Delirium tremens jako příčina srdeční zástavy?

Renata Černá Pařízková

Klinika anesteziologie, resuscitace a intenzivní medicíny
Univerzita Karlova v Praze, Lékařská fakulta v Hradci Králové
Fakultní nemocnice Hradec Králové

Obsah

- Delirium tremens
- Kazuistika
- Křečové stavy jako příčina srdeční zástavy?
- Poučení

Delirium tremens

- **Delirium tremens**
  - „třesoucí šílenství“, „šílenství s třesem“
  - alkoholové delirium, alkoholická psychoza
  - život ohrožující stav, který vzniká u osob s chronickou závislostí na alkoholu **při náhlém přerušení jeho užívání**
  - nejtěžší stádium abstinenčního syndromu
  - úmrtnost 5–15% (před JIP 35%)

Kazuistika

Anamnéza

- **Muž 30 let, dosud neléčen**

- **Dovezen RZP v doprovodu městské policie**
  - Den před přijetím konflikty na nádraží, krádež piva
  - V den přijetí – přišel do restaurace s dřevěnou holí, agresivní chování (ohrožování, napadání)
  - Testy na alkohol a drogy negativní
  - Omezovací prostředky

Kazuistika

Psychiatrická klinika

- **Dezorientace, agresivita**
- **Halucinatorní prožitky, v čekárně jí papír**
- **Masivně zpocený, mírný třes HKK a trupu**
- **Validní informace nelze**
  - Jazyková bariéra
  - Vulgarismy, neologizmy

Delirium tremens při odnětí alkoholu vs.

Kazuistika

Psychiatrická klinika

- **Terapie – diazepam 10 mg iv.**

- **Ca, Mg, P, U, kys. močová, bilir, ALP, amyláza, CRP**
- **Cholesterol, HDLC, LDLC, TAG, TP, albumin**
- **Na 138 mmol/l, K 3,5 mmol/l, krea 135 umol/l**
- **ALT 1,94, AST 4,21, GMT 2,5**

- **Toxikologický screening**

Kazuistika

Psychiatrická klinika

- **Těžký delirantní stav**
- **Verbální i fyzická agresivita**
- **Fyzické omezení na lůžku (02:00)**

- **Terapie**
  - Heminevrin 2 cps - 01:00, 04:00
  - Rivotril 1 mg im. – 03:50, 2x 4:50
  - Haloperidol inj im. – 02:50, 3:50

Kazuistika

Psychiatrická klinika

- **TK 110/90 mmHg, TF 100-140/min, ordinace á 1h**
- **F 1/1 500 ml + 2 amp KCl 7,5% na 5 hod**

