
Rosuvastatin	Gemfibrozil	Haloperidol	Lysergic acid diethylamide		Naltrexone
		Lithium	Phencyclidine		Quinidine
		Protriptyline			Penicillamine
		Perphenazine			Pentamidine
		Promethazine			Propofol
		Chlorpromazine			Salicylates
		Trifluoperazine			Succinylcholine
		Venlafaxine			Theophylline
		Benzodiazepines			Terbutaline
		Barbiturates			Thiazides
					Vasopressin

Causes of Rhabdomyolysis

Acquired

Drugs / Toxins

Ethanol

Different mechanisms: e.g. Crush Injury; Ischaemia; myopathy; sarcolemmal damage

Infectious / Sepsis

Extreme Physical Exertion

Crush Injury / Compartment Syndrome

Ischaemia

Metabolic Disturbance

Primary Neurological disorders

e.g. status epilepticus, status dystonicus

Idiopathic

Genetic

Metabolic Muscle Disorders

Disorders of fatty acid metabolism (VLCAD deficiency, CPTII deficiency, glutaric aciduria type II, MAD deficiency) and disorders of glycogen metabolism (GSD type V, VII, IX, X, XI, XII, XIII, XIV)

Mitochondrial Disorders

Complex I, complex II, cytochrome *b* (complex III), complex IV, tRNA mutations: 3243A>G/T in tRNA^{Leu}; 4298G>A in tRNA^{Ile}; m.4281A>G in tRNA^{Ile}), and *DGUOK*

Disorders of intramuscular calcium release and excitation-contraction coupling:

