JOINT MEETING
In pediatria e medicina dell'adolescenza
Sobre pediatría y medicina de la adolescencia

21-22-23-24 OTTOBRE 2015 CATANZARO ITALIA

Azienda Ospedaliera di Catanzaro "Pugliese-Ciaccio"

Casi clinici

G. Raiola, V. Talarico

7th JOINT MEETING
In pediatria e medicina dell'adolescenza
Sobre pediatría y medicina de la adolescencia

21-22-23-24 OTTOBRE 2015 CATANZARO ITALIA

1° caso clinico

Bimbo di 18 mesi giungeva alla nostra osservazione per dolori addominali, vomito ed inappetenza insorti 6 giorni prima

ANAMNESI FAMILIARE E PATOLOGICA REMOTA: negative.

ESAME OBIETTIVO

-Segni vitali normali

- no febbre

- Agitazione

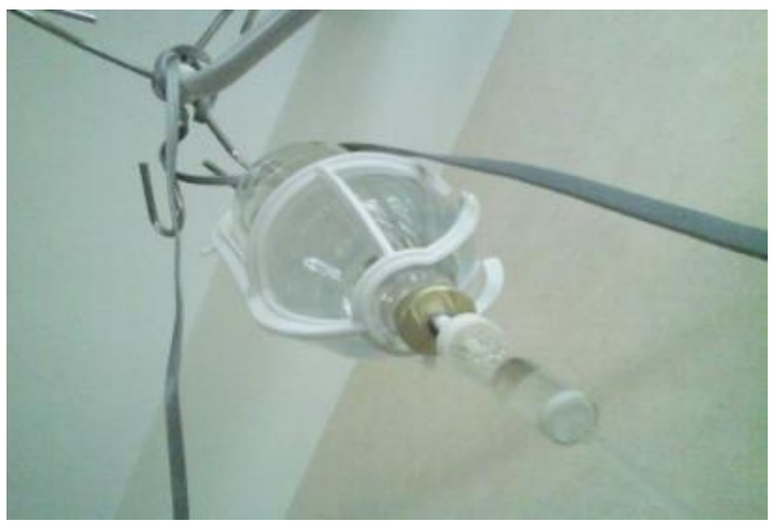
- Disidratazione lieve-moderata (6%)

- Obiettività toraco-cardiaca-dddominale negativa

- Peso 7.1 kg (-4.7 SDS), altezza71 cm (-2.9 SDS),

EGA ph: 7,44
pCO2 40 mmHg
HCO3- 30 mmol/L
BE 3.2
Na 143 mEq/l K 4,2 mEq/l

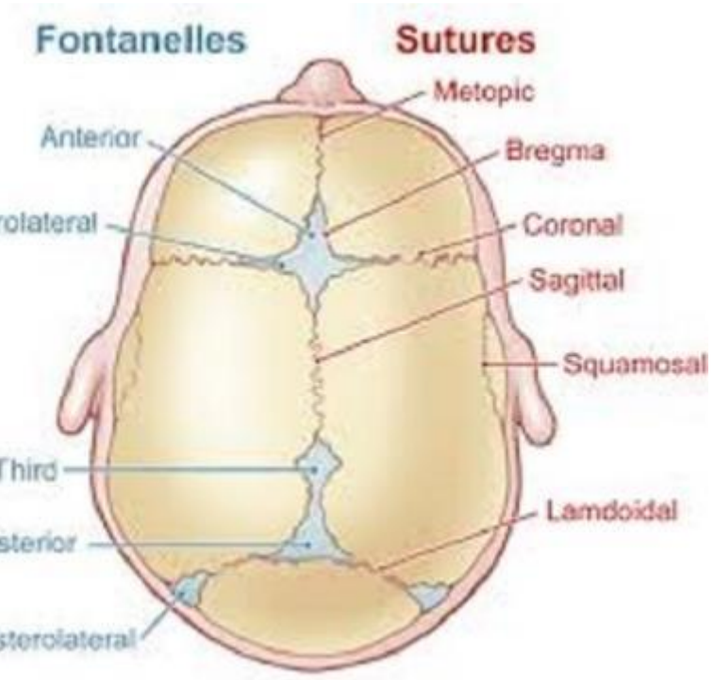
SDS)



Dopo 3 ore progressivo miglioramento del vomito

Dopo 6 scomparsa del vomito ma incremento del dolore addominale con assoluto rifiuto all'alimentazione

Il bambino non evacuava da 5 giorni



-Storia di stipsi da 2 mesi

-Non assunzione di farmaci, eccetto supplementazione con Vitamina D da 3 mesi

(100.0 Vit A):

<u>2 fl al giorno</u>

ESAMI DI LABORATORIO

EMOCROMO
GR 5530000, HB 14.6 g/dl, HT 42 %, MCV 75 fl
MCH 23,6 pg, PLT 525.000,
GB 9.600, (N 54,4%, L 29%)

Calcio sierico **11.8 mg/dl** (r

(range 0.2-1.3)

25- idrossi-vitamina D(25-OHD)
2.271 ng/ml (range 30-100)

Paratormone <3 pg/ml (range 4.6-58.1)

Intossicazione da Vitamina D

The Turkish Journal of Pediatrics 2012; 54: 93-98 Review

Vitamin D intoxication

Behzat Özkan[1], Şükrü Hatun[2], Abdullah Bereket[3]

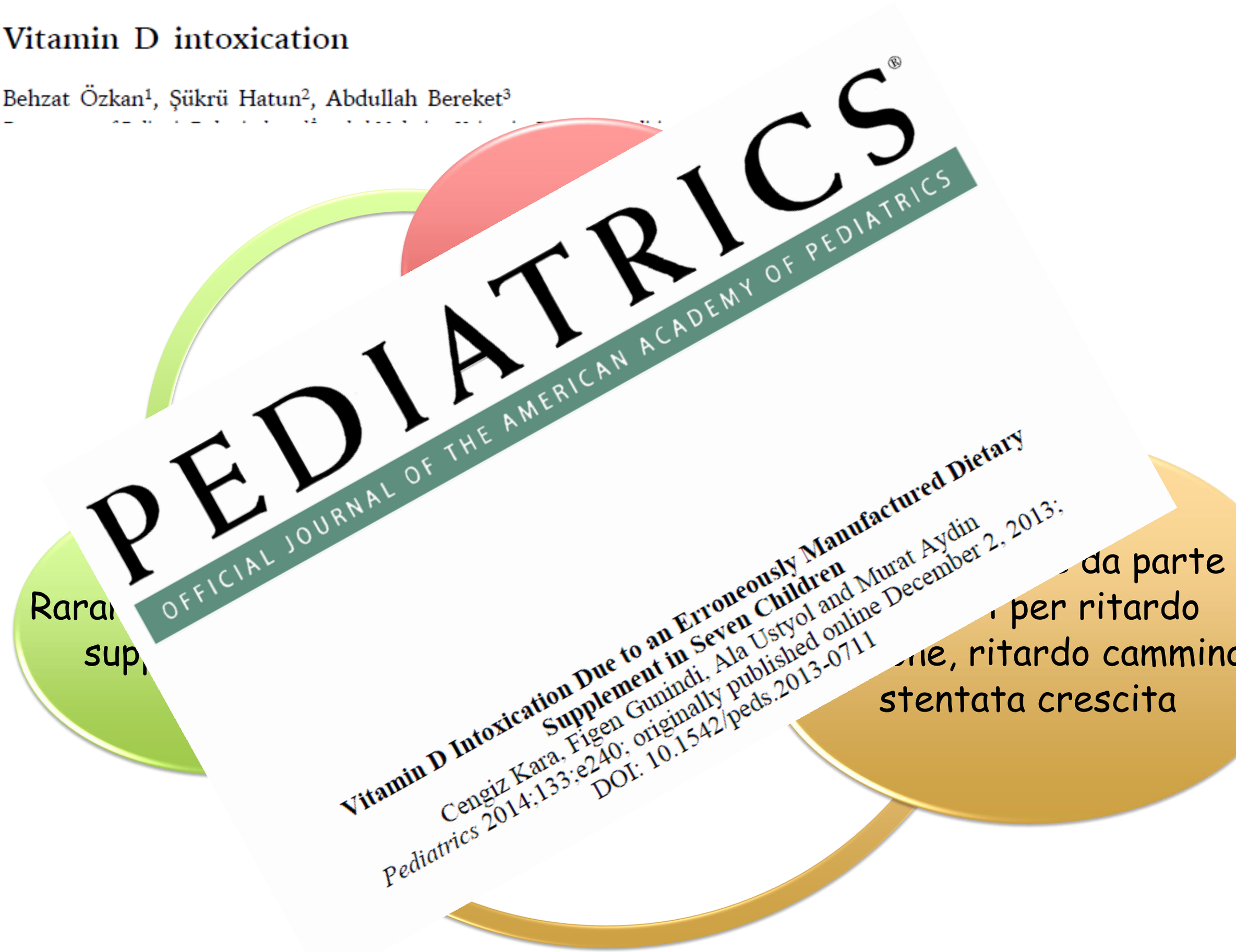


Figura 1. Stato vitaminico D in base alle concentrazioni circolanti di 25-OH-D durante l'età evolutiva e possibili conseguenze dell'ipovitaminosi D. I valori normali di 25-OH-D sono compresi tra 20 e 100 ng/ml. Valori >150 ng/ml possono associarsi ad ipercalcemia. Valori <20 ng/ml sarebbero indicativi di una condizione di

The Turkish Journal of Pediatrics 2012; 54: 93-98 *Review*

Vitamin D intoxication

Behzat Özkan[1], Şükrü Hatun[2], Abdullah Bereket[3]

Departments of Pediatric Endocrinology, [1]İstanbul Medeniyet University Faculty of Medicine, İstanbul, [2]Kocaeli University