- **Medikace a monitorace zrušeny pro neklid**

- **3:50 neklidný, plive po personálu**

- **4:50 agresivní, neusměřitelný**

Kazuistika

Psychiatrická klinika

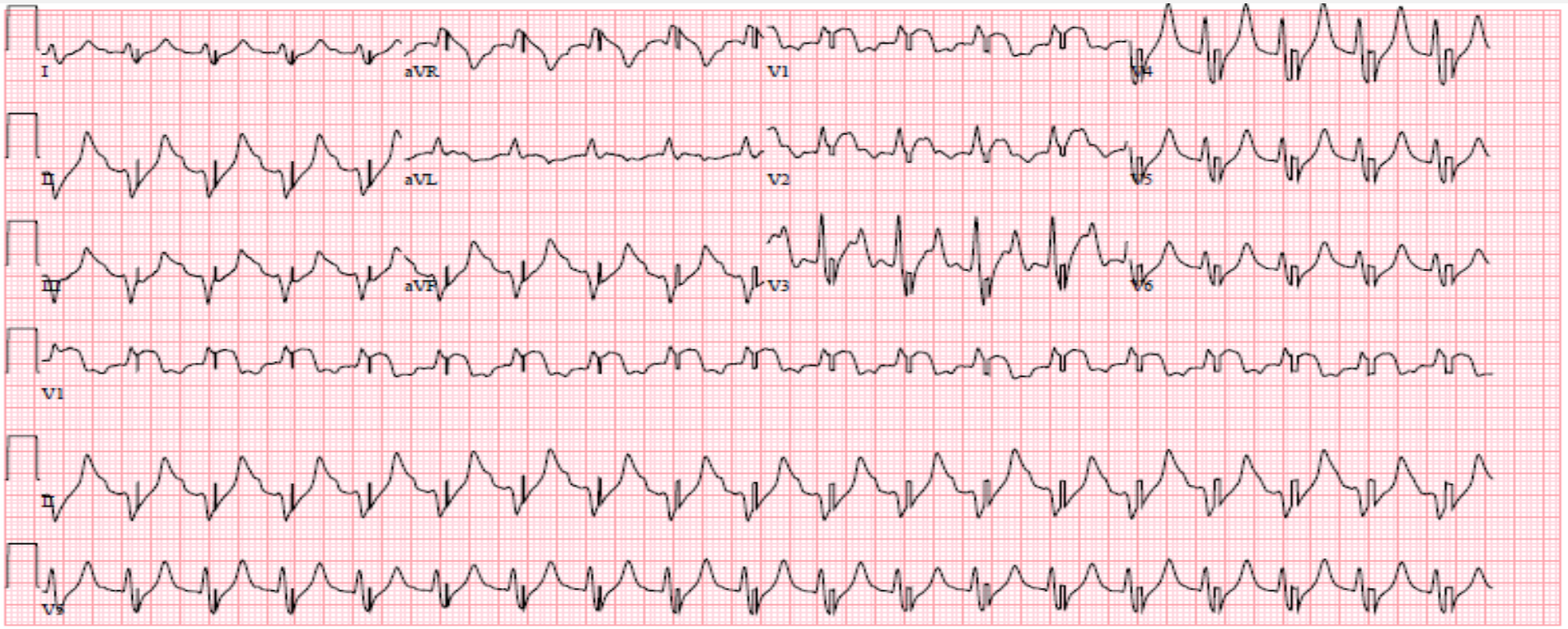
- **09:30**
  - Delirium, nezvladatelný stav
  - „Neurovegetativní doprovod“
  - Interní konzílium – z důvodu překladu na JIP

- **10:00**
  - Srdeční zástava (asystolie), KPR
  - Překlad ad LO KARIM

Kazuistika

KARIM

- **10:40**
  - GCS 3, mydriáza
  - Noradrenalin 1…2u/kg/min
  - Echo srdce bedside – PK norma, hypovolémie
  - Zajištění pacienta, volumoterapie, odběry


I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
II
V5

Kazuistika

KARIM

Hodnoty	Psychiatrie	KARIM
Na^+	138	140
K^+	3,5	8,5
P	1,03	5,4
Ca ion		1,01
Glykémie	7,6	3,3
Kreatinin	135	272
Myoglobin	---	7203 > 30 000 (za 60 min)
CKMB	---	74 > 122 (za 60 min)
Laktát	---	10
pH	---	6,9
BE	---	-17

Kazuistika

KARIM

Hodnoty	Psychiatrie	KARIM
Na^+	138	140
K^+	3,5	8,5
Laktát	---	10
pH	---	6,9
BE	---	-17

Metabolická acidóza

Rhabdomyolýza (hyperkalémie)

Kazuistika

KARIM

- **12:00**
  - Srdeční zástava, PEA? KPR
  - Výsledky – hyperkalémie
  - EKG – konzultace kardiologa – konzervativní postup
  - CT hrudníku a mozku
    - ✓ vyloučena PE
    - ✓ tenzní PNO (hrudní drén)
  - CRRT

Kazuistika

KARIM

- **17:00**
  - Trvalá progrese šoku a MOF
  - Dif dg. srdeční zástavy (4H, 4T)
    - ✓ Hyperkalémie při rhabdomyolýze
    - ✓ PNO sekundárně (při KPR?), další drény
- **20:00**
  - Irreverzibilní kardiogenní šok, MOF, paliativní péče
- **21:55 -** exitus letalis

Diskuze

Abuzus alkoholu

- **Chronický abúzus alkoholu**
  - 18% všeobecné populace
  - 20% u pacientů na urgentních příjmech
  - 8-40% akutní delírium na chirurgických JIP

