



Società Italiana dell'Ipertensione Arteriosa
Lega Italiana contro l'Ipertensione Arteriosa

EVENTO FORMATIVO INTERREGIONALE SIIA
PIEMONTE | LIGURIA | VALLE D'AOSTA

Torino, 29 novembre 2025

Ipertensione Arteriosa e miopatia ipokaliemica

Massimiliano Uccelli
SC Medicina – Sanremo (IM)



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Torino, 29 novembre 2025

**No Nephrologist or Endocrinologist were harmed
in the making of this case report**



**THE REAL
WILD WEST**





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- Donna, 38 anni, non precedenti anamnestici di rilievo
- Da 8 mesi lamenta astenia, facile faticabilità, dolori muscolari sotto sforzo (va in palestra)
- Normopeso, non fumatrice, non beve alcolici, lavora come impiegata
- Valori pressori abituali nei limiti di norma





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Esegue esami ematici tramite il MMG:

CPK: 2002

GOT 220 U/I GPT 81 U/I

Emocromo : Hb 15,4 wbc 5200 plt 243.000

Ecocolordoppler Venoso AA.II. , nella norma

Visita Neurologica: nds eccetto **lieve iporeflessia** profonda

Viene inviata ad eseguire ETG addome superiore per studio del fegato





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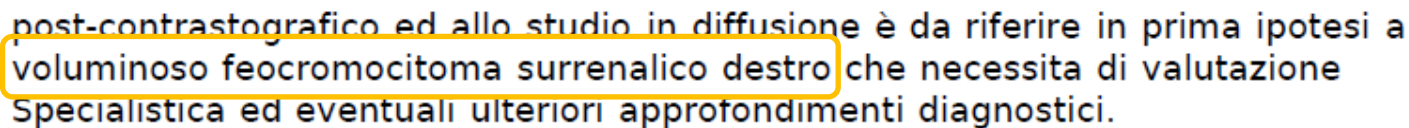
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ETG addome: nulla a fegato milza pancreas:
voluminosa neoformazione in loggia surrenalica dx di
circa 65 x 75 mm

Viene inviata a valutazione internistica e viene
ricoverata per accertamenti







Riassumendo:

1. Ipostenia e dolori muscolari, modesta rabdomiolisi
2. MRI: sospetto morfologico di feocromocitoma

Signs/symptoms	Patient percentage
Classic triad (headache+diaphoresis+tachycardia)	21 (5/24)
Hypertension	33 (8/24)
Labile blood pressure	4 (1/24)
Palpitations	8 (2/24)
Headache	8 (2/24)
Abdominal pain	4 (1/24)
Adrenal hemorrhage	4 (1/24)
Asymptomatic	25 (6/24)



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EO: normopeso, PA 160/90 mmHg, lievissima succulenza perimalleolare,
Struttura osteomuscolare e rappresentazione adiposa armonica
Obiettività toracica e addominale inespressiva

Azotemia	13	<	mg/dl
Sodio	146	>	mmoli/l
Potassio	1.4	<	mmoli/l
<i>Ricontrollato. Valore comunicato telefonicamente al medico curante.</i>			
Glicemia	72		mg/dl
Calcio	7.6	<	mg/dl
Creatinina	0.50		mg/dl
GOT/AST	81	>	U.I./l
GPT/ALT	63	>	U.I./l
Fosfatasi Alcalina	31	<	U.I./l
Gamma G.T.	12		U/L
LDH	353	>	U.I./l