Def

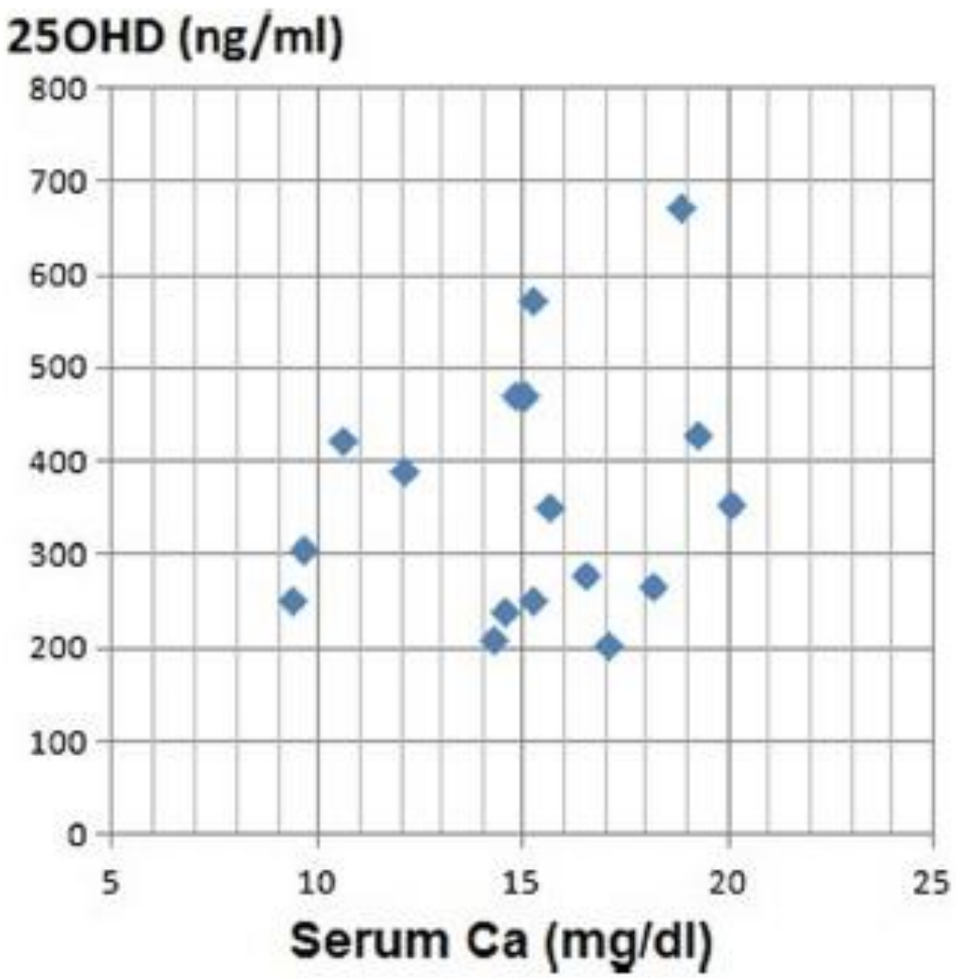
Rac

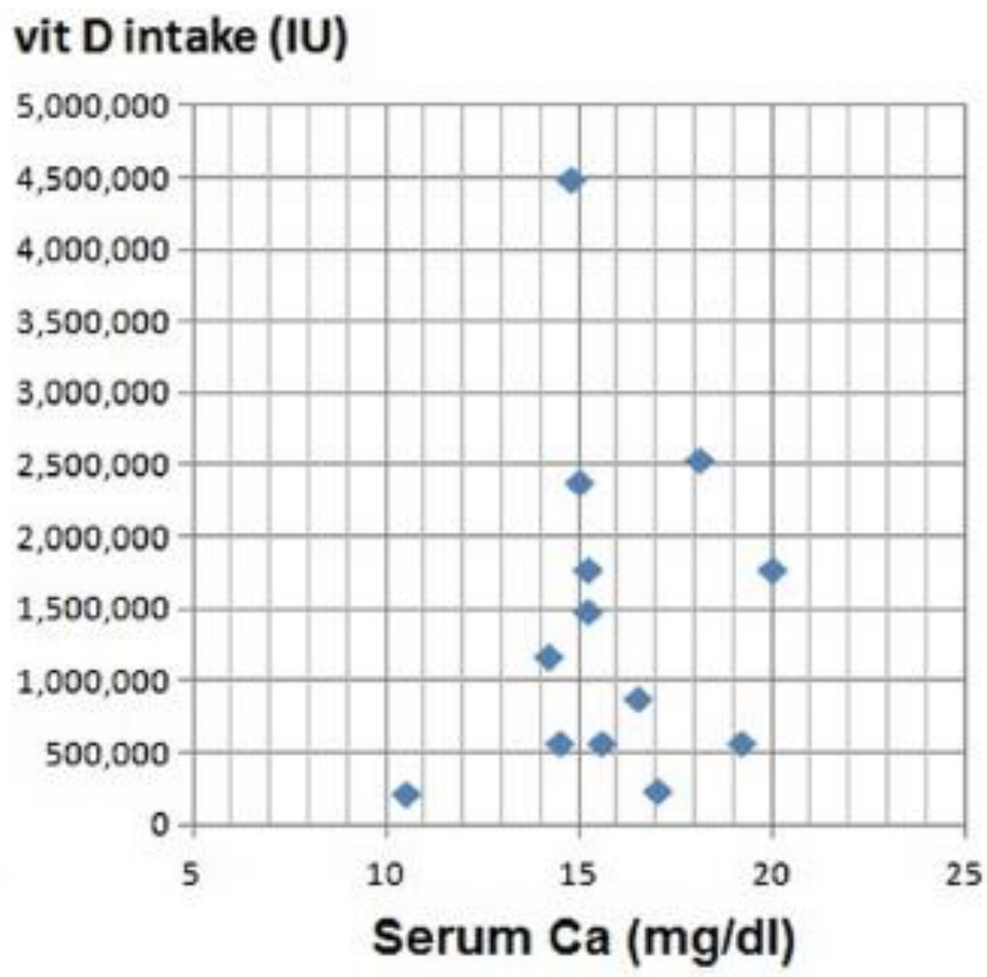
and sarcoidosis, in addition to excess oral or parenteral intake of vitamin D[2,5-7]. There is no consensus on the dose of oral vitamin D that leads to intoxication; individual variability must be considered with VDI[4]. In studies conducted on animals, the toxic dose has been reported as 0.5 mg/kg (20,000 IU/kg), whereas the lethal dose (LD 50) has been reported as 88 mg/kg (3.5 millions IU/kg). However, in humans, the mean lethal dose (LD50) of vitamin D has been reported as 21 mg/kg (840,000 IU/kg)[8,9].

Vitamin D Supplementation and Risk of Toxicity in Pediatrics: A Review of Current Literature

Maria G. Vogiatzi, Elka Jacobson-Dickman, Mark D. DeBoer, for the Drugs, and Therapeutics Committee of The Pediatric Endocrine Society

Weill Cornell Medical College (M.G.V.), New York, New York 10065; SUNY Downstate Medical Center (E.J.-D.), Brooklyn, New York 11203; and University of Virginia Health System (M.D.D.), Charlottesville, Virginia 22908

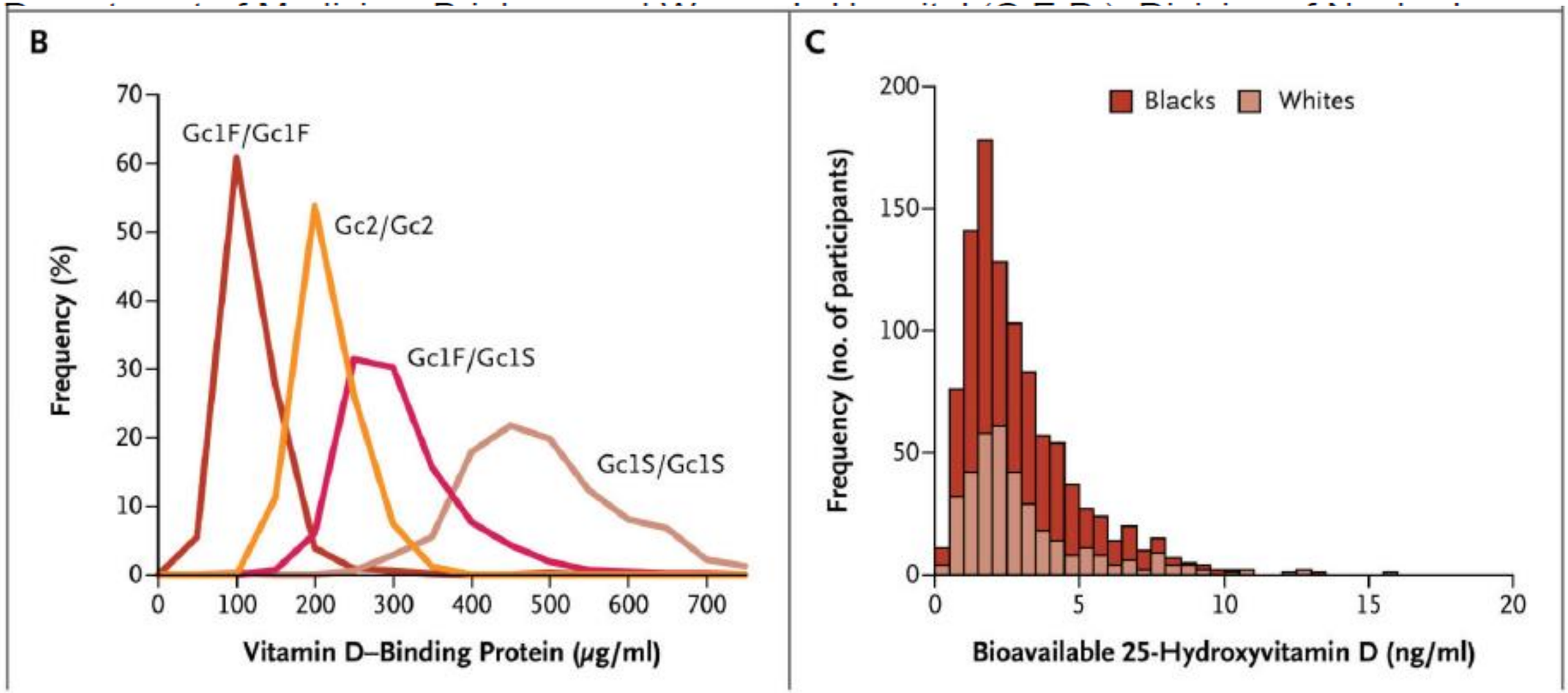




N Engl J Med. 2013 November 21; 369(21): 1991–2000. doi:10.1056/NEJMoa1306357.

Vitamin D–Binding Protein and Vitamin D Status of Black Americans and White Americans

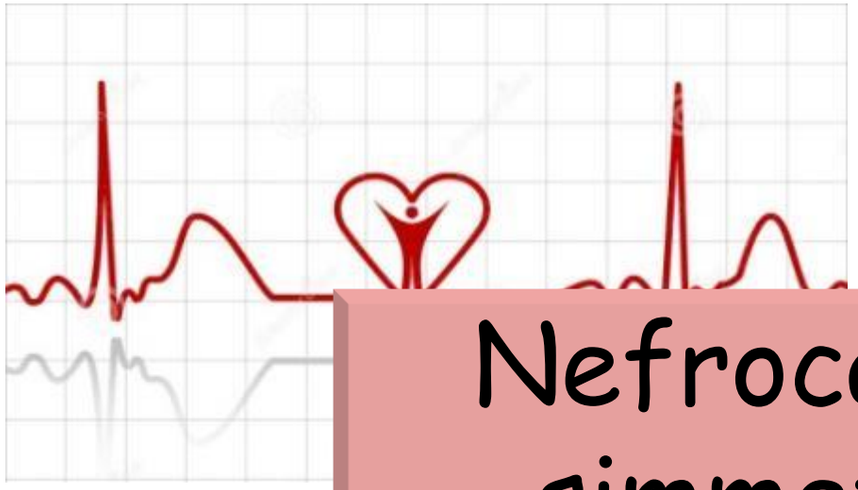
Camille E. Powe, M.D., Michele K. Evans, M.D., Julia Wenger, M.P.H., Alan B. Zonderman, Ph.D., Anders H. Berg, M.D., Ph.D., Michael Nalls, Ph.D., Hector Tamez, M.D., M.P.H., Dongsheng Zhang, Ph.D., Ishir Bhan, M.D., M.P.H., S. Ananth Karumanchi, M.D., Neil R. Powe, M.D., M.P.H., M.B.A., and **Ravi Thadhani, M.D., M.P.H.**



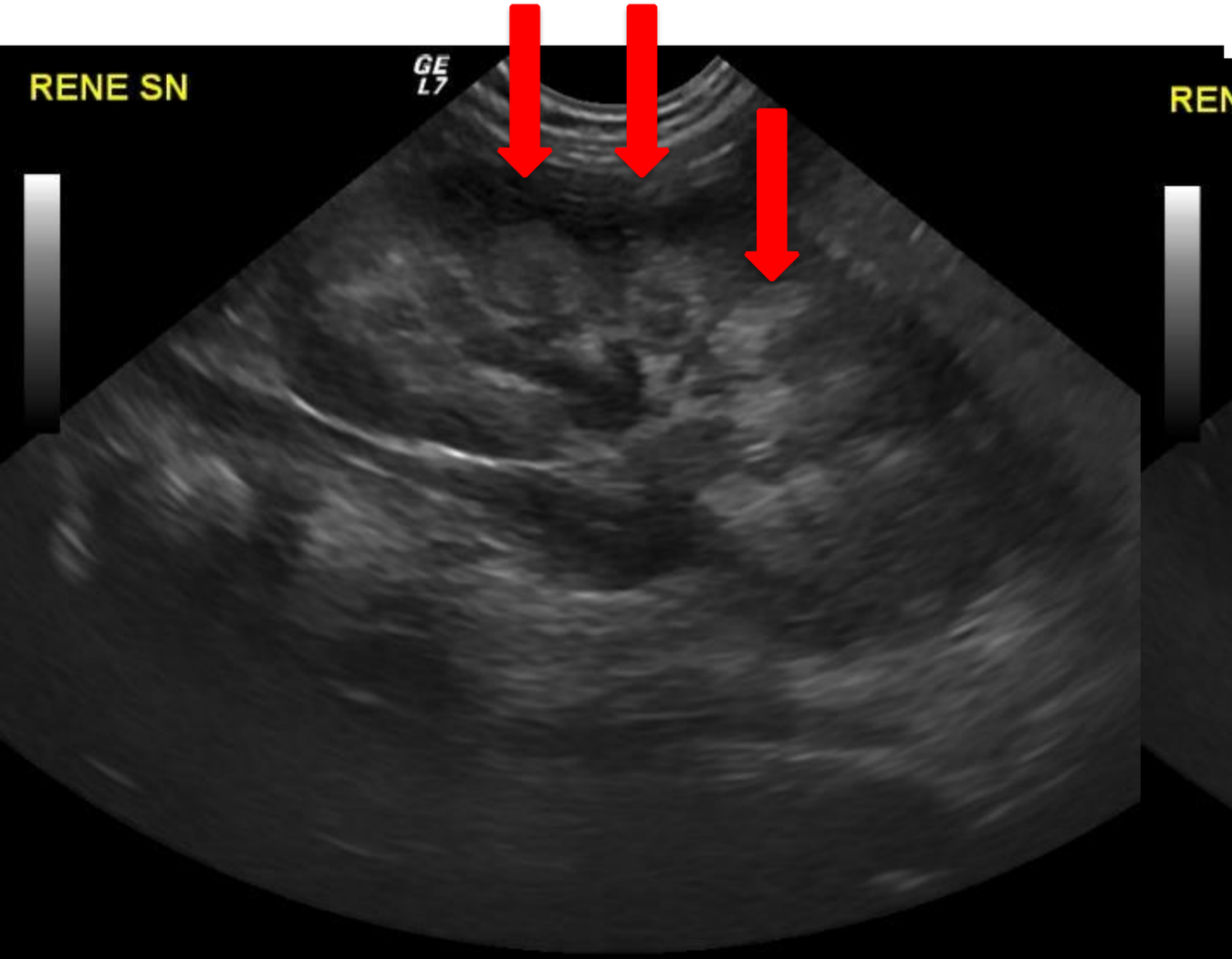
Vitamin D binding protein polymorphic variants play an important role in vitamin D levels and effects.