Whiteman PJ, Hoffman RS, Goldfrank LR. Alcoholism in the emergency department: an epidemiologic study. Acad Emerg Med 2000; 7(1): 14–20.
Awissi DK, Lebrun G, Fagnan M et al. Alcohol, nicotine, and iatrogenic withdrawals in the ICU. Crit Care Med 2013; 41(9 Suppl 1): S57-S68. (http://dx.doi.org/10.1097/CCM.0b013e3182a16919)

Diskuze

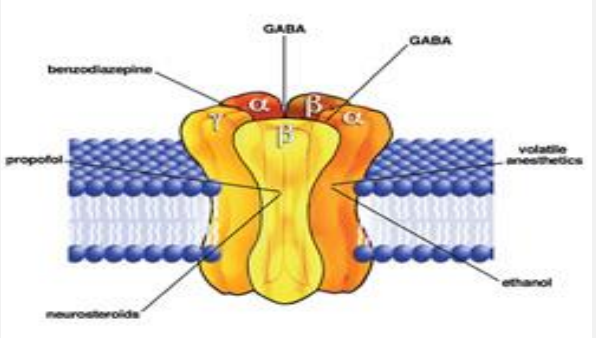
Delirium tremens

- **Delirium tremens**
  - Těžká alterace mentálního stavu
  - Sympatomimetická hyperadrenergní aktivita
  - Vysoká mortalita
  - Časná diagnostika a specifická léčba!!!

Diskuze

Delirium tremens

- **Chronický abuzus a neurotransmitery**
  - ↑ sekrece endogenních opioidů
  - interakce serotoninových a dopaminergních receptorů
  - interakce adrenergního systému



Diskuze

Delirium tremens

- **Chronický abuzus a neurotransmitery**
  - down-regulace GABA-A receptorů
    - ✓ alkohol receptory aktivuje (chronicky - down-regulace)
    - ✓ syndrom z odnětí – ztráta stimulace a současně jejich nedostatek (tremor, dysforie, tachykardie, anxieta, křeče)
  - up-regulace excitačních postsynaptických glutamátových NMDA receptorů
    - ✓ alkohol receptory inhibuje (chronicky – up-regulace)
    - ✓ syndrom z odnětí – hyperreaktivita NMDA receptorů (tremor, ataxie, epi paroxysmy)

Review Article JOURNAL OF CLINICAL AND EXPERIMENTAL HEPATOLOGY

Delirium Tremens: Assessment and Management

Sandeep Grover*, Abhishek Ghosh†

*Professor, Department of Psychiatry, Post Graduate Institute of Medical Education and Research, Chandigarh, India and †Assistant Professor, Department of Psychiatry, Post Graduate Institute of Medical Education and Research, Chandigarh, India

Panel 2: Treatment regimes for Delirium Tremens

REGIMES	DOSES
Front loading	With diazepam: aim is to achieve light sedation (patient still could be aroused with verbal stimulation) or to bring down CIWA-Ar <8 5 mg IV → 5 mg IV (repeat after 10 min) 10 mg IV → 10 mg IV (repeat after 10 min) 20 mg IV after 10 min 5–20 mg IV per hour [Dosing must be continued till the aim of light sedation or the CIWA-Ar score has been achieved]
Symptom triggered	With diazepam 10–20 mg IV every 1–4 h → repeat doses till CIWA-Ar score <8
	With lorazepam: 4 mg IV to be repeated every 10 min till either of the aims of front loading is achieved If severe delirium still persists even after 16 mg IV→ 8 mg IV bolus is to be administered
Fixed dose	Not to be used for DT; Only for outpatient management of alcohol withdrawal syndrome

CIWA-Ar: Clinical Institute Withdrawal Assessment-Alcohol (revised); IV: Intravenous.

Panel 3: Treatment of benzodiazepine refractory delirium tremens

"Persistent CIWA-Ar >25, frank delirium or inability to control symptoms despite medication" and/or "requirement of ≥200 mg in the initial 3 h or ≥400 mg of diazepam in the first 8 h or ≥30 mg in the initial 3 h or ≥60 mg of lorazepam in the initial 8 h