pH	7.54	>
pCO2	50.4	
pO2	62.9	>
cCl-	92	<
cHCO3 - (P,st)	43.4	



The Spectrum of Rhabdomyolysis

PATRICIA A. GABOW, WILLIAM D. KAEHNY, AND STEPHEN P. KELLEHER

146

THE SPECTRUM OF RHABDOMYOLYSIS

TABLE 5. Reported Causes for Rhabdomyolysis

I. Excessive Muscular Activity Contact sports (46, 125) Noncontact sports (3, 7, 10, 34, 51, 56, 87, 91, 111, 143) Seizures (26, 39, 98, 153, 160, 164) Delirium tremens Status asthmaticus (25) Psychosis (30)	Succinylcholine (140) Clofibrate (157) Epsilon aminocaproic acid (15, 17, 136)
II. Direct Muscle Injury Trauma (20, 33, 39, 49, 102, 178) Burns (43)	VII. Toxins Ethanol (4, 39, 42, 78, 84, 94, 121, 122, 129, 139, 146, 150, 158, 176) Isopropyl alcohol Carbon monoxide (89) Mercuric chloride (27) Ethylene glycol (114) Toluene, paint sniffing (162) Quail ingestion, ? hemlock (13) Snake bite (61, 130) Hornet or wasp sting (154) Brown spider bite Haff's disease
III. Ischemia Compression (24, 39, 48, 113, 117, 119, 131, 147) Vascular occlusion (53, 64, 112) Sickle cell trait (79) Air embolism (43)	VIII. Infections Bacterial tetanus Legionnaire's disease (124) pyomyositis (6) other (134, 159) Viral influenza (31, 60, 72, 105, 106, 133, 152, 181) infectious mononucleosis (67) other (12, 31, 63, 67, 72, 133, 144, 175)
IV. Immunological Diseases Dermatomyositis (65, 71, 93) Polymyositis (65, 82, 92, 123, 156)	IX. Genetic Disorders Abnormal carbohydrate metabolism myophosphorylase deficiency (51, 52, 99, 115, 145) alpha-glucosidase deficiency amylo-1-6-glucosidase deficiency phosphohexomerase deficiency (141) phosphofructokinase deficiency (83) Abnormal lipid metabolism carnitine deficiency (172) carnitine palmityl transferase deficiency (8, 62, 118, 132)
V. Metabolic Disorders Diabetes mellitus hypersmolar nonketotic coma (51) ketonacidosis (127) Hypokalemia diuretics (110) carbenoxolone (9, 35) amphotericin (38) parenteral nutrition (107) licorice (166) primary hyperaldosteronism (37) cortisone therapy (57) renal tubular acidosis (23)	X. Miscellaneous Idiopathic, recurrent (16, 29, 41, 80, 86, 90, 165) Temperature extremes hyperthermia (26, 76, 85, 95, 155, 170) hypothermia (125) Electric shock, lightning (179)
VI. Drugs Heroin (32, 39, 50, 73, 78, 128, 135, 137, 147, 148, 150, 167, 177) Methadone (45) Phencyclidine (2, 11, 28, 116) Amphetamines (51, 70) L.S.D. Glutethimide (58) Salicylate overdose (14, 155)	

Hypokalemia

diuretics (110)

carbenoxolone (9, 35)

amphotericin (38)

parenteral nutrition (107)

licorice (166)

primary hyperaldosteronism (37)

cortisone therapy (57)

renal tubular acidosis (23)



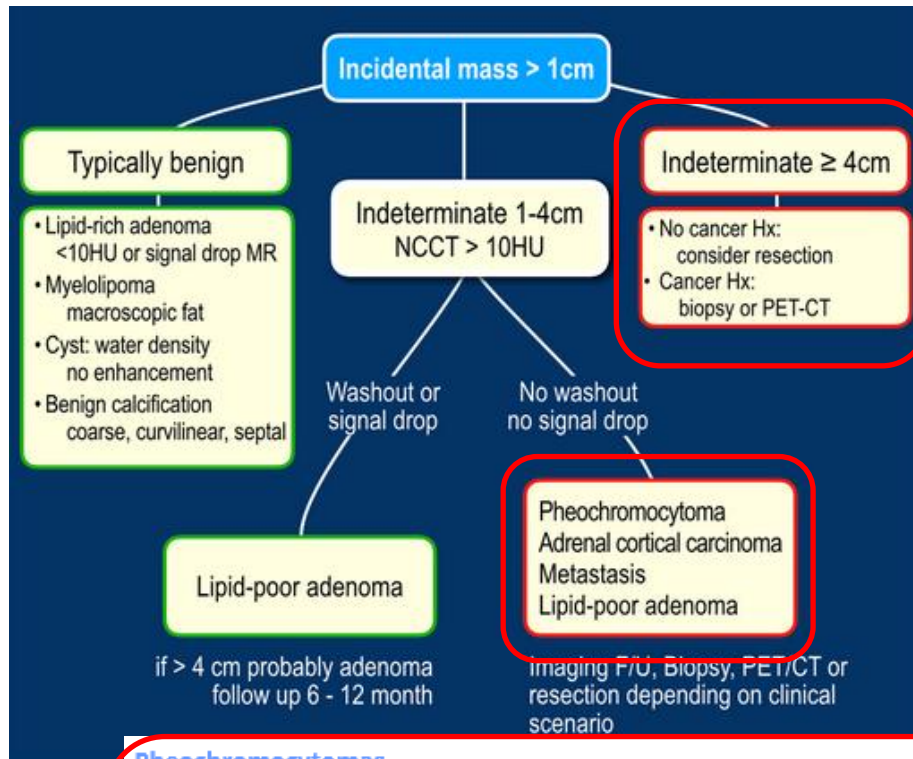
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Torino, 29 novembre 2025