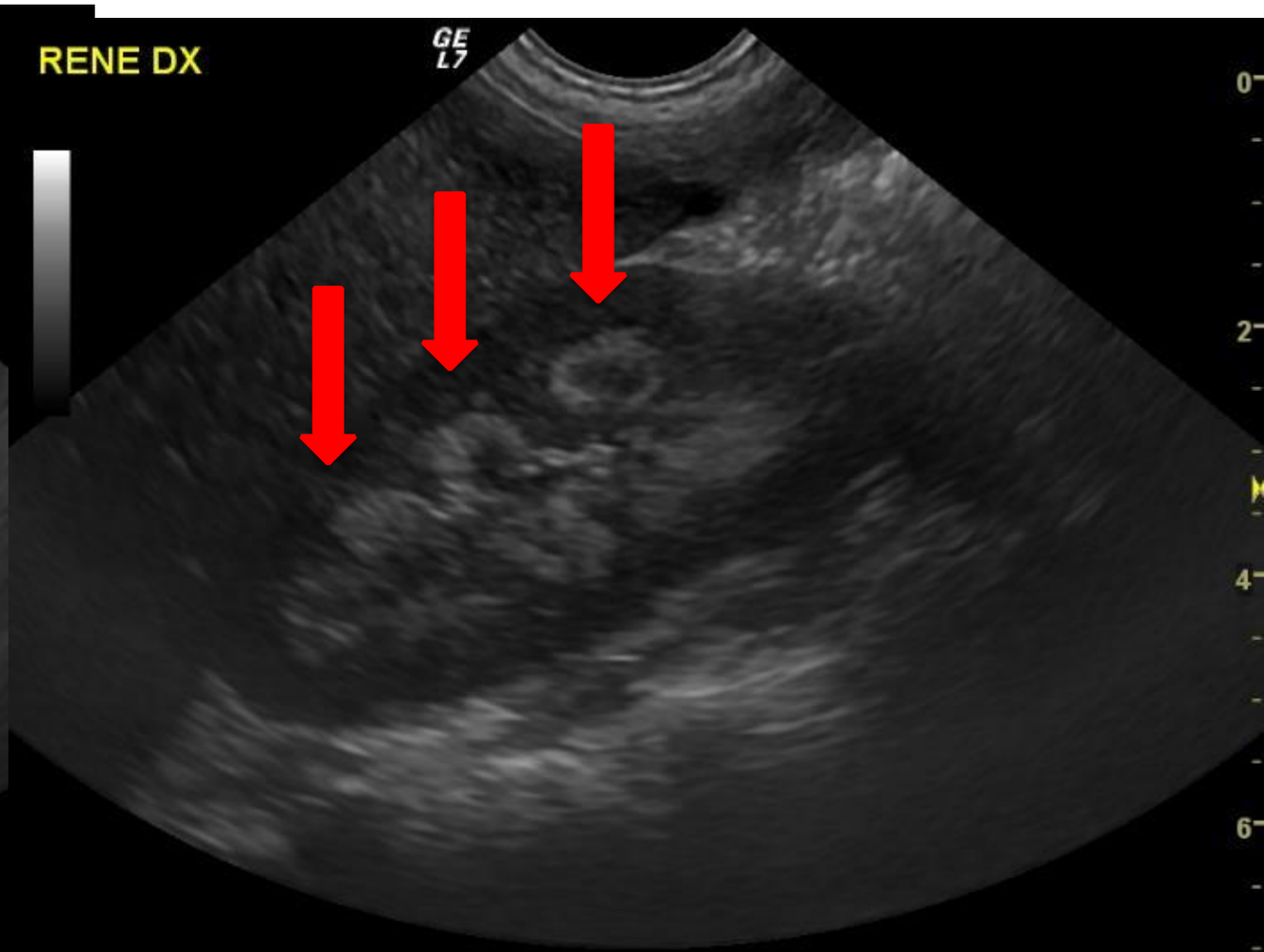
Table II. Symptoms and Findings Associated with Hypercalcemia due to Vitamin D Intoxication [15,16]

Gastrointestinal	- Nausea and vomiting
	- Anorexia, abdominal pain
	- Intestinal decreased motility, constipation
	- Growth retardation, pancreatitis, peptic ulcer
Renal	- Polydipsia, polyuria, dehydration and fever
	- Hematuria, hypernatremia, hypomagnesemia, hypokalemia
	- Nephrolithiasis, nephrocalcinosis, distal renal tubular acidosis
	- Nephrogenic diabetes insipidus, chronic interstitial nephritis
	- Acute and chronic renal failure
Central nervous system	- Hypotonia, paresthesia
	- Deep tendon reflexes reduction, headache
	- Confusion, seizures, cerebral vasospasm
	- Mesial temporal sclerosis, apathy, lethargy, stupor, coma
	- Psychiatric disorders (anxiety, psychosis, hallucination, depression)
Cardiovascular	- Arrhythmia, bradycardia (QT interval shortening, QRS widening, PR elongation, ST elevation, T- wave and U- wave widening)
	- Heart valves, coronary arteries and myocardial fibers-accumulation of calcium
	- Hypertension
	- Cardiomyopathy
	- Cardiac arrest
Musculoskeletal	- Muscle weakness
	- Bone pain
	- Osteopenia/osteoporosis
	- Long bones metastatic calcification
	- Osteopetrosis
Eyes	- Band keratopathy
	- Conjunctival calcification
Skin	- Metastatic calcification
	- Itching



Nefrocalcinosi midollare simmetrica bilaterale





urinary Ca/creatinine ratio: 0,8 mg/mg (N<0.21)

Terapia

- Idratazione endovenosa
- Diuretici dell'ansa
- glucocorticoidi
- Calcitonina
- bifosfonati

Terapia

Idratazione endovenosa

soluzione fisiologica 150 ml/Kg/die

Idratazion

Diure

e a 1-2 mg/kg/die,
ogni 4-6 ore

Terapia

Idratazione endovenosa

Diuretici dell'ansa

glucocorticoidi

Prednisone 1-2 mg/kg/die

Terapia

Idratazione endovenosa

diuretici dell'ansa

glucocorticoidi

Calcitonina

1-2 UI/Kg/dose, in 2-4 dosi

Terapia

- Idratazione endovenosa
- Diuretici dell'ansa
- glucocorticoidi
- Calcitonina
- bisosfonati

pamidronate 0.5-1 mg/kg/dose

DE GRUYTER J Pediatr Endocr Met 2015; aop

Patient report

Elif Sagsak*, Senay Savas-Erdeve, Meliksah Keskin, Semra Cetinkaya and Zehra Aycan

The use of pamidronate for acute vitamin D intoxication, clinical experience with three cases

costoso

Severi effetti collaterali

In 2003, **alendonate** per os è stato usato per la prima volta in un bambino per il trattamento dell'ipercalcemia da intossicazione da Vitamina D
(partendo da una dose di 5 mg/die e incrementando a 10 mg/die)

Acta Paediatr. 2012 Mar;101(3):e122-5. doi: 10.1111/j.1651-2227.2011.02497.x. Epub 2011 Nov 4.

Comparison of oral alendronate versus prednisolone in treatment of infants with vitamin D intoxication.

Sezer RG[1], Guran T, Paketçi C, Seren LP, Bozaykut A, Bereket A.

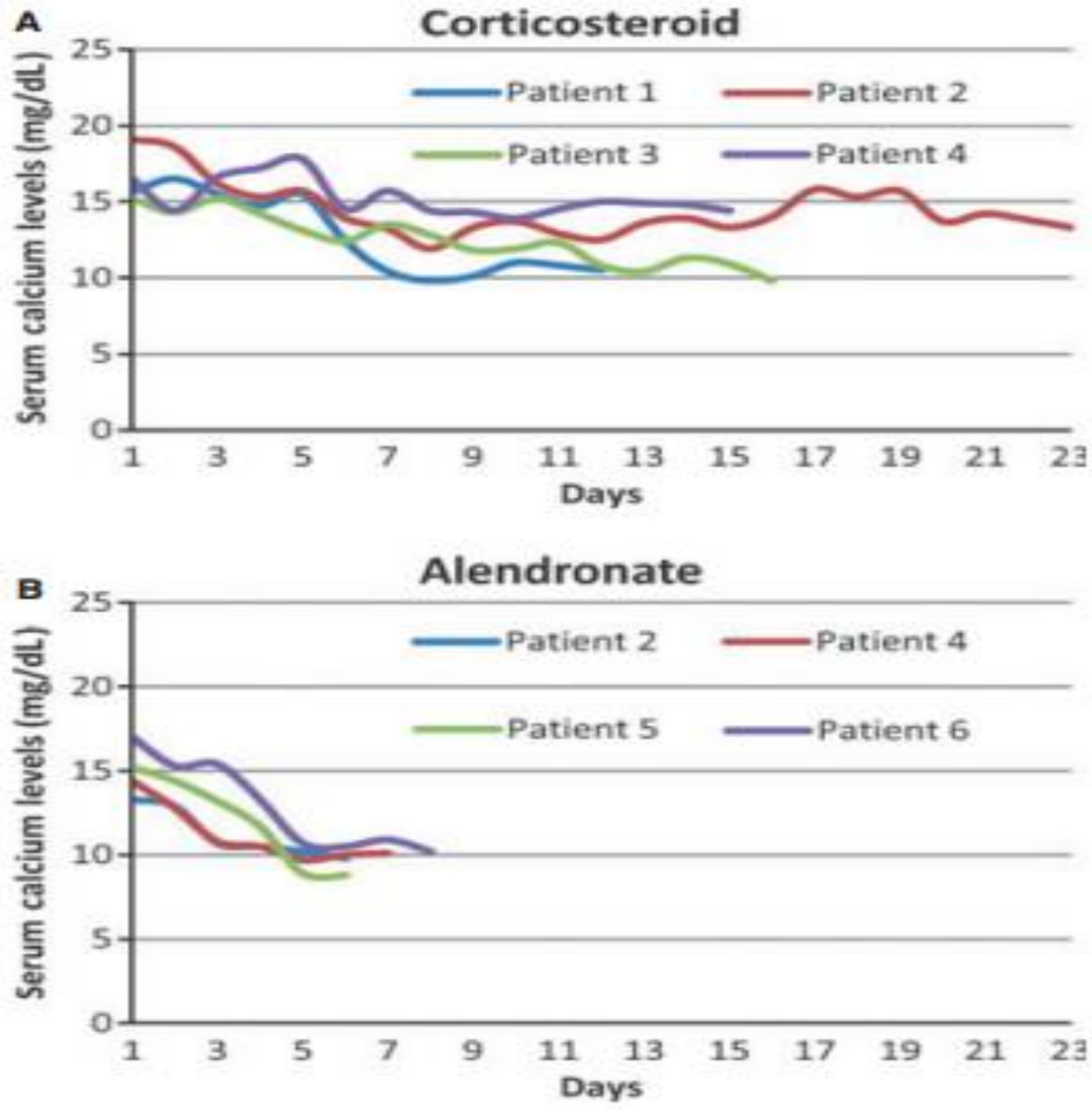


Figure 1 (A) Serum calcium levels of patients on corticosteroid treatment. (B) Serum calcium levels of patients after single oral dose of alendronate (Patient 2 and 4 were switched to alendronate treatment after 23 and 15 days of corticosteroid treatment, respectively).