RYR1

Miscellaneous

SIL1, TSEN54

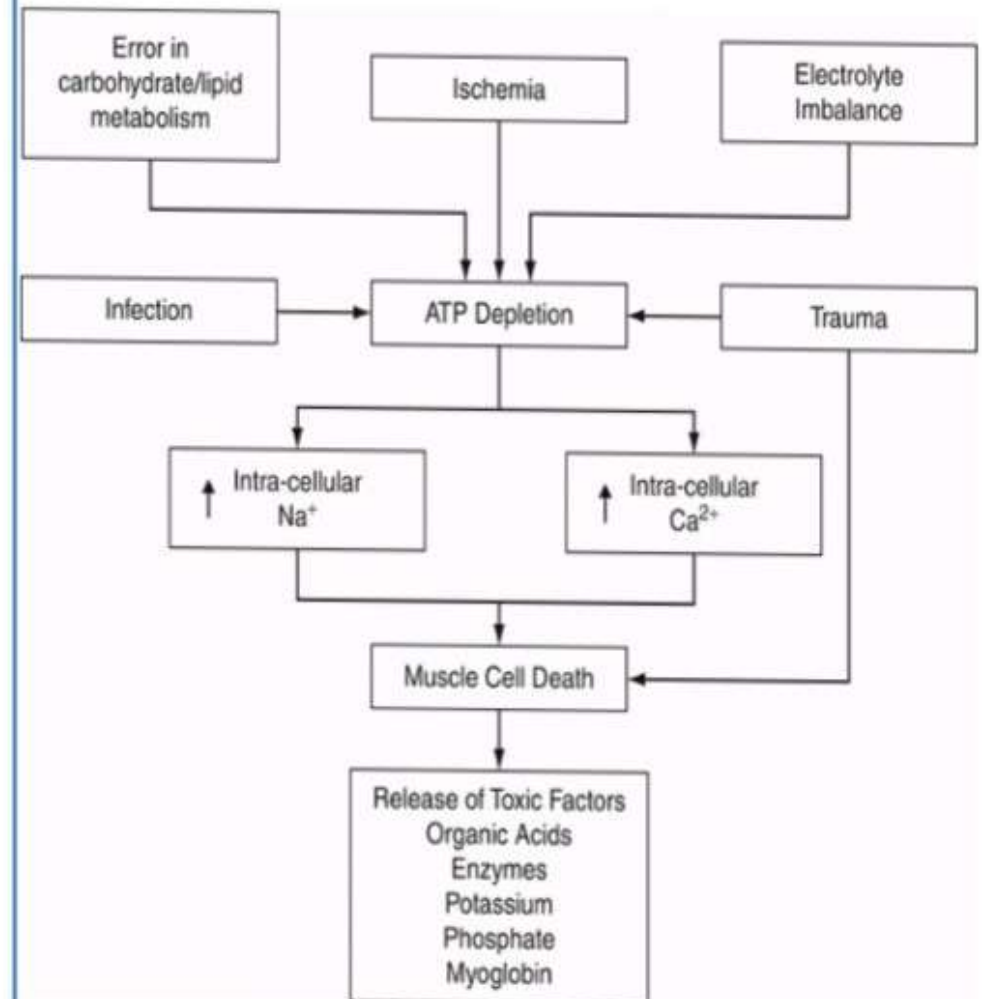
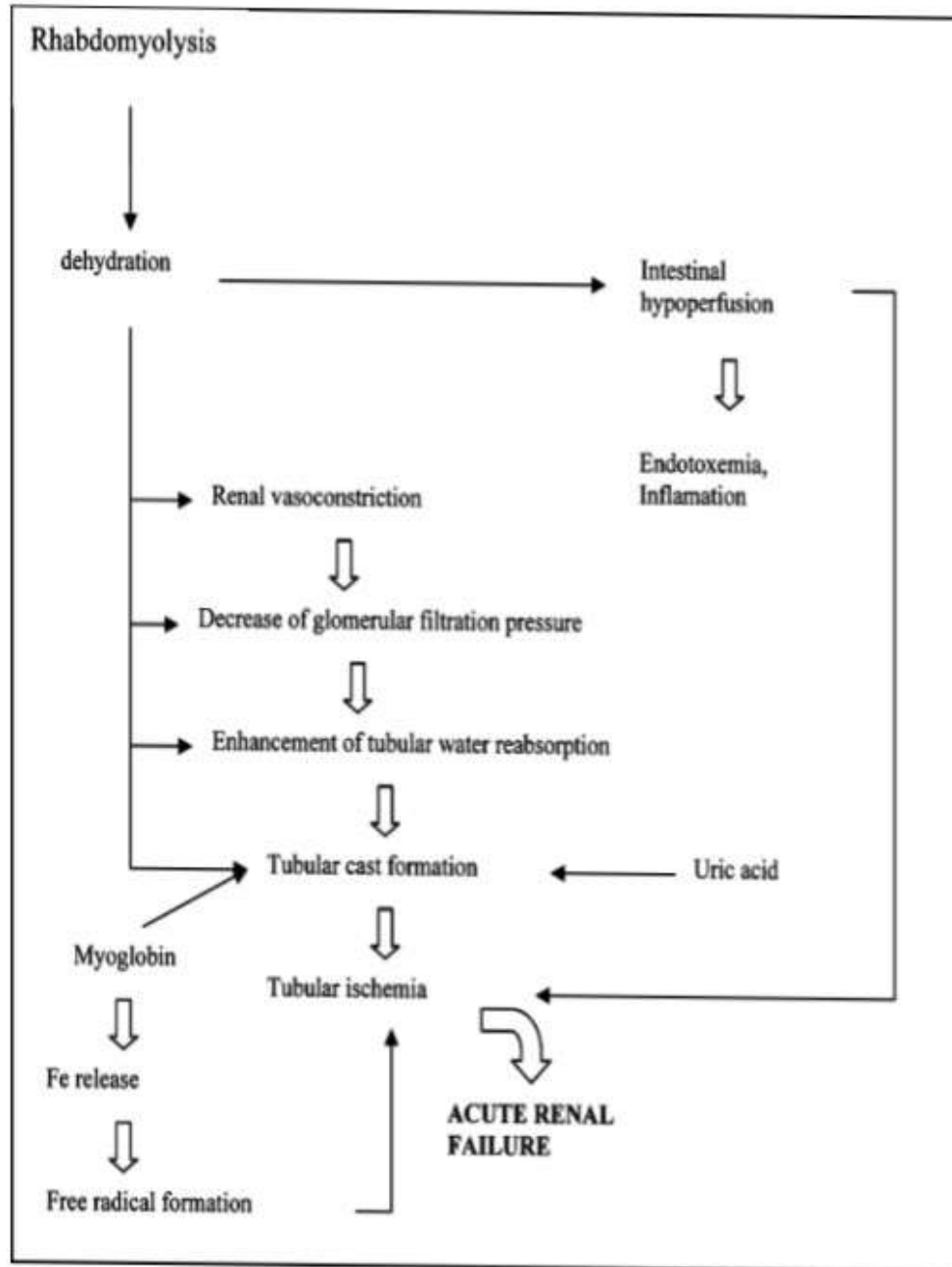
Muscular Dystrophies

Dystrophinopathies (DMD/BMD), *ANOS* and LGMD (*DYSF, FKRP*), alpha-sarcoglycanopathy