Riassumendo:

1. Ipostenia e dolori muscolari, modesta rabdomiolisi, ipertensione
2. MRI: massa surrenalica 7 cm, sospetto **morfologico** di feocromocitoma
3. Severa ipokaliemia (1,4 mEq/L)



Cosa ci dicono i radiologi..

Pheochromocytomas

Pheochromocytomas are rare tumors that originate in the adrenal medulla. Usually, tumors are larger than 3 cm when found. Nonfunctioning tumors are typically larger than functioning tumors.

A typical pheochromocytoma will have an unenhanced density >10 HU, or higher in case of hemorrhage.

They are highly vascular, resulting in strong enhancement, but in contrast to adenomas they usually have delayed washout [4,5].



Adrenal Mass and Hypokalaemia: The Zebra Among Horses

Zsuzsanna Reti ¹, Laszlo Szabo ¹, Radu M. Neagoe ², Melinda Kolcsar ³



Abstract

Pheochromocytoma rarely presents with unexplained hypokalaemia, although there are some case reports in the literature. The mechanism behind this could be the increased cellular potassium uptake promoted by beta-2-adrenoreceptor hyperactivation and insulin resistance.

What is the most sensitive and specific imaging technique for identifying pheochromocytomas?

 Ariel, Hoffman MD; Jonathan, Letko DO; Marc-Eli, Faldas DO; Jessica, Coulter MD

[Author Information](#) 

Evidence-Based Practice 27(4):p 26-27, April 2024. | DOI: 10.1097/EBP.0000000000002018

Huang et al. *BMC Medical Imaging* (2024) 24:175
<https://doi.org/10.1186/s12880-024-01350-0>

BMC Medical Imaging

RESEARCH

Open Access

Diagnostic performance of magnetic resonance imaging features to differentiate adrenal pheochromocytoma from adrenal tumors with positive biochemical testing results



Rukun Huang^{1,2†}, Tingsheng Lin^{1,2†}, Mengxia Chen^{1,2†}, Xiaogong Li^{1,2*} and Hongqian Guo^{1,2*}