Take home messages

La terapia con Vitamina D se effettuata con criterio ed attenzione, seguendo le indicazioni delle linee guida, risulta efficace e sicura

Bisogna pore attenzione a tutte le terapie effettuate dai bambini, senza sottovalutare integratori alimentari e/o farmaci complementari

Bisogna essere sicuri che i genitori abbiano capito correttamente la prescrizione,
per evitare eccessive assunzione

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2° caso clinico

Ragazzo di 16 anni giunge alla nostra osservazione per la presenza di paralisi faciale

Fronte con poche rughe

dal lato affetto
l'occhio viene chiuso
parzialmente per la paresi
del muscolo orbicolare.

Fenomeno di Bell caratterizzato
dalla rotazione del bulbo oculare
verso l'alto nel tentativo
non riuscito di chiudere l'occhio

lato affe
stirato e
l'aspetto a

Paralisi periferica faciale

l'emirima dal lato affetto
risulta essere più bassa
della controlaterale

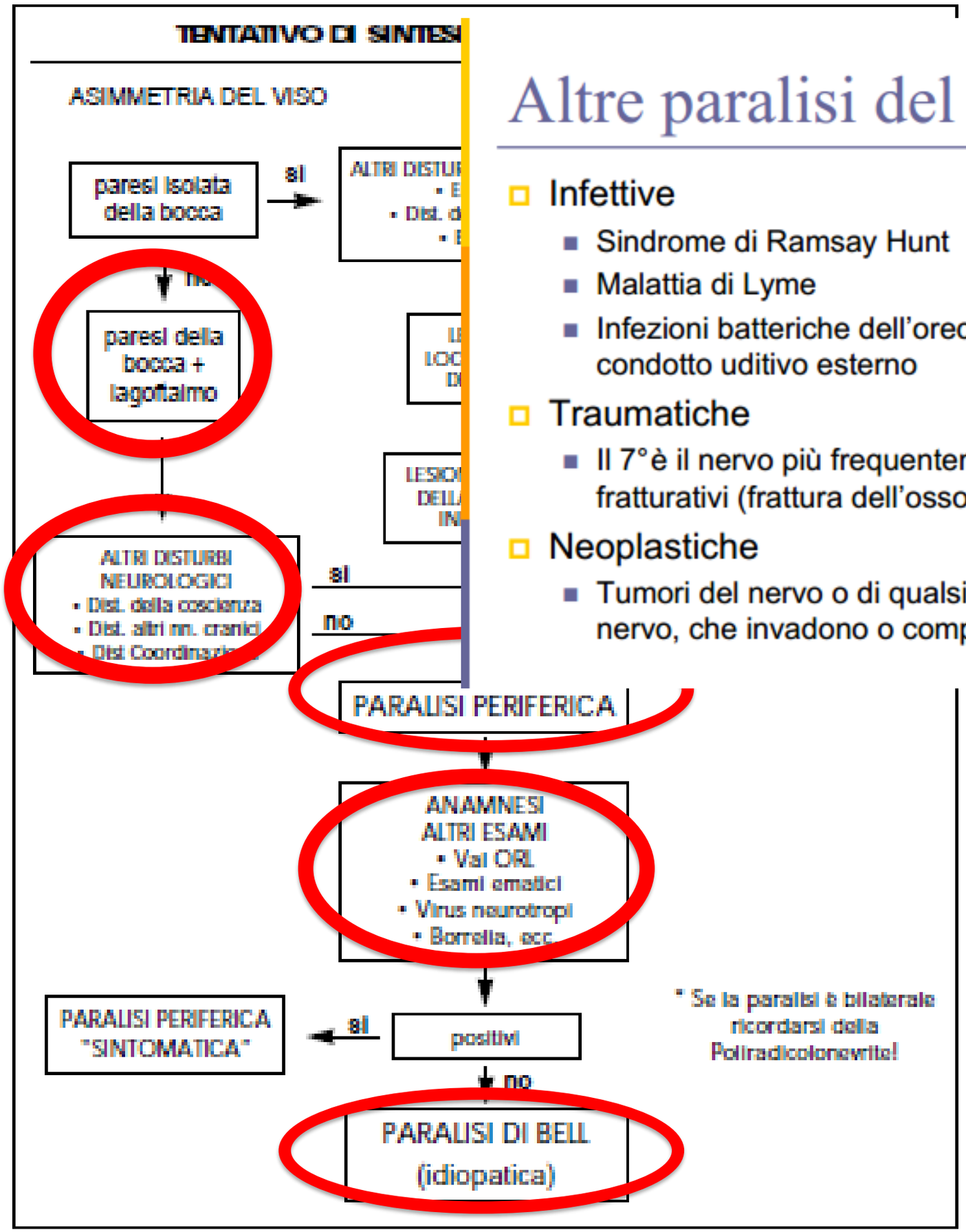


Figura 3

Altre paralisi del facciale

- Infettive
  - Sindrome di Ramsay Hunt
  - Malattia di Lyme
  - Infezioni batteriche dell'orecchio medio, della mastoide o del condotto uditivo esterno
- Traumatiche
  - Il 7° è il nervo più frequentemente coinvolto nei trau mi cranici fratturativi (frattura dell'osso temporale)
- Neoplastiche
  - Tumori del nervo o di qualsiasi struttura, lungo il decorso del nervo, che invadono o comprimono il nervo

ESAMI DI LABORATORIO

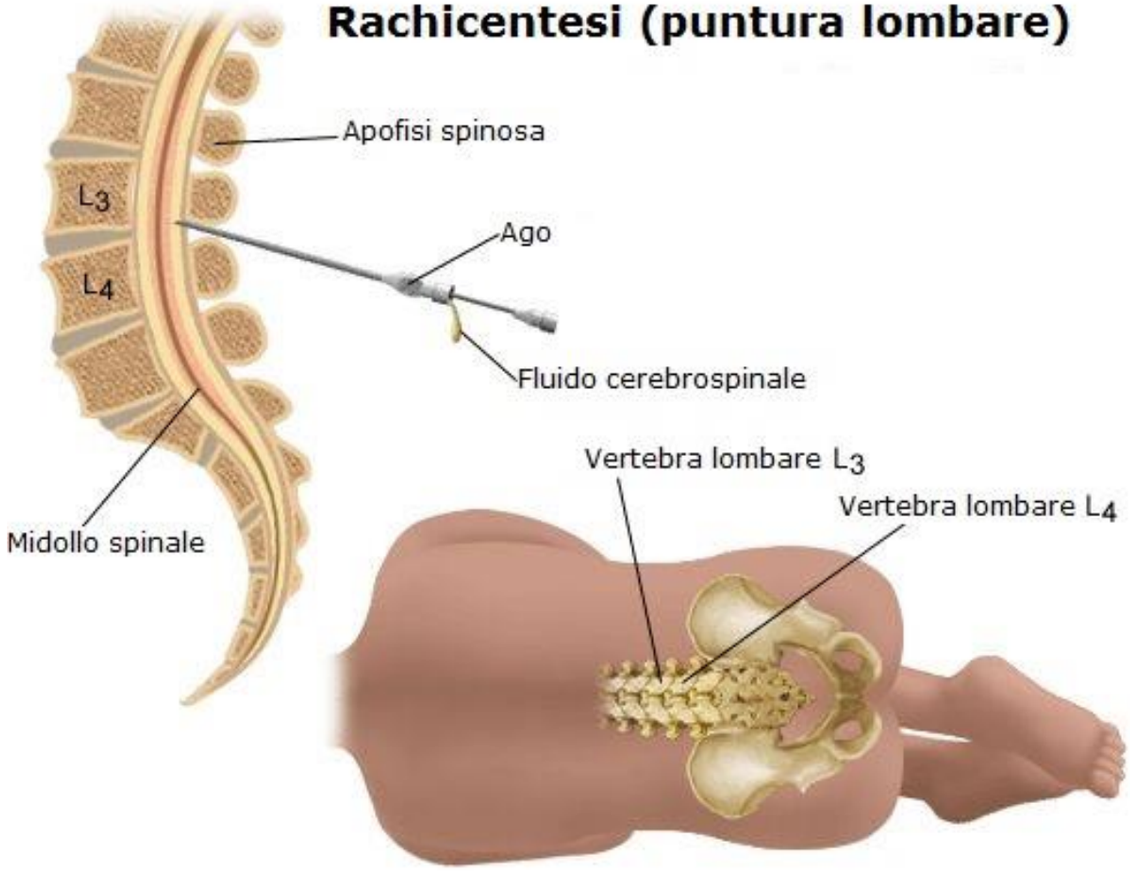
Emocromo: GR: 5.820.000/mmc, Hb: 13,7 g% ml, Hct: 38%, GB: 12.600/mmc)

Funzionalità epatica e renale nella norma

Borrellia, Virus neurotropi, ecc:negativi

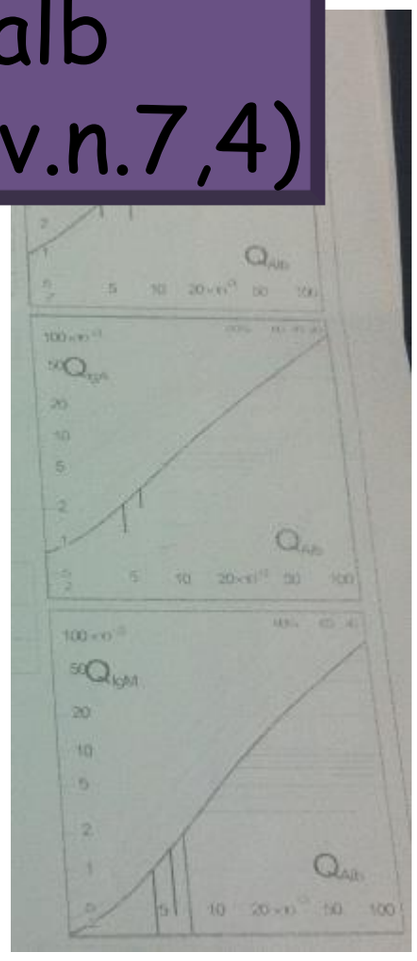
Emerge che il ragazzo da alcuni giorni
ori e

alte vie



- Dissociazione albumino-citologica

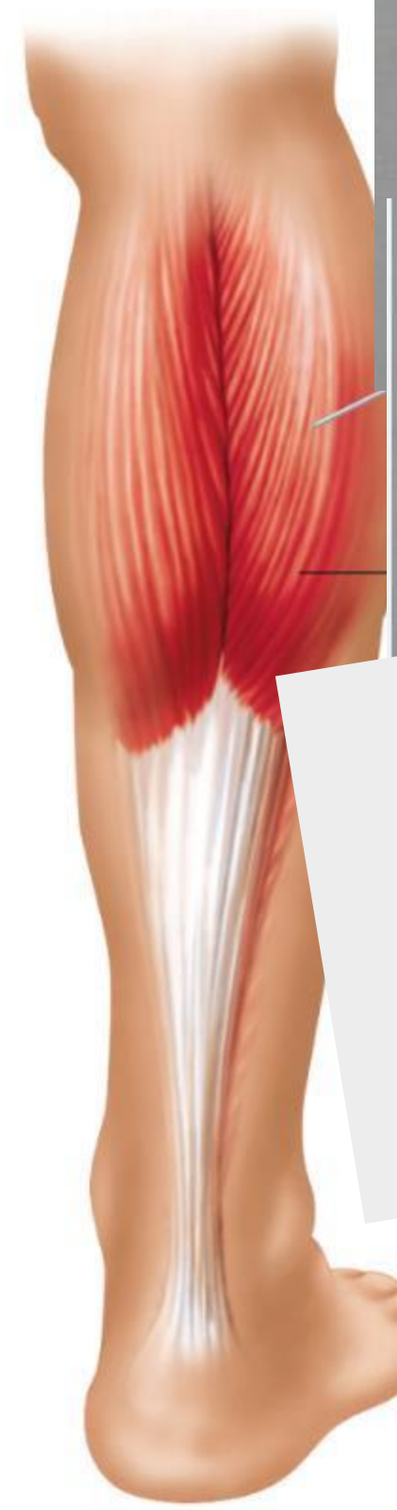
- Rapporto alb liquor/siero 19 (v.n.7,4)