FIRST CHOICE	**Phenobarbital** 60 mg IV bolus every 15 min BDZ dose to be halved (if patient is not intubated) Intubation is necessary if there are signs of respiratory depression and BDZ therapy is to be continued
SECOND CHOICE (If refractory to Phenobarbital as well)	**Propofol** infusion 0.3–1.25 mcg/kg/h RASS – (−3) to (−4) Maximum dose – 4 mcg/kg/h Maximum duration of administration – 48 h
THIRD CHOICE (If ventilator support is not available for propofol, it can be used as an alternative)	**Dexmedetomidine** Doses up to 0.7 µg per kilogram per hour
OTHER ALTERNATIVES	**Haloperidol**: uncontrolled agitation or hallucinations (0.5–5.0 mg intravenously or intramuscularly every 30–60 min as needed for severe agitation or hallucinosis – not to exceed 20 mg; or 0.5–5.0 mg orally every 4 h up to 30 mg **Ketamine**: total infusion rate of 0.20 mg/kg/h

Psychosomatics 2018:59:293–297
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Case Report

A Possible Role of Takotsubo Cardiomyopathy in Ventricular Fibrillation During Delirium Tremens: A Case Report and Literature Review

Takuto Ishida, M.D., Ph.D., Hiroyuki Uchida, M.D., Ph.D., Kazuki Miyazaki, M.D., Takahiro Yukawa, M.D., Kazuhiro Sugiyama, M.D., Yuuichi Hamabe, M.D., Masaru Mimura, M.D., Ph.D., Takefumi Suzuki, M.D., Ph.D.

CARDIOLOGY

Short Communication

Cardiology 2015;132:172–175
DOI: 10.1159/000435781

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Published online: August 8, 2015

Delirium Tremens Leads to Poor Outcomes for Acute Coronary Syndrome

Parijat S. Joy[a] Gagan Kumar[b]

[a]Department of Internal Medicine, University of Iowa Hospitals and Clinics, Iowa City, Iowa, and [b]Department of Critical Care, Phoebe Putney Memorial Hospital, Albany, Ga., USA

TABLE. Cases of Takotsubo Cardiomyopathy Associated With Alcohol Withdrawal

First author	Age/sex	Clinical features of alcohol withdrawal	Delirium tremens	Clinical features of Takotsubo cardiomyopathy	K, mmol/L	Mg, mg/dL	Presence of QT prolongation, QTc (msec)	VF
Suzuki[11]	64/M	Tremor	–	CPA	2.3	Not reported	+, 500	+
Mitchell[12]	49/F	Seizure, tachycardia, altered mental status	+	Pulmonary edema, cardiogenic shock	Not reported	Not reported	Not reported	–
Karla[13]	25/F	Seizure	Possible	CPA	3.2	1.4	+, 660	+
Thompson[14]	61/M	Severe alcohol withdrawal (symptoms were not reported)	Possible	Chest pain	Not reported	Not reported	Not reported	
Stollberger[15]	63/M	Seizure	Possible	Dyspnea, pulmonary edema	Not reported	Not reported	+, Value was not reported	–
Alexandre[16]	56/M	Confusion, asthenia, anorexia, tremor	+	Dyspnea, pulmonary edema	3.7	Not reported	+, Value was not reported	–
Yazdan-Ashoori[6]	57/F	Tremor, tachycardia, agitation, diaphoresis, seizure, fever	+	Hypotension, peripheral edema, elevated juglar venous pressure	2.7	Not reported	+, 558	–
Stout[17]	45/F	Tremor, tachycardia, nausea, emesis	–	Epigastralgia	Not reported	Not reported	Not reported	–
Omar[18]	57/F	Restlessness, agitation, tachycardia, tachypnea	+	Tachypnea, tachycardia, pulmonary edema, cardiogenic shock	Not reported	Not reported	+, Value was not reported	–
Agu[19]	57/F	Anxiety, tremor, diaphoresis, hallucination, tachycardia, agitation	+	Respiratory distress, pulmonary edema, VT	3.1	0.9	Not reported	–
Harris[20]	53/M	Seizure, tremor, disorientation, agitation	+	Hypotension	Not reported	Not reported	+, 645	–

CPA = cardiopulmonary arrest; COPD = chronic obstructive pulmonary disease; K = potassium; Mg = magnesium; VF = ventricular fibrillation; VT = ventricular tachycardia.