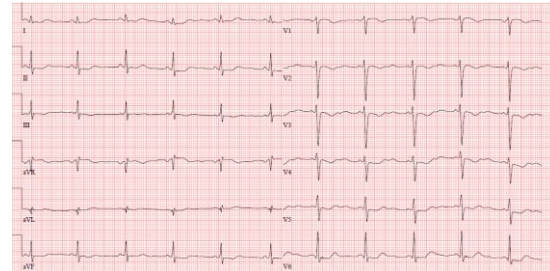


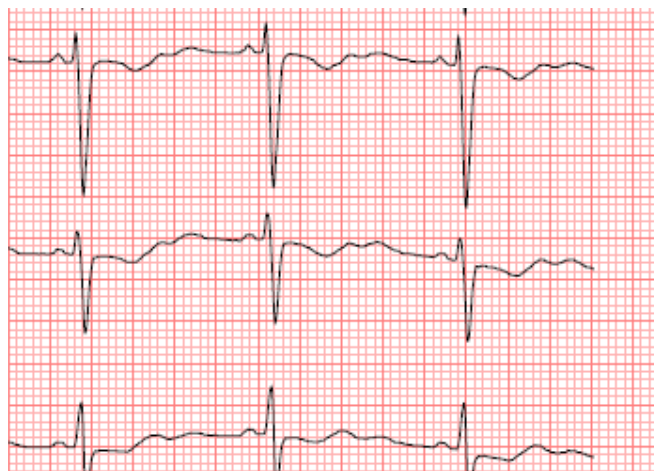
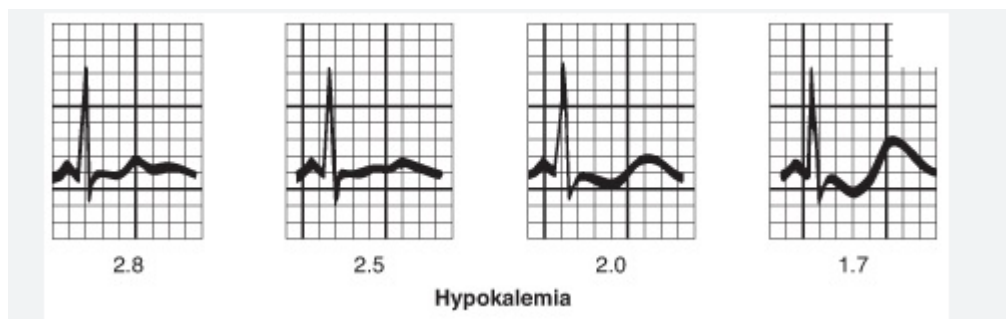
Urine: **Cloruri 65 mEq/L**

Plasma: **K+ 1.4 mEq/L pH 7.54**

- Comincia correzione **urgente** e.v. dell'ipokaliemia, sotto monitoraggio ECG
- Correzione di concomitante ipomagnesiemia
- Concomita lieve volume espansione, alcalosi metabolica verosimilmente cloruro-resistente

Frequenza ventricolare	65	BPM
Intervallo PR	110	ms
Durata QRS	102	ms
QT/QTc	456/474	ms
Assi P-R-T	43 67	1





esami	valore
K+	1,4 mEq/L ↓
pH	7,54
HCO ₃	43 mmol/L
Cl-	92 mEq/L ↓
Metanefrine 24 h x 3	Nella norma
Cortisolo /ACTH	12.90 mcg/dL/ 38pg/mL →
DHEA	122
aldosterone	89→ 145 ↑
renina	0.5
testosterone	21 ng/dL

Nugent: 0.8 mcg/dL

Aggiunge terapia con spironolattone 25 mg x 2 → 100 mg x 2



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Logo italiana contro l'Ipertensione Arteriosa

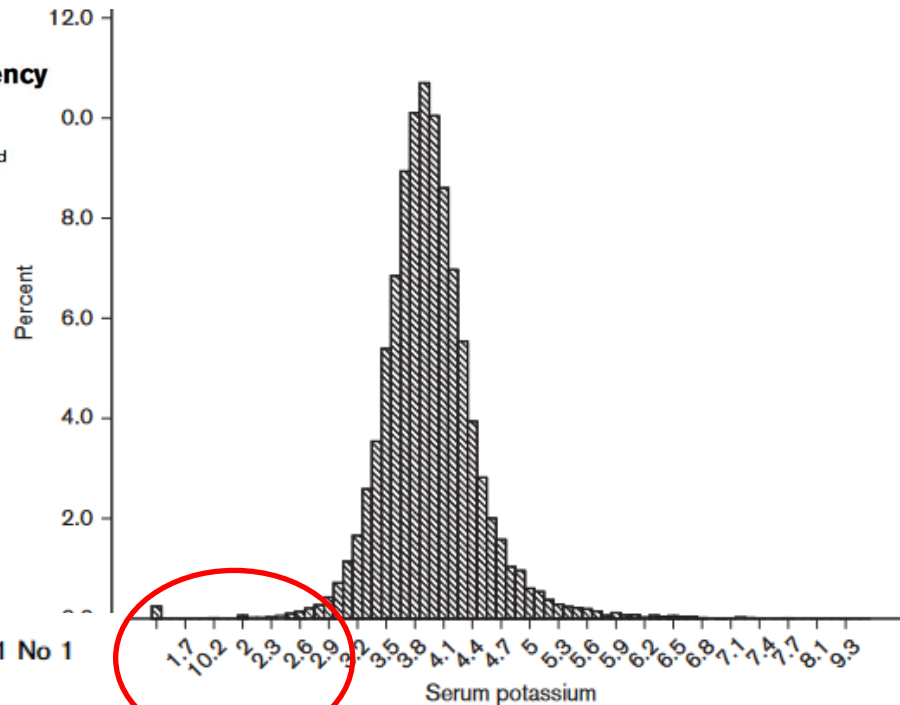
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Severe hypokalemia (≤ 2.5 mEq/L) was observed in 87 patients (0.4%)

Distribution of serum potassium on admission to the emergency department. Serum potassium in mmol/l ($N=43\,805$).