MICROBIOLOGIA

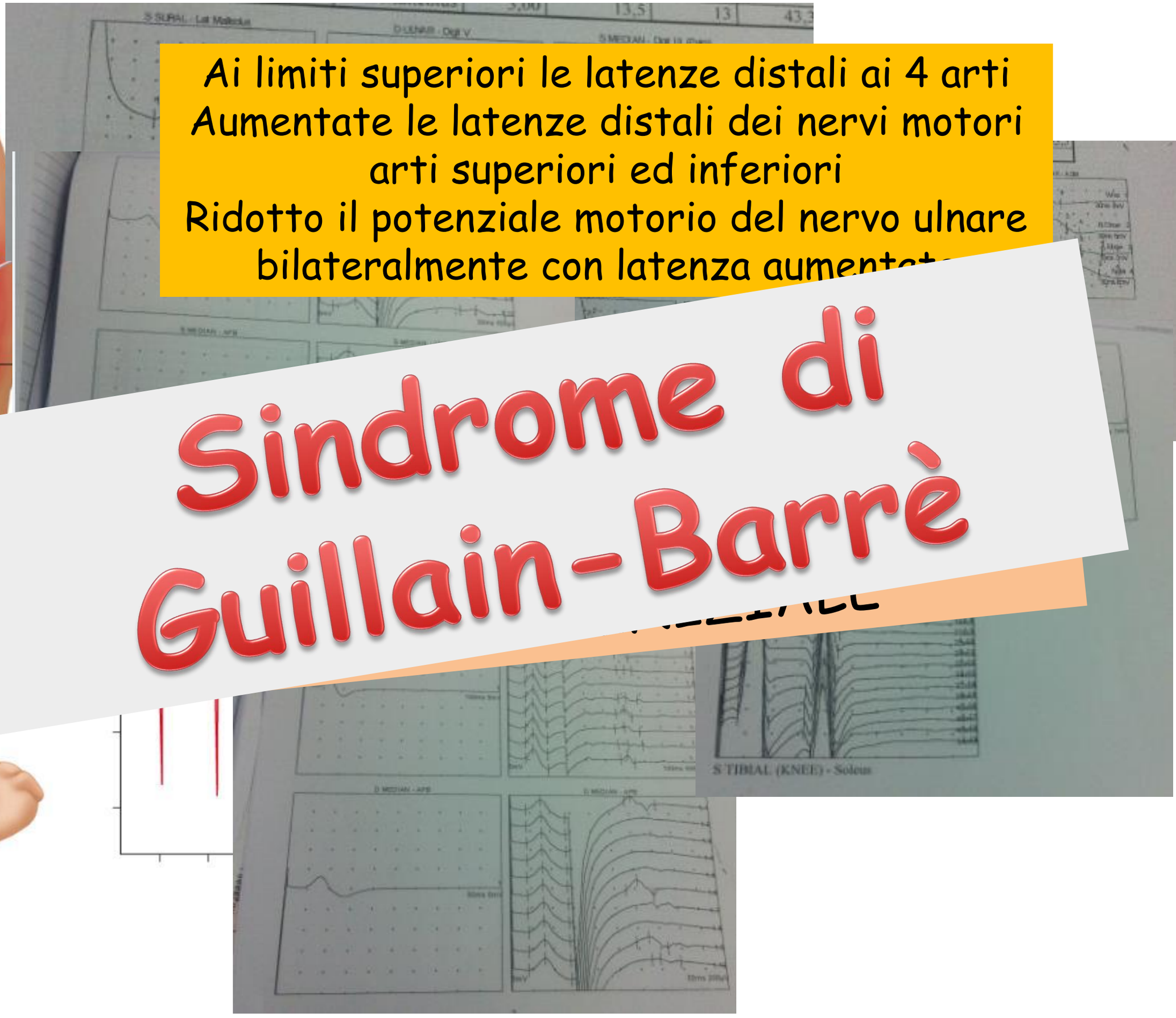
INQUADRAMENTO DIAGNOSTICO LIQUOR

- ES. FISICO: (Met. ottico)	Limpido, incolore e trasparente		
- GLUCOSIO su LIQUOR: (Met. enzimatico)	63	mg/dl	(40 - 70)
- GLUCOSIO su SIERO: (Met. enzimatico)	86	mg/dl	(70 - 100)
- ALBUMINA su LIQUOR: (Met. nefelometrico)	1140	mg/l	(0 - 350)
- ALBUMINA su SIERO: (Met. nefelometrico)	60	g/L	(35 - 60)
- IgG su LIQUOR: (Met. nefelometrico)	177	mg/l	(0 - 34)
- IgG su SIERO: (Met. nefelometrico)	14	g/L	(7 - 16)
- CONTA CELLULE AL CONTAGLOBULI: (Contaglobuli: citofluorimetria in fluorescenza)	0	cellule/ul	POSITIVO > 25 cellule/ul



Ai limiti superiori le latenze distali ai 4 arti
Aumentate le latenze distali dei nervi motori arti superiori ed inferiori
Ridotto il potenziale motorio del nervo ulnare bilateralmente con latenza aumentat

Sindrome di Guillain-Barré



Neurology OPEN ACCESS Research Article

Guillain-Barré syndrome: clinical profile and management

Sindrome di Landry-Guillain-Barré-Strohl

una poliradicoloneuropatia infiammatoria demielinizzante a decorso acuto e/o subacuto, caratterizzata da infiltrati linfocitari e macrofagici a livello dei nervi periferici e delle radici nervose e da distruzione della mielina

1 a 4 soggetti su 100.000 tra adulti e bambini/anno

Età infantile: soprattutto fra i 4 e i 9 anni di età e rara sotto i 2 anni

Tabella I Le poliradicoloneuropatie

POLIRADICOLONEUROPATIE IMMUNOMEDIATE

- Poliradicoloneuropatie acute
  - Sindrome di Guillain-Barré
  - Sindrome di Miller Fisher
  - Neuropatia assonale acuta motoria
  - Neuropatia assonale acuta sensitivo-motoria
- Poliradicoloneuropatie croniche
- Polineuropatie in corso di malattie autoimmuni sistemiche
  - Lupus eritematoso sistemico
  - Sindrome di Sjögren
  - Malattia celiaca
- Polineuropatie in corso di vasculiti
  - Panarterite nodosa
  - Porpora di Schonlein-Henoch
- Polineuropatie paraneoplastiche

POLINEUROPATIE IN CORSO DI MALATTIE SISTEMICHE

- Diabete mellito
- Disfunzioni tiroidee
- Porfiria
- Crioglobulinemia
- Amiloidosi
- Gammapatie monoclonali

POLINEUROPATIE CARENZIALI

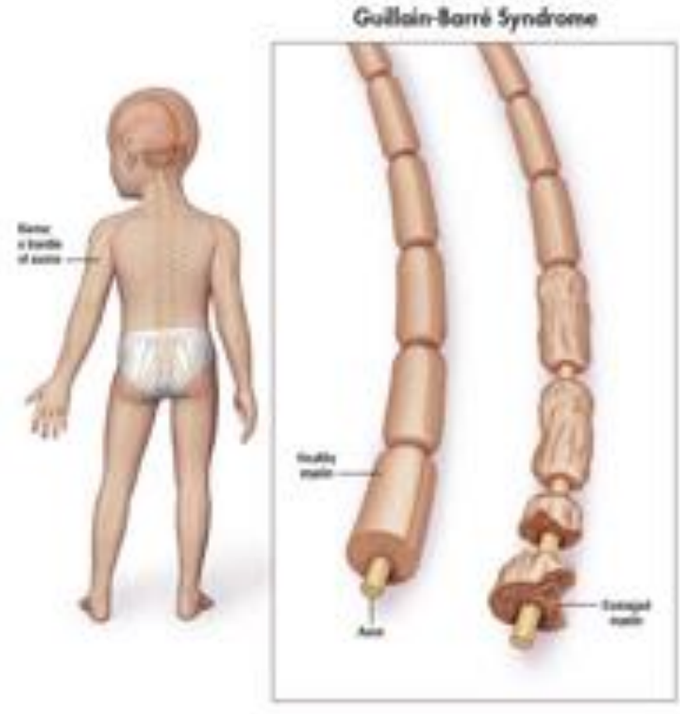
- Deficit di vitamina B6
- Deficit di vitamina B12
- Deficit di acido folico

POLINEUROPATIE IATROGENE E TOSSICHE

Neurology OPEN ACCESS Research Article

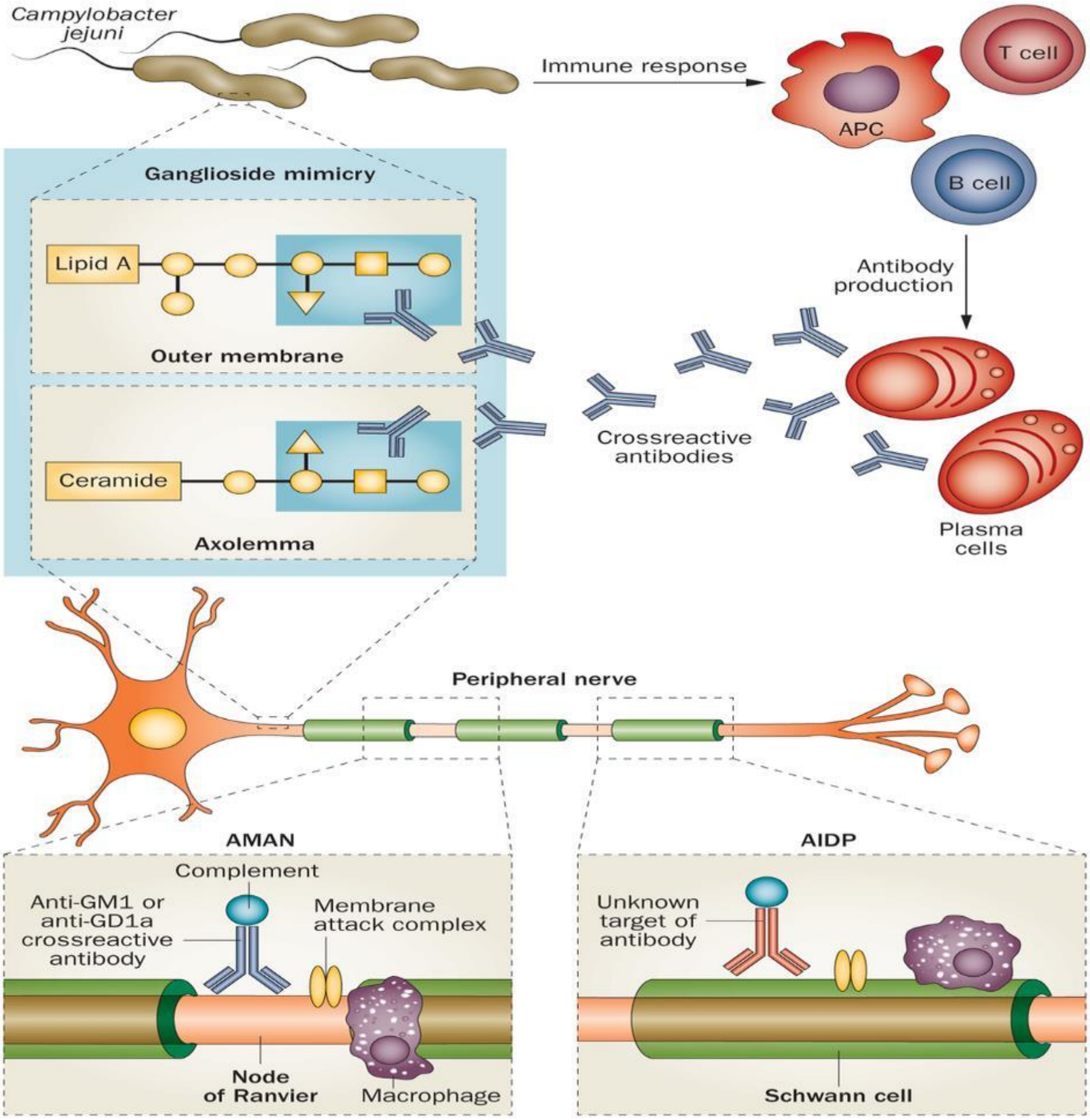
Guillain-Barré syndrome: clinical profile and management