Original article
Peer reviewed article

ECG changes amongst patients with alcohol withdrawal seizures and delirium tremens

Florim Cuculi[a], *Richard Kobza*[b], *Tobias Ehmann*[a], *Paul Erne*[b]

[a] Department of Internal Medicine, Kantonales Spital Sursee-Wolhusen, Wolhusen, Switzerland
[b] Department of Cardiology, Kantonsspital Luzern, Luzern 16, Switzerland

Table 1

Laboratory characteristics on admission.

Laboratory parameter	Mean (SD)	Range	Normal Range	Below normal	Above normal
Sodium	135.3 (6.1)	98–176 mmol/l	136–145 mmol/l	41%	2%
Potassium	3.7 (0.5)	2.5–5.3 mmol/l	3.5–5.1 mmol/l	35%	2%
Leucocytes	8.0 (3.2)	2.6–17.0 × 10^9/l	4–10 × 10^9/l	8%	25%
Haemoglobin	137.7 (16.8)	98–176 g/l	140–160 g/l (m) 120–160 g/l (f)	49%	12%
Thrombocytes	139.2 (91.6)	33–538 × 10^9/l	150–450 × 10^9/l	65%	2%
MCV	98.1 (7.1)	81–110 fl	83–99 fl	0%	55%

Original article
Peer reviewed article

ECG changes amongst patients with alcohol withdrawal seizures and delirium tremens

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[a] Department of Internal Medicine, Kantonales Spital Sursee-Wolhusen, Wolhusen, Switzerland
[b] Department of Cardiology, Kantonsspital Luzern, Luzern 16, Switzerland

Table 2

Comparison of clinical and laboratory parameters between subjects with normal and prolonged QTc values.

	QTc normal (n = 18) Mean (standard deviation)	QTc prolonged (n = 31) Mean (standard deviation)
Age (years)	43.8 (7.7)	51.3 (10.7)
Sodium mmol/l	135.4 (6.8)	135. 2 (5.7)
Potassium mmol/l	3.7 (0.6)	3.7 (0.5)
Kreatinin µmol/l	69.0 (26.1)	86.4 (59.1)
Temperature C	37.7 (1.0)	37.5 (0.5)
Hospital stay in days	13.3 (6.9)	14.1 (9.6)
Neuroleptics use	n = 3 (60%)	n = 2 (40%)
Delirium tremens	n = 13 (39%)	n = 20 (61%)
Withdrawal seizures	n = 5 (29%)	n = 11 (71%)

Torsades de Pointes in Severe Alcohol Withdrawal and Cirrhosis: Implications for Risk Stratification and Management

Ted Yamamoto, MD; and Scott E. Friedman, MD

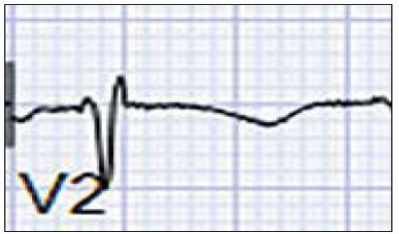
Close monitoring of the QT interval, timely and aggressive management of withdrawal, repletion of electrolytes, and telemetry monitoring may prevent life-threatening arrhythmias for patients being treated for acute alcohol withdrawal.

Figure 1. 2009 Electrocardiogram (QTC 409 ms)

Figure 2. Hospital Day 0 Electrocardiogram (QTC 519 ms)

Figure 3. Hospital Day 3 Electrocardiogram (QTC 549 ms)

Figure 4. 30-Day Follow-Up Electrocardiogram (QTC 499 ms)



- **Hypomagnezémie, hypokalcémie**
- **Torsades de pointes**

Diskuze

Delirium tremens a kardiovaskulární systém

- **Srdeční selhání, arytmie, zástava**
  - Hyperadrenergní reakce (prolongovaná)
  - QT interval
  - Minerálová dysbalance
    - ✓ ↓ K, Mg, glykémie vstupně
    - ✓ Křečová aktivita – rhabdomyolýza
      - ↑ K, ↑ CK-MB, ↑ myoglobin, MAC
      - Rychlý průběh

Nass *et al. BMC Neurology* (2017) 17:187
DOI 10.1186/s12883-017-0965-4

BMC Neurology

RESEARCH ARTICLE Open Access

Laboratory markers of cardiac and metabolic complications after generalized tonic-clonic seizures

Robert D. Nass[1], Sina Meiling[1], René P. Andrié[2], Christian E. Elger[1] and Rainer Surges[1,3*]