Etiology and symptoms of severe hypokalemia in emergency department patients

Grischa Marti^b, Christoph Schwarz^e, Alexander B. Leichtle^a,
Georg-Martin Fiedler^a, Spyridon Arampatzis^c, Aristomenis K. Exadaktylos^d
and Gregor Lindner^{b,d}



Adrenocortical carcinoma: a practical guide for clinicians

Martin Fassnacht, Soraya Puglisi, Otilia Kimpel, Massimo Terzolo

Lancet Diabetes Endocrinol

2025; 13: 438–52

Published Online

March 11, 2025

[https://doi.org/10.1016/S2213-8587\(24\)00378-4](https://doi.org/10.1016/S2213-8587(24)00378-4)

Hormonal investigations

- Glucocorticoid excess
 - 1 mg dexamethasone suppression test or free cortisol in 24 h urine*
 - Basal adrenocorticotrophic hormone in plasma†
- Sex steroids and steroid precursors‡
 - Dehydroepiandrosterone sulphate
 - 17-hydroxyprogesterone
 - Androstenedione
 - Testosterone (only in women)
 - 17- β oestradiol (only in women who are postmenopausal and men)
- 11-deoxycortisol
- Mineralocorticoid excess
 - Potassium
 - Aldosterone to renin ratio (only in patients with arterial hypertension or hypokalaemia, or both)
- Exclusion of a pheochromocytoma
 - Fractionated metanephrines in 24 h urine or free plasma metanephrines

Imaging

- CT or MRI of abdomen and pelvis
- Chest CT
- [^{18}F]fluorodeoxyglucose PET or CT scans§
- Bone or brain imaging (when skeletal or cerebral metastases are suspected)

Panel 1: Diagnostic investigations in patients with suspected or proven adrenocortical carcinoma (adapted from Fassnacht et al¹⁶)



Metanefrine nella norma ...

Essendoci allarmati *ab initio* abbiamo comunque fatto

^{18}F -DOPA TC PET, «ovviamente» negativa

^{18}F FDG TC PET, con intensa attività metabolica localizzata

ADRENOCORTICAL CARCINOMA (ACC)

0.7-2 per million / year. Median age: 40-60 years



Signs & Symptoms

Autonomous adrenal hormone ↑
Mixed steroid excess is frequent



Androgen ↑
(virilization)



Hypocortisolism
(see Cushing's syndrome)

Rare

Mineralocorticoid ↑
(see primary hyperaldosteronism)

Oestrogen ↑
(feminization)



Abdominal mass
effect (30%)



Abdominal
discomfort
(nausea, vomiting,
abdominal fullness)



Back pain

Classical malignancy-associated
symptoms (rare)



Weight loss



Night sweat



Fatigue



Fever

10-15% incidentally discovered

Functional Breakdown of Adrenal Cortical Carcinomas

Nonfunctional	21%-50%
Functional	50%-79%
Cushing syndrome	33%-53%
Cushing syndrome + virilization	20%-24%
Virilization alone	10%-20%
Feminization	6%-10%
Hyperaldosteronism	2.5%-5%

Aldosterone hypersecretion is the
least common (0-7%) among the functional
adrenocortical carcinoma (ACC).



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novembre 2025

> *Am J Med.* 1955 Dec;19(6):966-75. doi: 10.1016/0002-9343(55)90163-7.