Etiology of GBS is not completely understood but believed to be due to autoimmune cause where majority of cases are triggered by infection stimulating anti-ganglioside antibodies production. Approximately 70% of cases of GBS occur 1–3 weeks after an acute infectious process. The organisms thought to be involved are *Campylobacter jejuni* (diarrhea), *Mycoplasma pneumonia*, *Haemophilus influenzae*, cytomegalovirus, Epstein-Barr virus and influenza [13]. Administration of outmoded anti-rabies vac-



Etiology

- *Campylobacter jejuni, Helicobacter pylori, Mycoplasma pneumoniae*
- West Nile virus,Epstein-Barr Virus,Echo Virus,Coxsakie Virus,Influenza Virus
- Mumps,Measles
- Vaccines against rabies,influenza, poliomyelitis (oral), conjugated meningococcal vaccine particularly serogroup C and recently with H1N1.
- Some Surgery


Campylobacter jejuni
Immune response
T cell
APC
B cell
Antibody production
Ganglioside mimicry
Lipid A
Outer membrane
Ceramide
Axolemma
Crossreactive antibodies
Plasma cells
Peripheral nerve
AMAN
Complement
Anti-GM1 or anti-GD1a crossreactive antibody
Membrane attack complex
Node of Ranvier
Macrophage
AIDP
Unknown target of antibody
Schwann cell

Risk Factors:
- Possibly Autoimmune
- Association with Immunizations
- Frequently preceded

GUILLAIN-BARRE' SYNDROME

Original Article

Clinical Profile of Guillain Barre Syndrome

Shubhangi Vithal Dhadke[1], Vithal Narayan Dhadke[2], Sachin S Bangar[1], Milind B Korade[3]

in 40-70% of the patients the first neurological symptoms were tingling, numbness, paraesthesia or pain in the toes

Atrophy

...metrical Paralysis

Causes Problems With:
- Respiration
- Talking
- Swallowing
- Bowel & Bladder Function

Begins in lower extremities and ascends bilaterally =
1) Weakness
2) Ataxia
3) Bilateral Paresthesia Progressing to Paralysis.

C.J. MILLER

©2007 Nursing Education Consultants, Inc.

paralisi ***ascendente*** ***bilaterale***

ORIGINAL ARTICLE

Atyp

How to cite this art
Findin

Children

Ghofrani M. Atypical
4):17-22.

Discussion

We have reported eight
of the total patients who
symptoms of GBS as upp
(3%), neck stiffness (3%)
weakness) (9.1%), headac
Jin Park et al. reported an 1
GBS presentation of ge
ophthalmoplegia (visual d
postural dizziness; The cu
the symptoms of a 10-ye
with ptosis and weakness

Paralisi faciale??

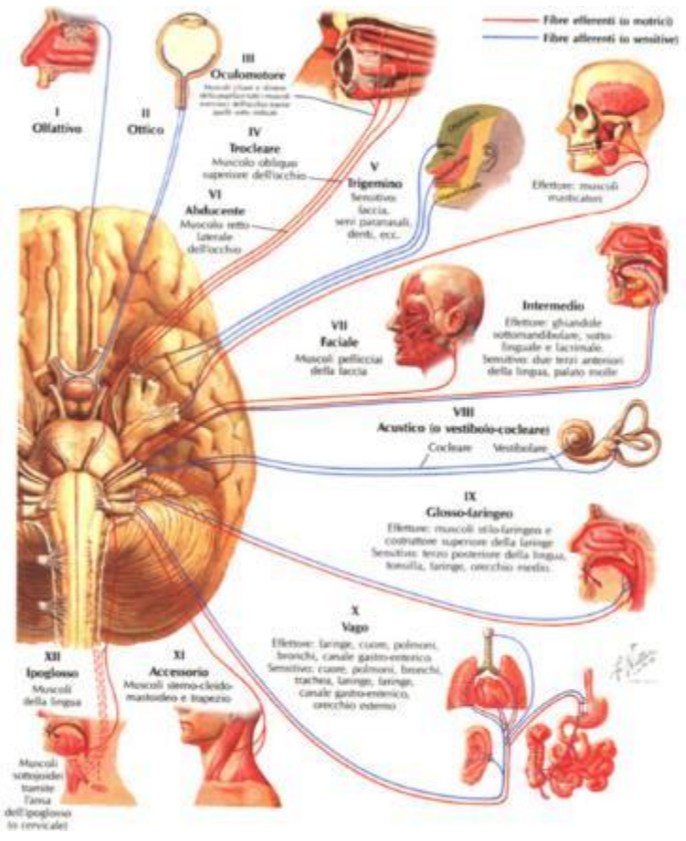
Original Article

Guillain-Barré Syndrome: A Clinical Study of Twenty Children

Journal of Clinical and Diagnostic Research. 2015 Jan, Vol-9(1): SC09-SC12

I. Features required for diagnosis
(A) Progressive motor weakness of more than one limb
(B) Loss of tendon jerks
II. Features strongly supportive of the diagnosis
(A) Clinical features
1. Progression over four weeks
2. Relative symmetry of weakness
3. Mild sensory symptoms or signs
4. Cranial nerve involvement
5. Recovery, usually beginning two to four weeks after progression stops
6. Autonomic dysfunction
7. Absence of fever at the onset of neuritic symptoms
(B) CSF* features
1. CSF protein raised after the first week of symptoms
2. Counts of 10 or fewer mononuclear leucocytes x 10^6/1
(C) Electrodiagnostic features
Reduction of conduction velocity, conduction block or abnormal temporal dispersion, increased distal latency or abnormal F wave in more than one nerve

[Table/Fig-1]: Diagnostic criteria for Guillain-Barre syndrome after Asbury and Cornblath [5]
*CSF=cerebrospinal fluid



Original Article

Clinical Profile of Guillain Barre Syndrome

Shubhangi Vithal Dhadke[1], Vithal Narayan Dhadke[2], Sachin S Bangar[1], Milind B Korade[3]

Clinical Features	Male	Female	Total
Quadriparesis	25	14	39
Paraparesis	1	0	1
Cranial Involvement	16	9	25
Bladder Involvement	0	0	0
Sensory Symptom	7	6	13
Sensory Signs	0	0	0
Atypical presentation (Descending Paralysis)	0	0	0
Respiratory Paralysis	7	6	13
Fisher Variant	0	0	0

Table 5 : Cranial nerve involvement

Cranial Nerve	Male	Female	Total
Occulomotor	1	0	1
Facial-Unilateral	0	1	1
Facial-Bilateral	8	3	11
Glossopharyngeal, Vagus	7	5	12
Spinal Accessory	0	0	0
Hypoglossal	0	0	0
Total	16	9	25

LETTER TO EDITOR

Guillain-Barre Syndrome Presenting With Bilateral Facial Nerve Palsy

How to Cite This Article: Inaloo S, Katibeh P. Guillain-Barre Syndrome Presenting With Bilateral Facial Nerve Palsy. Iran J Child Neurol 2014 Winter; 8(1):70-72.

PubMed ▾ guillain barre and unilateral facial palsy

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10 years
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Species
Humans
Other Animals

Ages
Child: birth-18 years
Infant: birth-23 months
Adult
Adult: 19-44 years
Aged: 65+ years
Customize ...

Search results

Items: 5

Filters activated: Child: birth-18 years. Clear all to show 16 items.

1. Delayed **facial** weakness in **Guillain**-Barré and Miller Fisher syndromes.
Tatsumoto M, Misawa S, Kokubun N, Sekiguchi Y, Hirata K, Kuwabara S, Yuki N.
Muscle Nerve. 2015 Jun;51(6):811-4. doi: 10.1002/mus.24475. Epub 2015 Jan 9.
PMID: 25287079
Similar articles

2. Acute motor-sensory axonal **Guillain**-Barré syndrome with **unilateral facial** nerve **paralysis** after rotavirus gastroenteritis in a 2-year-old boy.
Kamihiro N, Higashigawa M, Yamamoto T, Yoshino A, Sakata K, Nashida Y, Maji T, Fujiwara T, Inoue M.
J Infect Chemother. 2012 Feb;18(1):119-23. doi: 10.1007/s10156-011-0300-8. Epub 2011 Sep 14.
PMID: 21915637
Similar articles

Acquired **facial palsy** with hypertension secondary to **Guillain-Barre** syndrome

Items: 16

BMJ Case Reports

Unusual presentation of more common disease/injury

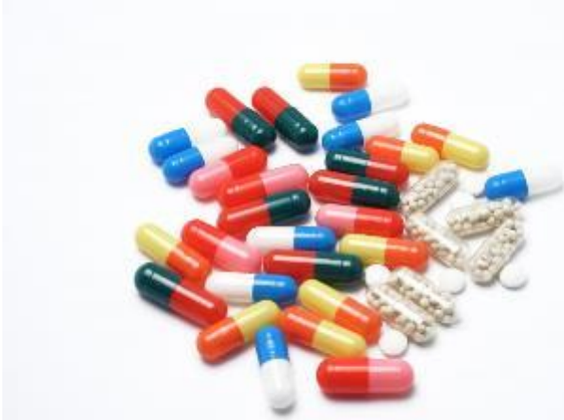
Unilateral facial palsy in Guillain-Barre syndrome (GBS): a rare occurrence

Rajesh Verma, Tejendra S Chaudhari, Prithvi Giri

As aforementioned, facial palsy in GBS is usually bilateral, but, it maybe asymmetrical and uncommonly unilateral involvement may be seen.[2] The facial nerve palsy in GBS is secondary to direct attack of antibodies either causing demyelination or axonal degeneration depending on the type of antibody involved. However, hypertension which is commonly seen in GBS due to autonomic disturbances, may also contribute to facial paralysis.[2] Although exact mechanism of hypertension causing facial palsy is poorly understood, it has been proposed that it could be because of edema or haemorrhage within the facial canal causing neural compression. This is especially true in paediatric population.[6] In a review of 35 children with severe hypertension, Lloyd *et al*[6] found that LMN-type facial palsy occurred in seven patients (20%).

Tab I. Classe di evidenza e livello di raccomandazione delle IVIG nelle malattie neurologiche del bambino.

Malattia		Classe di evidenza	Livello di raccomandazione
GBS		II	A
CIDP		III	B
JMG	crisi miastenica	II	B
	Th a lungo termine	IV	C
JDM		III	C
Opsoclono-mioclono		IV	C
Encefalopatia di Rassmussen		IV	C
Epilessia intrattabile del bambino		IV	C
Encefalite anti NMDA-R		IV	C
ADEM, SM/NO		IV	C



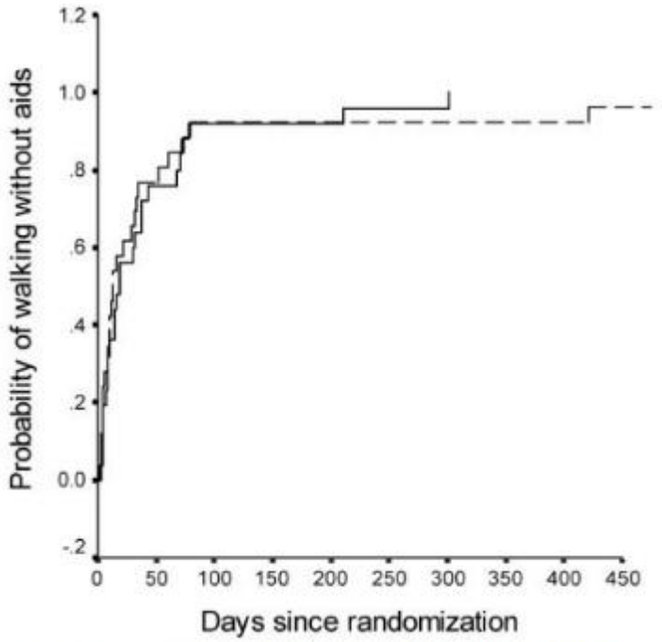


Fig 1. Time to recovery of ability to walk without aids. The solid line indicates 1 g/kg IVIG over 2 days; the dashed line indicates 0.4 g/kg IVIG over 5 days. In the Kaplan-Meier analysis with the log-rank test, the difference between groups was not significant (P = .94).