Table 3 Overview of documented complications in association with GTCS

Complication	N	%
Injuries	102/223	45.7
Troponin elevations	9/75	12
Aggressive, delirious or psychotic behavior	14 / 223	6.3
Respiratory problems	7/223	6.3
Prolonged impairment of consciousness	11 / 223	4.9
Acute kidney injury	5 /136	3.7
Rhabdomyolysis	4/207	1.9

J. Med. Toxicol. (2015) 11:195–200
DOI 10.1007/s13181-014-0451-y

TOXICOLOGY INVESTIGATION

Prevalence of Rhabdomyolysis in Sympathomimetic Toxicity: a Comparison of Stimulants

Ayrn D. O'Connor · Angie Padilla-Jones · Richard D. Gerkin · Michael Levine

Table 1 Summary of findings regarding single-agent exposures

Single-agent exposure (*n*)	Max CK IU/L median (range)	Rhabdomyolysis (CK>1000 IU/L)	Severe rhabdomyolysis (CK>10,000 IU/L)
Synthetic cathinones (19)	2638 (62–350,000+)	12/19 (63 %)	5/19 (26 %)
Methamphetamine (55)	665 (61–50,233)	22/55 (40 %)	2/55 (3.6 %)
Cocaine (9)	276 (87–25,614)	3/9 (33 %)	1/9 (11 %)
Other (6)	142 (51–816)	0/6 (0 %)	0/6 (0 %)

Psychosomatics 2018:59:405–407 © 2017 Academy of Consultation-Liaison Psychiatry. Published by Elsevier Inc. All rights reserved.

Case Report

A Case of Delirium and Rhabdomyolysis in Severe Iatrogenic Opioid Withdrawal

Jessie Hanna, M.D., Samantha Swetter, M.D.

Indian J Nephrol. 2011 Jan-Mar; 21(1): 66.
doi: 10.4103/0971-4065.78085
PMCID: PMC3109789
PMID: 21655176

Hypokalemia induced rhabdomyolysis

V. V. Jain, O. P. Gupta, S. U. Jajoo, and B. Khiangate

- **K – regulace krevního průtoku ve svalech**
  - K – vasodilatace v aktivním svalu
  - ↓K – ischémie – křeče – nekroza (rhabdomyolýza)
    - ✓ Potenciace hypovolémie

On the Mechanism of Rhabdomyolysis in Potassium Depletion

JAMES P. KNOCHEL and EDWARD M. SCHLEIN

From the Department of Internal Medicine, The University of Texas Southwestern Medical School at Dallas, 75235, and The Veterans Administration Hospital, 4500 South Lancaster Road, Dallas, Texas 75216

The Journal of Clinical Investigation Volume 51 July 1972

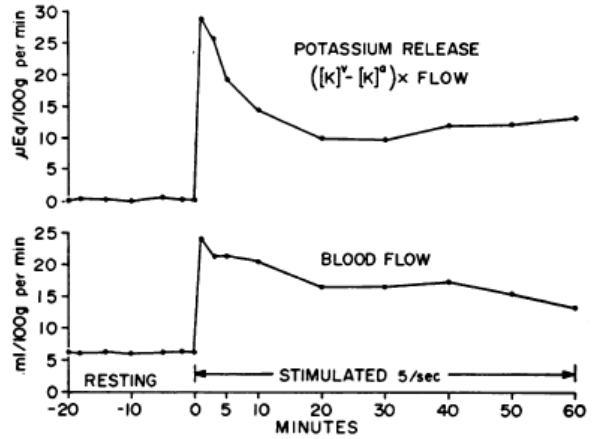


FIGURE 1 Serial values for potassium release and blood flow before and during stimulation of normal gracilis muscle.

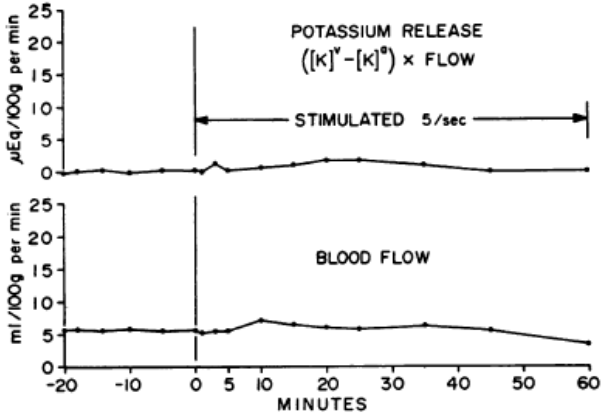


FIGURE 2 Serial values for potassium release and blood flow before and during stimulation of potassium-depleted gracilis muscle.