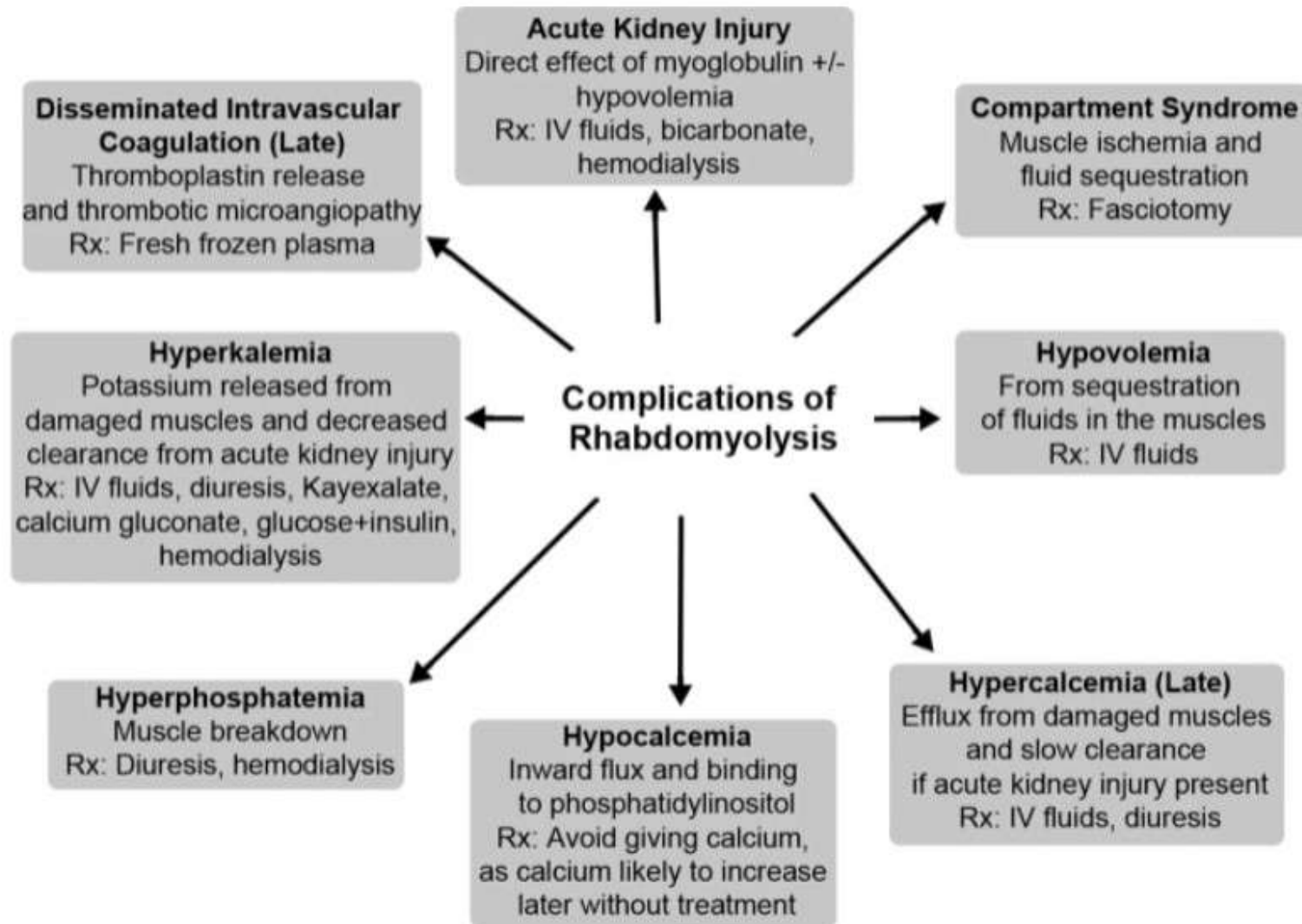
Anaesthesia

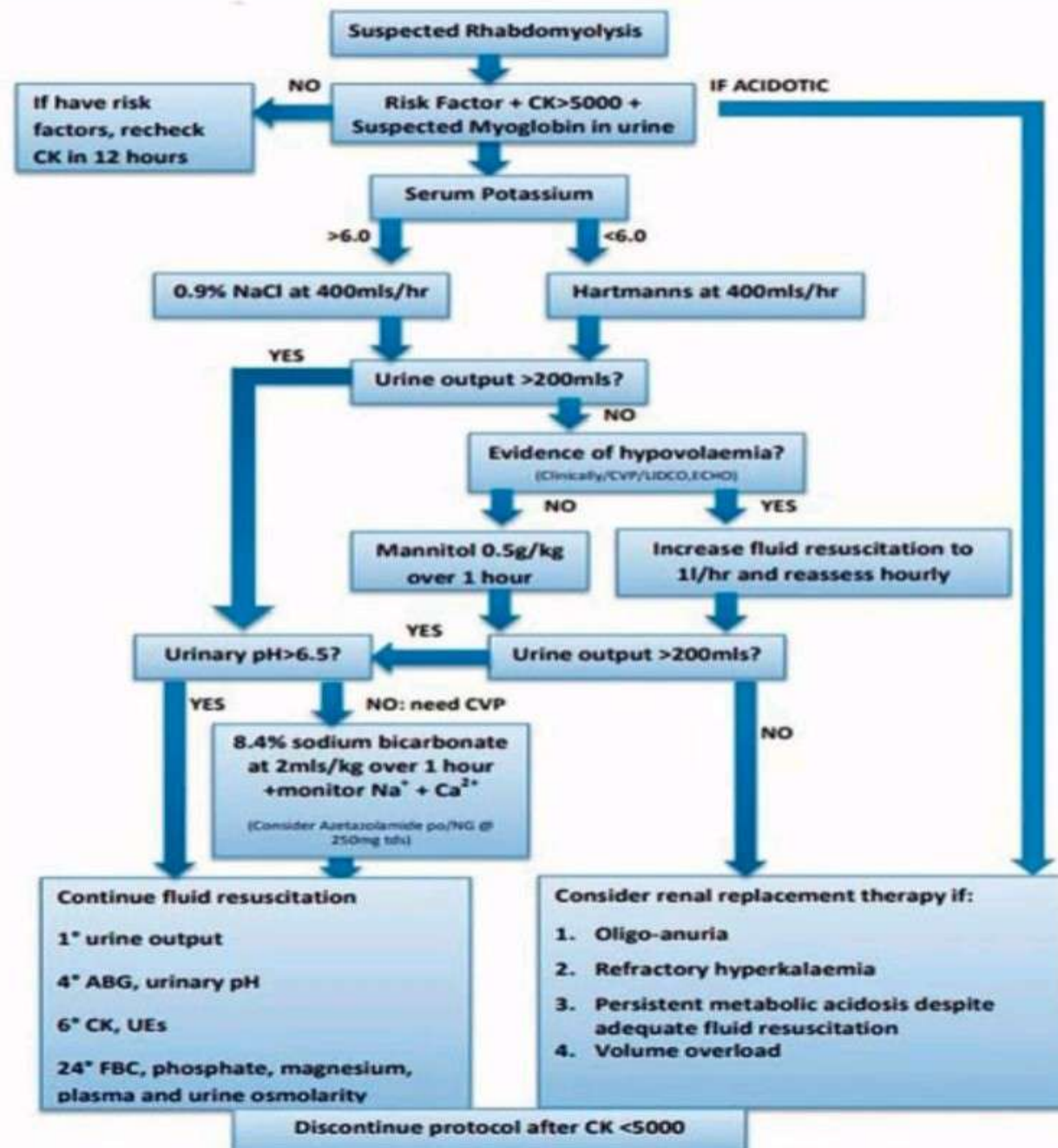
Malignant hyperthermia
Muscular dystrophies
Muscle channelopathies
Propofol Infusion Syndrome

PATHOPHYSIOLOGY



COMPLICATIONS





MANAGEMENT

TREATMENT

- **1.Fluid Resuscitation** (give IV fluids: either isotonic saline or RL) and Monitor I&O's). A good starting point is 400ml/hr.
- **2.Electrolyte Management:** Check K, Ca, Phos. Also regularly check ECG.
- **3.Aldinize urine .**
- **4.Treat pain** with non-nephrotoxic agents. Discontinue all nephrotoxic or myotoxic medications.
- **5.Consider complications** such as compartment syndrome.
- If **McMohan Score** is high, but CK is low, this does NOT rule out Rhabdomyolysis. This may be a sign of an early disease course.
- If McMohan score <3, CK<5,000, no AKI, no electrolyte derangement with a normal EKG, and a cause has been identified and stopped, the patient is likely safe for discharge.

Table 11–2. McMahon risk prediction score.

Variable	Score
Age	
>50 to ≤70 years	1.5
>70 to ≤80 years	2.5
≥80 years	3.0
Female sex	1
Initial creatinine	
1.4–2.2 mg/dL	1.5
>2.2 mg/dL	3.0
Initial calcium <7.5 mg/dL	2.0
Initial CK >40,000 U/L	2.0
Initial phosphate	
4–5.4 mg/dL	1.5
>5.4 mg/dL	2.0
Underlying cause other than seizures, syncope, exercise, statins, or myositis	3.0
Initial serum bicarbonate <19 mEq/L	2.0

**A SCORE MORE THAN 6
INCREASES RISK OF AKI.**

**RISK INCREASES BY 59 %
IF SCORE MORE THAN 10.**