Intravenously Administered Immunoglobulin in the Treatment of Childhood Guillain-Barré Syndrome: A Randomized Trial

Rudolf Korinthenberg, MD*; Joachim Schessl, MD*; Janbernd Kirschner, MD*; and

Pediatrics International (2003) **45**, 543–549

Original Article

High-dose immunoglobulin therapy for Guillain–Barré syndrome in Japanese children

STUDY GROUP FOR PEDIATRIC GUILLAIN–BARRÉ SYNDROME
Participants in this study and their institutions are listed in Appendix 1

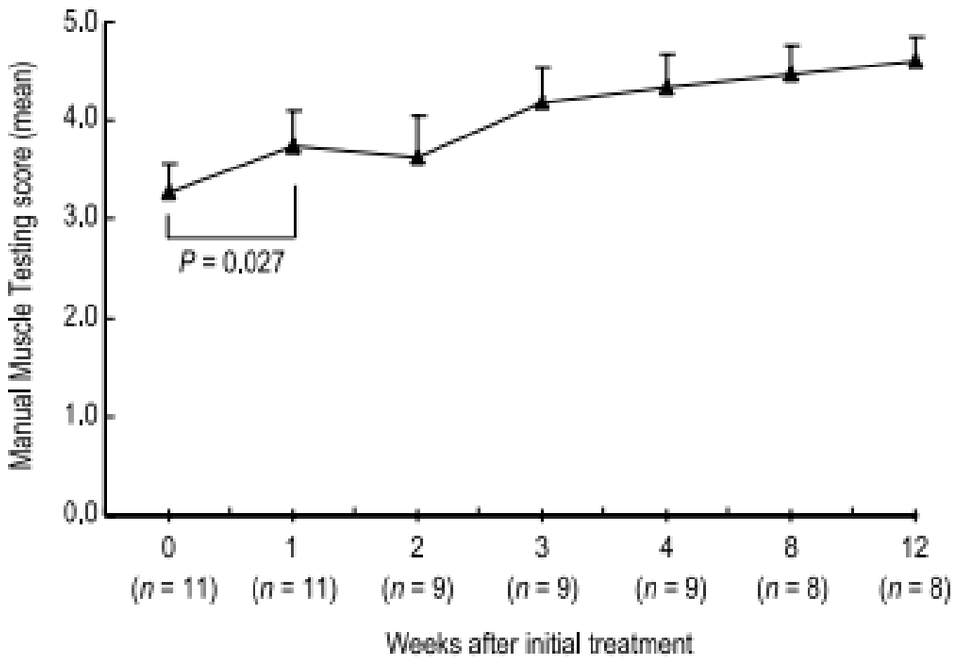


Fig. 3 Graph showing changes in Manual Muscle Testing (MMT) score with time after intravenous immunoglobulin (IVIg) treatment.

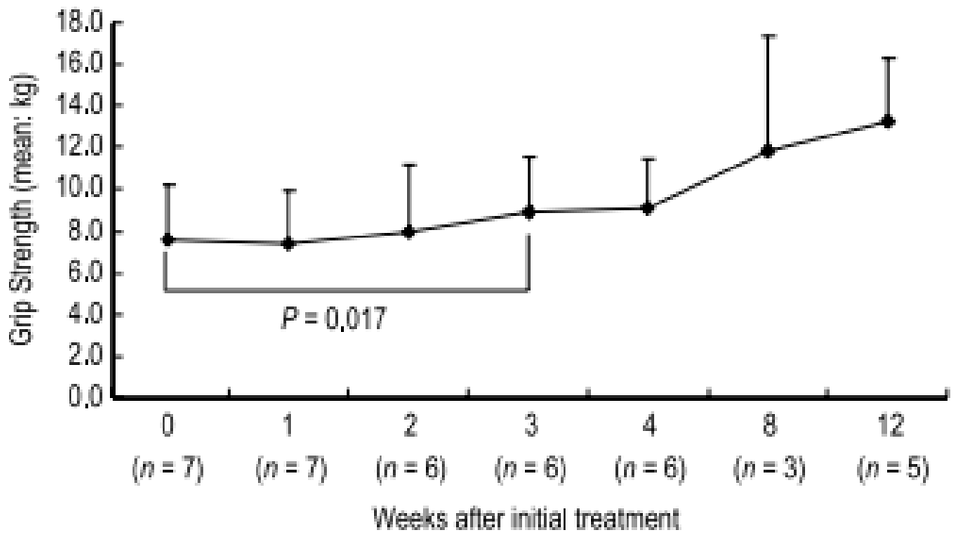


Fig. 4 Graph showing changes in grip strength over time after intravenous immunoglobulin (IVIg) treatment.

Il 73% dei pazienti presentava un miglioramento di almeno un grado della scala di disabilità dopo una settimana dall'inizio del trattamento con IVIG. Dopo 4 settimane dal trattamento il miglioramento era presente nell'82% dei pazienti

Take home messages

Sebbene la diagnosi di paralisi faciale periferica sia molto semplice, ricordarsi che di base c'è un insulto neurologico da non sottovalutare

Non sempre la Sindrome di Guillain Barrè, si manifesta con la sintomatologia classica, ma vi sono forme atipiche o parziali

7th
JOINT MEETING
In pediatria e medicina dell'adolescenza
Sobre pediatría y medicina de la adolescencia
21-22-23-24 OTTOBRE 2015 CATANZARO ITALIA

3° caso clinico

Bimba di 5 anni, di madre ucraina e padre italiano, veniva condotta presso il nostro dipartimento per dolore addominale insorto da due giorni.

ANAMNESI FAMILIARE E PATOLOGICA REMOTA: negative.

carbone attivo

Dopo 24 h il quadro cambia…

-abbattimento
- Peggioramento del dolore addominale

-no f

-Obie disteso,

dolen

-Scor

-PA =

Il dolore addominale era emerso dopo l'assunzione di un pasto inusualmente abbondante

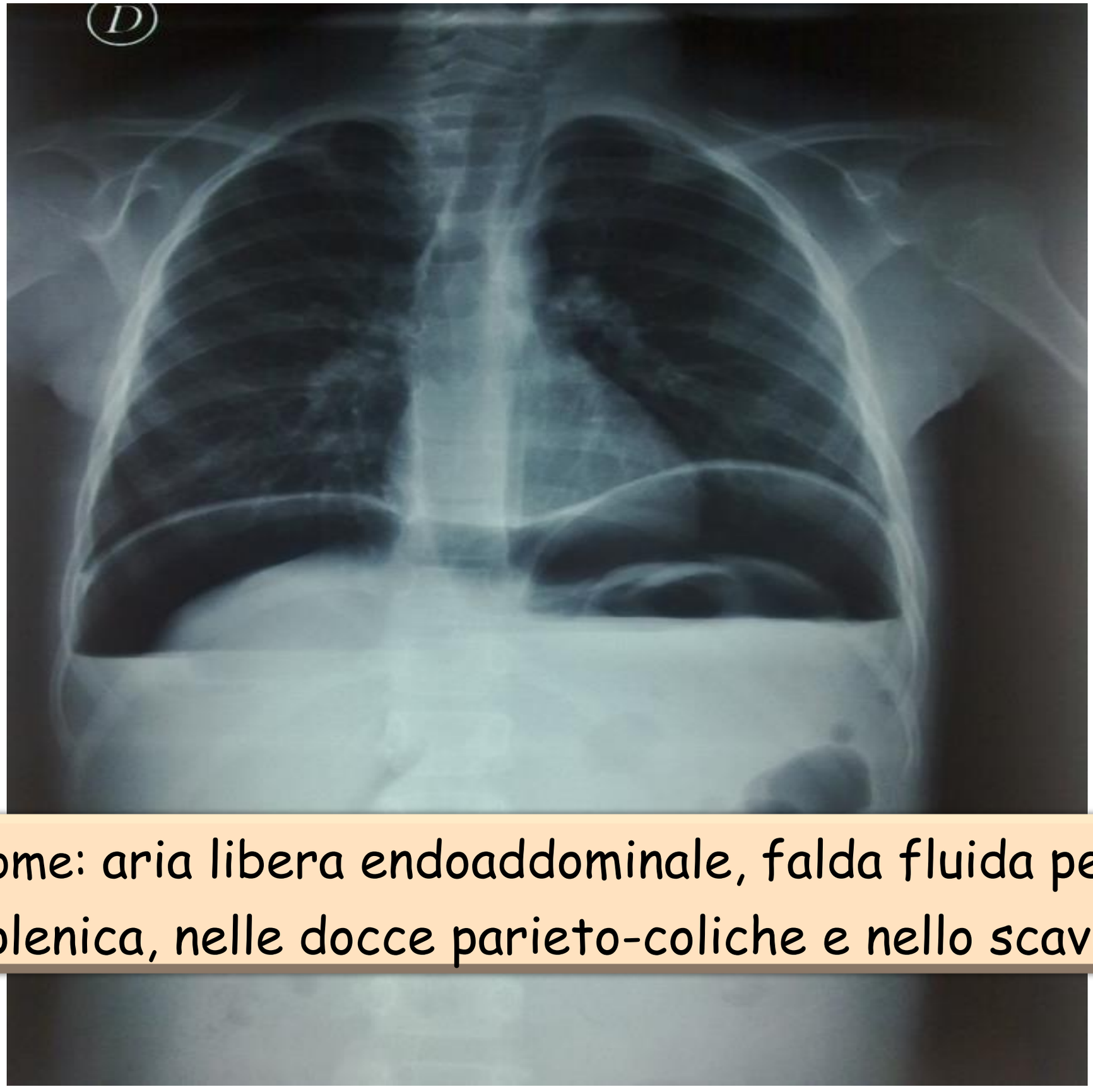
No storia di trauma nè di assunzione di sostanze caustiche

Alvo chiuso a feci e gas

ESAMI DI LABORATORIO

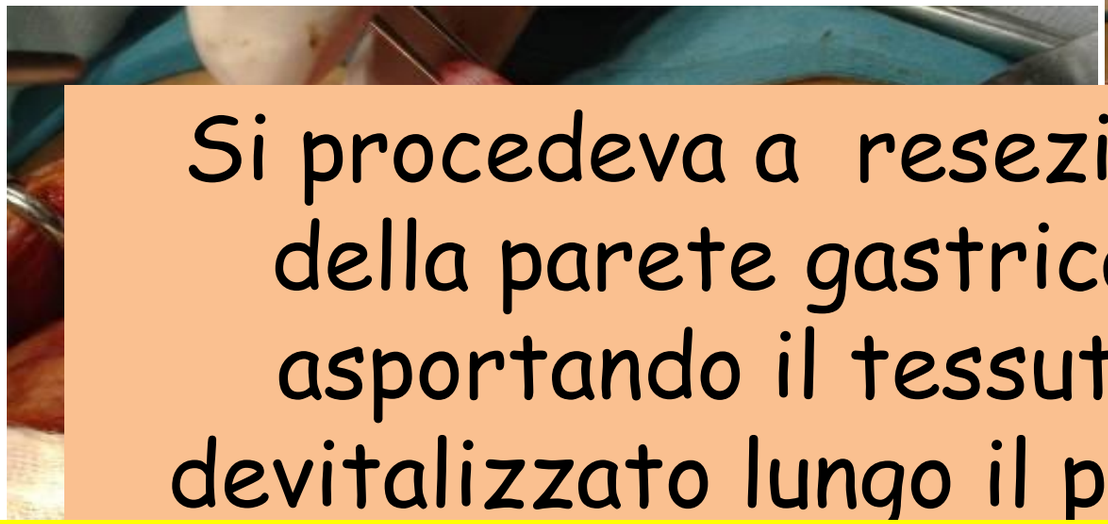
Emocromo: GR: 6.830.000/mmc, Hb: 12.8 g% ml, Hct: 37.6%, GB: 16.580/mmc N 45%, L 30%)

Sodio 129 mEq / L,
Calcio 7.1 mg/dl,
Potassio 3.6 mEq / L,
proteine.: 4.2 mg / dl,
albumina 2.3 mg / dl,
PT 59.8 sec



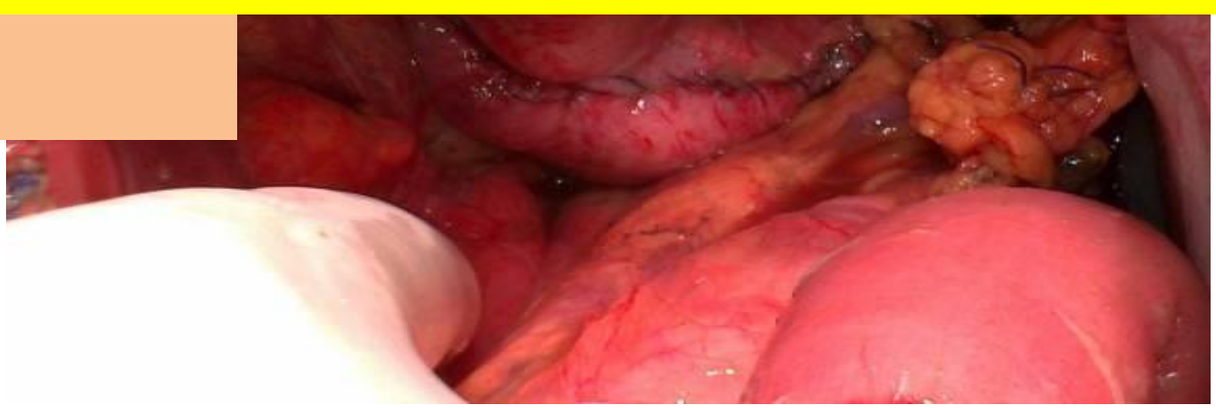
TC addome: aria libera endoaddominale, falda fluida periepatica, perisplenica, nelle docce parieto-coliche e nello scavo pelvico

Posizionato SNG ⇨ Laparotomia urgente



Si procedeva a resezione della parete gastrica, asportando il tessuto devitalizzato lungo il piano gastrica.