Adrenal cortical carcinoma producing solely mineralocorticoid effect

L V FOYE Jr, T V FEICHTMEIR

PMID: 13275491 DOI: [10.1016/0002-9343\(55\)90163-7](https://doi.org/10.1016/0002-9343(55)90163-7)

1955

Endocrine-Related Cancer (2005) 12 149–159

Aldosterone-producing adrenocortical carcinoma: an unusual cause of Conn's syndrome with an ominous clinical course



Teresa M Seccia¹, Ambrogio Fassina², Gastone G Nussdorfer³,
Achille C Pessina⁴ and Gian Paolo Rossi⁴

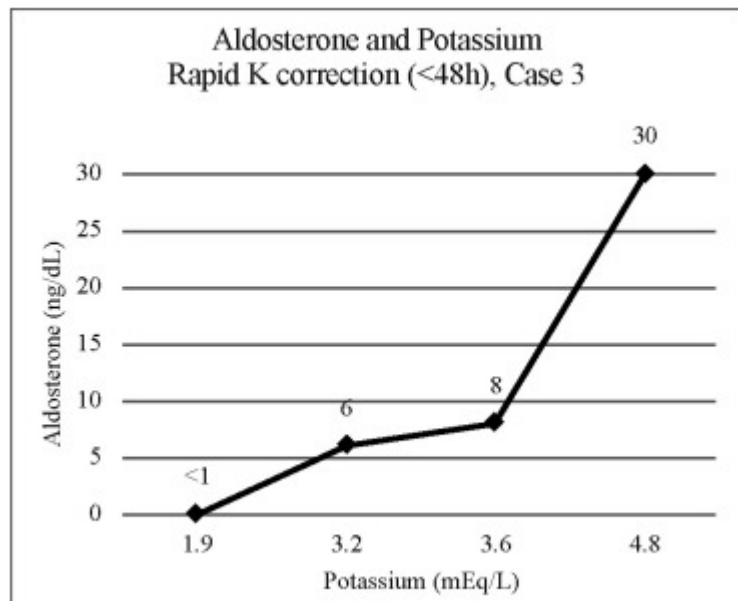
**Adrenal cortical carcinoma:
an unusual cause
of hyperaldosteronism**

ADAM H. TELNER, MD, FRCP[C]

CAN MED ASSOC J, VOL. 129, OCTOBER 1, 1983

Hypokalemia may Mask Primary Aldosteronism: A Case Series

Michael Morkos MD, MS, Yu-Chien Cheng MD, Leon Fogelfeld MD  



Classificazione	Livello di Potassio
Ipopotassiemia Lieve	3 – 3.5 mEq/L
Ipopotassiemia Moderata	< 3 mEq/L
Ipopotassiemia Severa	<2.5 mEq/L / Sintomi

Riduzione di 1 mEq/l di potassio

Deficit di potassio di 100 – 200 mEq

pH Aumenta di 0.1

Potassio scende di 0.6 mEq/l

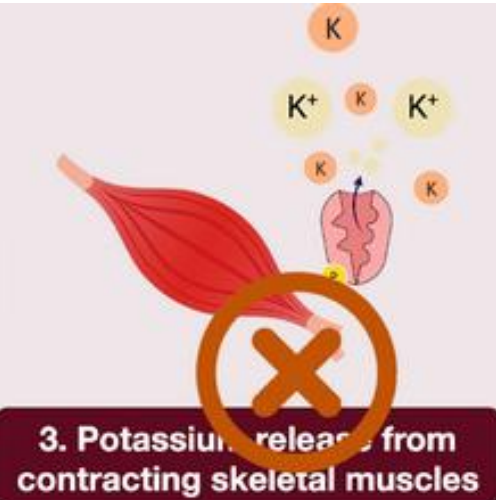
60 mEq di potassio

incremento di 1 mEq/l

1. Physical activity



2. Requires a higher amount of energy



3. Potassium release from contracting skeletal muscles



4. Potassium-mediated arteriolar vasodilation



Hypokalemic Rhabdomyolysis

- Potassium-depleted skeletal muscle release limited K
- Hampers the physiologic vasodilation
- Relative ischemia
- Skeletal muscle injury or frank necrosis

Primary Aldosteronism Associated with Severe Rhabdomyolysis Due to Profound Hypokalemia

Atsushi Goto¹, Yoshihiko Takahashi¹, Miyako Kishimoto¹, Shigeru Minowada²,
Hitoshi Aibe³, Kanehiro Hasuo³, Hiroshi Kajio¹ and Mitsuhiro Noda¹

				ness	3456	1.6	Saito S	2007	Abstract
2	73	M	Weakness, Myalgia	6821	1.7	Tomaru A	2007	Abstract	
3	31	M	Weakness	1148	2.1	Nakagawa H	2006	Abstract	
4	41	F	Weakness, Myalgia	6600	1.4	Uetake Y	2005	Abstract	
5	58	F	Myalgia	16190	1.6	Makita T	2004	Abstract (17)	
6	55	M	Weakness	881	1.8	Atsumi T	1979	Article (18)	
The present case	55	M	Weakness, Myalgia	15760	1.4	Goto A	2008	-	

Information was obtained from abstracts or articles in Japanese. Only first authors are listed, and an abstract for No.5 and an article for No. 6 are cited in the references. Abstracts for No.1-4 can be found in the website of the Japanese Society of Internal Medicine (<https://www.naika.or.jp>), which requires ID and password to login. Abbreviation: CPK, creatinine phosphokinase.