On the Mechanism of Rhabdomyolysis in Potassium Depletion

JAMES P. KNOCHEL and EDWARD M. SCHLEIN

From the Department of Internal Medicine, The University of Texas Southwestern Medical School at Dallas, 75235, and The Veterans Administration Hospital, 4500 South Lancaster Road, Dallas, Texas 75216

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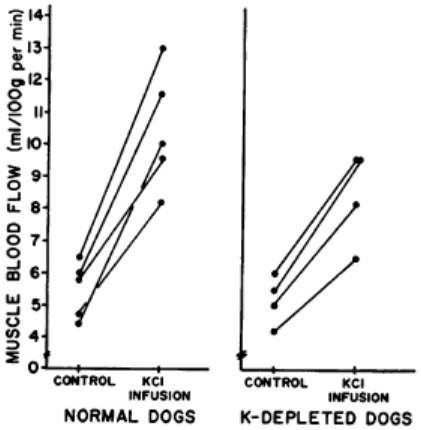

FIGURE 4 Effect of potassium infusion on resting muscle flow rate.

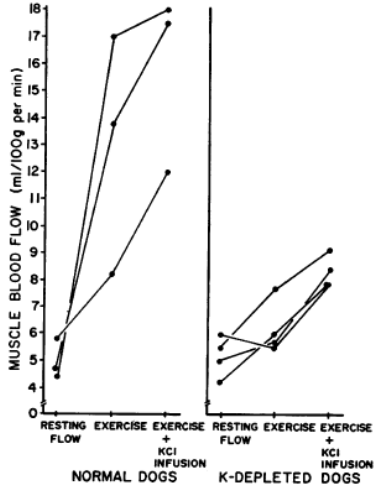

FIGURE 5 Effect of potassium infusion during exercise on muscle blood flow rate.

Hindawi Publishing Corporation
Case Reports in Obstetrics and Gynecology
Volume 2015, Article ID 947617, 3 pages
http://dx.doi.org/10.1155/2015/947617

Case Report

Hypokalemia-Induced Rhabdomyolysis as a result of Distal Renal Tubular Acidosis in a Pregnant Woman: A Case Report and Literature Review

Manasawee Srisuttayasathien

Department of Obstetrics and Gynecology, Chaophraya Yommaraj Hospital, Suphan Buri 72000, Thailand

Format: Abstract Send to

Pediatr Nephrol. 2006 Feb;21(2):289-91. Epub 2005 Oct 27.

Hypokalemia causing rhabdomyolysis resulting in life-threatening hyperkalemia.

Agrawal S[1], Agrawal V, Taneja A.

Author information

1 Department of Paediatrics, Max Medcentre, New Delhi, India. shrutiagrawal2000@yahoo.com

Abstract

Hyperkalemia is commonly associated with renal failure and is rare if renal function is normal. It is rarely caused by rhabdomyolysis and can be life threatening if unrecognized. Rhabdomyolysis is caused by many disorders, including hypokalemia. The available literature, both for human beings and experimental animals, contains evidence of hypokalemia causing rhabdomyolysis and rhabdomyolysis leading to hyperkalemia. Hypokalemia as a cause of rhabdomyolysis often goes unnoticed, because of the counteracting effect of rhabdomyolysis on serum potassium. There are no pediatric reports showing hypokalemia as a cause of rhabdomyolysis leading to life-threatening hyperkalemia. This case emphasizes the vigilance required toward the occurrence of rhabdomyolysis, which if not detected promptly may prove fatal.

Závěr

- **Delirium tremens - riziko fatálních komplikací**
  - kalium, myoglobin
- **Vstupní hypokalémie nevylučuje/precipituje rhabdomyolýzu (rychlost progrese) hypoperfuzí**
- **CAVE: kombinace křeče + hypokalémie**