Perforazione Gastrica spontanea



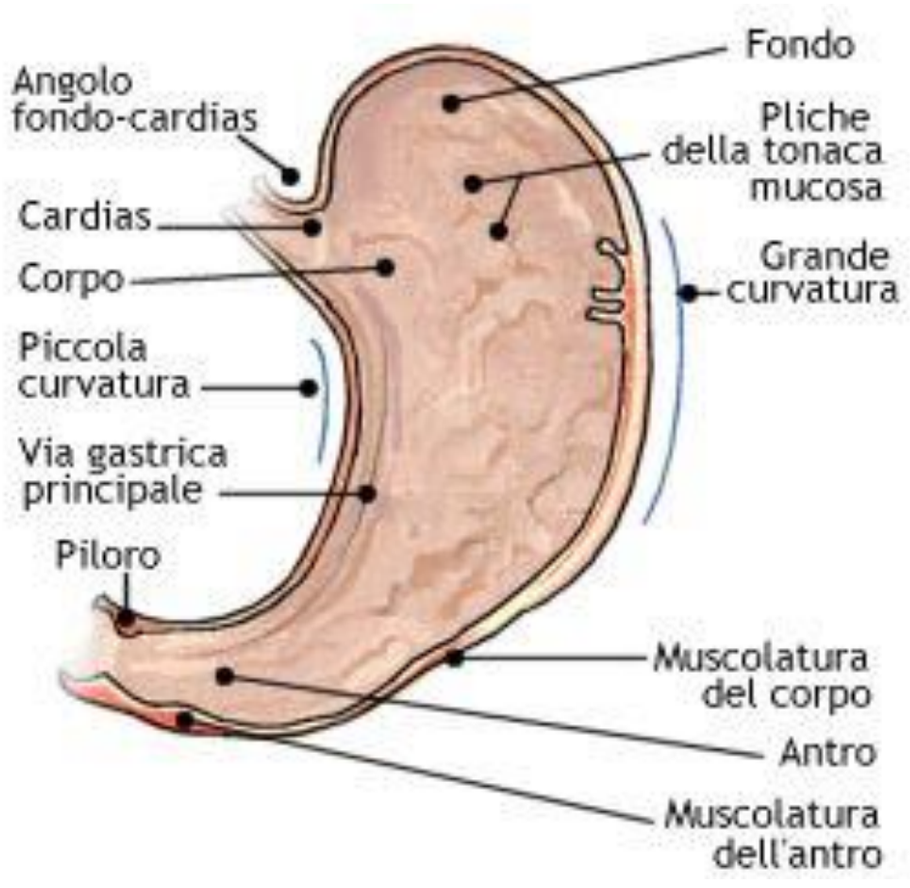
Surg Today (2012) 42:997–1000
DOI 10.1007/s00595-012-0162-4

CASE REPORT

Gastric rupture with necrosis following acute gastric dilatation: report of a case

Takehiro Mishima · Norihiro Kohara · Yoshitsugu Tajima · Junpei Maeda · Keiji Inoue · Tsuyoshi Ohno · Amane Kitasato · Takehito Watanabe · Junji Irie · Tomohiko Adachi · Tamotsu Kuroki · Susumu Eguchi · Takashi Kanematsu

Spontaneous gastric rupture is a rare condition [7, 9, 10], first reported in 1842 [10].



molto mobile
distensibile
situato in profondità

Eziopatogenesi

Indian J Surg (July–August 2014) 76(4):319–320
DOI 10.1007/s12262-013-0980-7

CASE REPORT

Neonatal Gastric Perforations: Are They Really Spontaneous?

Gaurav Gupta • Sachin Kumar • Sangeeta Gupta •
K. B. Golhar • Swapnil Deshpande

Dickens et al. reported the incidence of neonatal gastric perforation as 34 per 1,000 live

NEONATI

- Prematurità
- Asfissia
- Basso peso alla nascita
- Intolleranza alimentare
- Atresia esofagea
- Pancreas anulare
- Ostruzione intestinale
- Anomalie congenite

Gastric Perforation Caused by a Bulimic Attack in an Anorexia Nervosa Patient: Report of a Case

at: http://www.researchgate.net/publication/5773479

pture in non-neonatal

Atsunori Nakao, Hiroshi Isozaki, Hiromi Iwagaki, Taiichiro Kanagawa, Norihisa Takakura, and Noriaki Tanaka

First Department of Surgery, Okayama Medical School, 2-7-10 Shikata-cho, Okayama, Okayama 700-8558, Japan

ARTICLE *in* ACTA CHIRURGICA BELGICA · OCTOBER 2007

Table 1. Reported cases of gastric perforation or necrosis due to bulimia in cases with anorexia nervosa

Patient no.	First author[Ref.]	Year	Age	Sex	Site of necrosis	Outcome
1	Evans[2]	1968	20	Female	Near-total	Recovery
2	Kerstein[3]	1974	18	Female	Near-total	Recovery
3	Saul[1]	1981	22	Female	Anterior wall	Fetal
4	Abdu[4]	1987	14	Female	Great curvature	Recovery
5	Abdu[4]	1987	17	Female	Near-total	Recovery
6	Deret[5]	1987	48	Male	Fundus	Recovery
7	Reeve[6]	1988	32	Male	Near-total	Recovery
8	Hirooka	1991	22	Female	Anterior wall	Recovery
9	Beiles[7]	1992	24	Female	Anterior wall	Recovery
10	Willeke[8]	1996	19	Female	Near-total	Recovery
11	Toyoshima[9]	1997	22	Female	Great curvature	Recovery
12	Nakao	Present case	17	Female	Anterior wall	Recovery

50% dei casi,
dilatazione acuta gastrica

Surg Today (2012) 42:997–1000
DOI 10.1007/s00595-012-0162-4

CASE REPORT

Gastric rupture with necrosis following acute gastric dilatation: report of a case

Takehiro Mishima · Norihiro Kohara · Yoshitsugu Tajima · Junpei Maeda · Keiji Inoue · Tsuyoshi Ohno · Amane Kitasato · Takehito Watanabe · Junji Irie · Tomohiko Adachi · Tamotsu Kuroki · Susumu Eguchi · Takashi Kanematsu

L'eccesso di cibo induce flatulenza che riduce lo svuotamento gastrico.

La fermentazion del cibo, pr ccesso di gas e quindi a distens

La parete gastrica diventa grande, ttile con allungamento dei vasi sanguigni che si os uiscono, con conseguente ischemia

Accorciamento angolo di His e sc acciamento giunzione cardio-esofage n valvola nidirezionale ed ulte polamento di aria

umento della pressione intra-addominale dopo tosse o vomito precipitano rottura

Spontaneous Rupture of the Stomach in Preschool Age Children: A Report of Two Cases

Yasuo Adachi,[1] Hideo Takamatsu,[1] Hiroyuki Noguchi,[1] Hiroyuki Tahara,[1] Motoi Mukai,[1] and Hiroshi Akiyama[2]

QUANDO SOSPETTARLA?

1. Tympanitic abdominal distention.
2. Rigidity of the abdominal parietes.
3. Subcutaneous emphysema.
4. Evidence of shock.[11]

See discussions, stats, and author profiles for this publication at: http://www.researchgate.net/publication/5773479

Spontaneous gastric rupture in non-neonatal children: A case report

ARTICLE *in* ACTA CHIRURGICA BELGICA · OCTOBER 2007

Table 1

Cas[illegible] spontaneous id[illegible] gastric rupture in pre-school children

Authors	Age	Sex	Site of rupture	Complications	Operation	Outcome
Sichita (6) (1981)	2 years	F	Lesser curvature, posterior	ND	Simple closure	Survived
Sanada (6) (1983)	2 years	F	ND	ND	ND	ND
Yokoyama (6) (1984)	1 year	M	Corpus	ND	Partial gastrectomy	Died
Yokoyama (6) (1984)	2 years	F	Greater curvature, posterior	ND	Partial gastrectomy	Survived
Sakai (6) (1987)	4 years	F	ND	ND	ND	ND
Asakura (6) (1987)	4 years	F	ND	ND	ND	ND
Mitani (6) (1990)	4 years	F	Greater curvature, posterior	ND	Partial gastrectomy	Survived
Sato (6) (1991)	3 years	F	Greater curvature, posterior	ND	Partial gastrectomy	Survived
Fukada (6) (1993)	1 year	F	Greater curvature, posterior	ND	Simple closure	Survived
Adachi (6) (1998)	2 years	F	Greater curvature, posterior	No	Simple closure	Survived
Adachi (6) (1998)	4 years	F	Greater curvature, posterior	No	Simple closure	Survived
Shimizu (1) (2003)	3 months	F	Greater curvature 5 cm	Metabolic acidosis	Simple closure	Survived
Soong (2) (1996)	5 years	F	Greater curvature 7.5 cm	No	Simple closure	Survived
Qin (7) (2000)	4 years	F	Greater curvature 7.5 cm	convulsions	Simple closure in two layers	Brain impairment
Qin (7) (2000)	12 years	F	No surgery	Died 2 hours after admission		Died
Qin (7) (2000)	7 months	F	Greater curvature 7.5 cm	Metabolic acidosis	Simple closure in two layers	Survived
Liriher (2007)	3 years	F	Greater curvature 7.5 cm	Metabolic acidosis, pulmonary infection	Sleeve gastrectomy	Survived

F, female ; M, male ; ND, not described.

Spontaneous Rupture of the Stomach in Preschool Age Children: A Report of Two Cases

Yasuo Adachi,[1] Hideo Takamatsu,[1] Hiroyuki Noguchi,[1] Hiroyuki Tahara,[1] Motoi Mukai,[1] and Hiroshi Akiyama[2]

Mortalità 30%

Table 1. Cases of spontaneous gastric rupture in preschool children in Japan

Authors	Age (years)	Sex	Site of rupture	Operation	Outcom
Sigeta (1981)	2	F	corpus, posterior, lesser	simple closure	S
Sanada (1983)*	2	F	ND	ND	ND
Yokoyama (1984)*	1	M	corpus	partial gastrectomy	D
Yokoyama (1984)*	2	F	corpus, posterior, major	partial gastrectomy	S
Sakai (1987)*	4	F	ND	ND	ND
Asakura (1987)*	4	F	ND	ND	ND
Mitani (1990)	4	F	corpus, posterior, major	partial gastrectomy	S
Sato (1991)	3	F	corpus, posterior, major	partial gastrectomy	S
Fukada (1993)	1	F	corpus, posterior, major	simple closure	S
Adachi (1996)	2	F	fundus, posterior, major	simple closure	S
Adachi (1996)	4	F	corpus, posterior, major	simple closure	S

F, female; M, male; D, dead; S, survived; ND, not described
* Cases referred to in ref. 14

Take home messages

In presenza di importante distensione addominale dopo un pasto inusualmente abbondante, è importante sospettare una rottura gastrica

Una rapida valutazione diagnostica e pronto approccio chirurgico rende più favorevole outcome

7th JOINT MEETING
In pediatria e medicina dell'adolescenza
Sobre pediatría y medicina de la adolescencia

21-22-23-24 OTTOBRE 2015 CATANZARO ITALIA

Non innamorarsi della prima diagnosi,

Grazie per l'attenzione