Primary hyperaldosteronism leading to hypokalemia is a rare but important cause of rhabdomyolysis :

Primary aldosteronism associated with severe hypokalemic rhabdomyolysis

Wan-Ting Tsai,^{1,6} Yen-Lin Chen,⁷ Wei-Shiung Yang,^{1,2} Hong-Da Lin,^{3,7}
Chih-Cheng Chien,⁴ Ching-Ling Lin⁵

Dear Sir,

Reports associating hypokalemic rhabdomyolysis with primary aldosteronism are rare.^{1,2} Hypokalemia does not develop in every patient with primary aldosteronism, and hypokalemic rhabdomyolysis is even rarer in patients with primary aldosteronism.³ We

Severe hypokalemia in the emergency department: A retrospective, single-center study


Ryuichirou Makinouchi¹ | Shinji Machida¹ | Katsuomi Matsui¹ | Yugo Shibagaki² | Naohiko Imai¹ 

TABLE 3 Etiology of severe hypokalemia.

	N = 54
Malnutrition [#]	16 (30%)
Use of Japanese herbal medicine ^a	14 (26%)
Diuretics ^b	13 (24%)
Diarrhea	7 (13%)
Hypothermia	3 (6%)
Insulin	3 (6%)
Vomiting	2 (4%)
Others	10 (19%)

TABLE 2 Symptoms of severe hypokalemia.

	N = 15
Weakness [#]	12 (80%)
Severe rhabdomyolysis	4 (27%)
Constipation	2 (13%)
Muscle cramps	1 (7%)

Note: Some patients had more than one symptom.

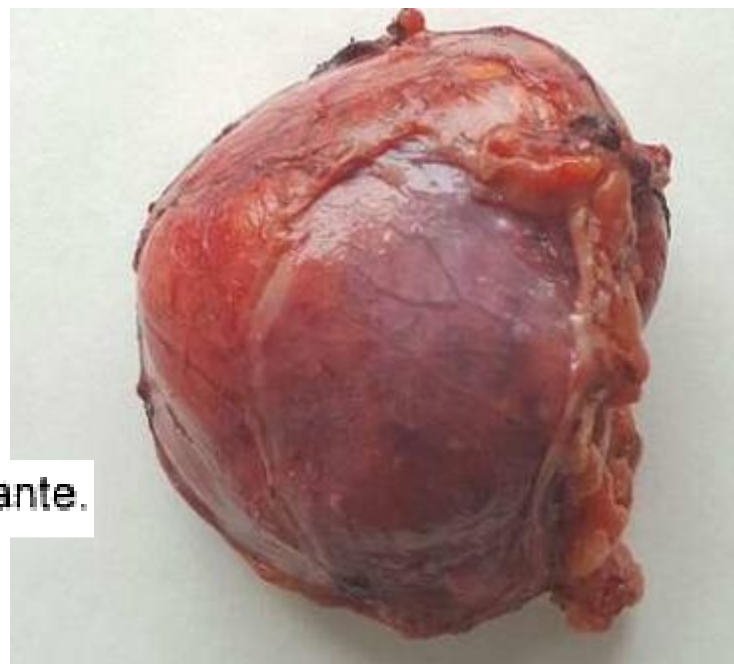
[#] $p = 0.001$.

- 24/01/2025 Intervento chirurgico di surrenectomia destra laparotomica.

El: Carcinoma cortico-surrenalico. Dimensioni 8.8x8.6x7.2 cm. Nuclei marcatamente atipici, figure apoptotiche con focale necrosi tumorale. Capsula focalmente infiltrata senza estensione all'adipe circostante. Si evidenziano emboli vascolari. Mitosi >40/50 HPF, alcune mitosi atipiche. Ki67 mediamente 15% con aree hotspot a circa 35-40%. ER 0%, PgR 30%. Margini indenni (R0), invasione linfo-vascolare presente, invasione sinusoidale presente. pT4 N0 (0/4).

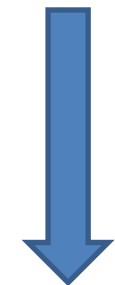
17/03/2025 Indicazione ad avvio di Mitotane adiuvante.

+ cortone acetato

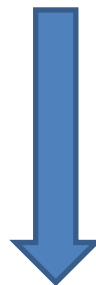




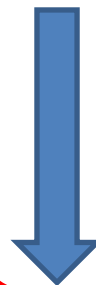
THE REAL WILD WEST



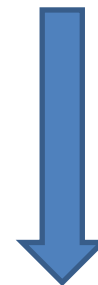
sintomi



CPK/GOT
Visita Neurologica



Gestione internistica



ETG

K+,
imaging

Marzo 24

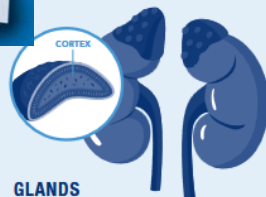
9 mesi

Ottobre 24 Novembre 24 Dicembre 24





NAVIGATING AN ADRENOCORTICAL CARCINOMA DIAGNOSIS



GLANDS

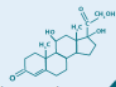
Adrenal glands produce hormones. The inner adrenal medulla make catecholamines (noradrenaline and adrenaline) and the outer adrenal cortex make steroid hormones that regulate salt (MINERALOCORTICOIDs or aldosterone) sugar (GLUCOCORTICOID or cortisol) and sex hormones (androgen or DHEAS). Adrenocortical Carcinoma (ACC) is a term specifically referring to a cancer of the adrenal cortex.

HORMONES

Approximately 50% of ACCs produce excessive symptomatic steroid hormones, or more than normal. In these cases, signs or symptoms of hormone excess will be present.

May also experience worsening high blood pressure, diabetes, weight gain, menstrual irregularities, hirsutism, or erectile dysfunction.

ACC is also associated with an inherited cancer syndrome requiring genetic testing and counseling.



DIAGNOSIS

Adrenocortical carcinoma (ACC) is rare. It is a malignant tumor, meaning it has the potential to spread (metastasize) to other organs in the body.

In more than 50% of cases, ACC is diagnosed by chance during imaging studies for other reasons.

Alternatively, the diagnosis is made during evaluation for potential hormone excess.

SYMPTOMS

The symptoms of ACC may vary depending on the extent of the disease (size and metastasis) and hormone excess.

- Asymptomatic: without symptoms
- Rarely abdominal discomfort associated with tumor "burden weight"
- Hormone excess: high blood pressure, diabetes, weight gain, menstrual irregularities, hirsutism, and/or libidinal/erectile dysfunction

DIAGNOSIS

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THE REAL WILD WEST



Visit endocrine.org for more information.

Editors: Irina Bancos, MD; Fady Hannah-Shmouni, MD, FRCP; Gary Hammer, MD, PhD





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PIEMONTE | LIGURIA | VALLE D'AOSTA

Torino, 29 novembre 2025

Interesse del caso:

- 1- ACC malattia rara, ad esordio clinico spesso subdolo per paucisintomaticità
- 2- nel nostro caso:
 - sintomi presenti ma interpretati tardivamente
 - diagnosi tardiva come statisticamente frequente in letteratura
- 3- dato di ipokaliemia particolarmente severa, persistente verosimilmente da lungo tempo, non diagnosticata e non trattata
- 4- rarità dell'iperaldosteronismo isolato nel contesto di un ACC

IL CONTRARIO DI SERENDIPITY È **ZEMBLANITY**

In tempi più recenti, il romanziere scozzese William Boyd ha coniato il termine **zemblanity** a significare l'opposto di **serendipity**: "fare scoperte infelici, sfortunate e prevedibili che si verificano secondo un disegno". Deriva da Novaja Zemlja, un luogo freddo e spoglio, il più lontano che si possa immaginare,



Zemblanity: the inexorability of unfortunate discoveries



Sanremo, Ospedale, 2025