

Il bambino con il mal di testa

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Il Bambino con il mal di testa



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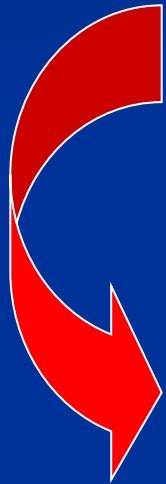
CEFALEA INFANTILE

La cefalea è un disturbo comune nei bambini e negli adolescenti

2-5 aa 5.9 al 37.7%

6-12 aa 40-50%

> 12 aa 80%¹



La cefalea è un disturbo frequente in età pediatrica ed è causa di grave preoccupazione, ansietà ed allarme nelle famiglie e nei pediatri rappresentando uno dei più motivi più comuni per un accesso presso il PSP

1- Sillanpaa M, Abu-Arafeh I. Epidemiology of recurrent headache in children.

In: Abu-Arafeh I, ed.

Childhood Headache. London: MacKeith Press;

2002:19-34

Fenomeno epidemiologico

- **La cefalea è un disturbo frequente in età infantile. Tra il 20-30 % degli adolescenti presentano cefalea 1 volta x settimana e il 6% diverse volte la settimana**

El-Chammas et al. Pharmacologic Treatment of pediatric headache
– A meta-analysis JAMA Pediatr 2013;1-11

COMPITO DEL PEDIATRA

1. Diagnosticare il tipo di cefalea
2. Individuare precocemente le forme gravi
3. Stabilire gli esami più opportuni da eseguire
4. Valutare l'eventualità del trattamento

STRUTTURE ALGOSENSIBILI

Extracraniche:

- Muscoli e vasi sanguigni del cuoio capelluto
- Tessuto epiteliale dei seni
- Strutture orecchie e orbite

Intracraniche:

- A. cerebrali e della dura
- Grosse vene e seni venosi

Meccanismi che stimolano il dolore dai vasi sanguigni

1. Vasodilatazione
2. Infiammazione
3. Trazione-dislocazione

CEFALEA

Classificazione –Eziologica

Forme primarie:

emicrania

Forme secondarie:

patologie locali o sistemiche

CEFALEA SECONDARIA

Cause Neurologiche

Trauma cranico
Emorragie endocraniche
Trombosi
Meningo-encefaliti
Tumori endocranici
Idrocefalo
Pseudotumor cerebri

Cause non neurologiche

Malattie virali
Sostanze tossiche
Sinusiti
Otitis
Mononucleosi (s. Alice nel paese delle meraviglie)

CEFALEA PRIMARIA

- Eemicrania
- Cefalea tensiva o psicogena
- Cefalea cronica giornaliera
- Cefalea a grappolo

CEFALEA

In rapporto al decorso si distinguono

- F. Acuta Infezione SNC
- F. acuta - ricorrente Eemicrania
- F. Cronico - progressiva Ipertens. endocr.
- F. Cronico - non progressiva Psicogena
- F. mista

EMICRANIA E CEFALEA

Non sono sinonimi: l'emicrania è una condizione particolare di cefalea

EMICRANIA

L'emicrania si manifesta con attacchi parossistici, episodici e periodici di cefalea che variano per intensità, frequenza e durata talvolta preceduti da disturbi visivi e accompagnati da nausea, vomito, fono-fotofobia.

EMICRANIA

Caratteristiche del dolore:

- Intensità: lieve → marcata
- Frequenza: settimanale → mensile
- Durata: minuti → ore

EMICRANIA

Fattori
scatenanti:

- Sforzo fisico
- Variazioni della temperatura
- Tensione psichica
- Cicli mestruali
- Uso di alcolici
- Cibi particolari
- Privazione del sonno
- Eccessiva applicazione
- Eventi drammatici

CEFALEA TENSIVA

- Cefalea tensiva (IHS): 10 episodi nell'arco della vita
 - crisi di maggior durata (30 minuti – 7 giorni) e minore intensità
 - dolore di tipo gravativo e a cerchio
 - non aggravato dall'attività fisica e dallo stress
 - non presenti nausea e vomito, e solo 1 tra fono/fotofobia
 - stato depressivo-ansioso
- Cefalea a grappolo: crisi subcontinue intervallate da brevi periodi di benessere

Fattori ambientali ed individuali predisponenti alla Cefalea Tensiva

Anttila P Lancet Neurol 2006

Stress psico-sociale
Disordini psichiatrici (s.depressivi nella CTEF)
Scarse relazioni con i coetanei
Disarmonia nel nucleo familiare
Disfunzione oro-mandibolare
Malattie somatiche
Malattie croniche (> CTC)

Stress associato alle trasformazioni
somatiche dell'adolescenza



Cefalea di Tipo
Tensivo nel giovane
adulto

Walkie KE Headache 2001

CEFALEA PRIMARIA

Attacchi parossistici, episodici e periodici

Emicrania

Cefalea tensiva

D.D.

Durata	1-72 h	30 m-7gg
Tipo	pulsante	gravativo-a cerchio
Aggravanti	stress – es. fisico	no
Stati psichici	no	ossessivo-ansioso
S. associati	fono/fotofobia	sì
	nausea-vomito	no

Emicrania e cefalea tensiva possono coesistere nello stesso soggetto

E

CT

Età di comparsa < 7 anni

+

Dolore aggravato dalle attività

+

Durata del dolore

+

Efficacia della terapia del dolore

+

Stress psico-sociale

+

Equivalenti emicranici

+

Familiarità

+

Evoluzione favorevole

+

**“ESISTE UN RAPPORTO
EMICRANIA / EPILESSIA ?”**

EMICRANIA-EPILESSIA

CO-MORBIDITÀ: lo stesso individuo può presentare, in periodi diversi, sia crisi emicraniche che crisi epilettiche.

EPILESSIA - CEFALEA

La cefalea può presentarsi nel corso di una crisi epilettica come:

- Aura
- Episodio critico (dolore improvviso, breve, lancinante)
- Manifest. post-convulsiva:
 - E. general.di lunga durata
 - E. parziali (EBPR-EBPO)

- Belcastro V, Striano P, Parisi P. From migralepsy to ictal epileptic headache: the story so far. Neurol Sci. 2013 Oct;34(10):1805-7. doi: 10.1007/s10072-012-1012-2. Epub 2012 Mar 17.
- Parisi P, Verrotti A, Costa P, Striano P, Zanusi C, Carrozzi M, Raucci U, Villa MP, Belcastro V. Diagnostic criteria currently proposed for "ictal epileptic headache": Perspectives on strengths, weaknesses and pitfalls. Seizure. 2015 Sep;31:56-63. doi: 10.1016/j.seizure.2015.07.005.

L'EEG è fondamentale nel distinguere episodi epilettici da quelli non epilettici, ma con significativi limiti

Un EEG interictale normale NON esclude la diagnosi di epilessia



ITER DIAGNOSTICO

- Anamnesi familiare e personale
- Decorso temporale della cefalea
- Esame obiettivo generale
- Esame neurologico

Negativo

- Visita oculistica
- Es. ematochimici

Positivo

- Es. ematochimici
- Visite specialistiche
(ORL, fundus)
- TC; RMN; AngioRM
- Ecocolordoppler
- Esame liquor

Road map

Cefalea insidiosa CI



Definizione CI

Codici bianchi in PS su casistica

Caratteristiche Cefalea secondaria a patologie
neurologiche



Red flags in patients presenting with headache: clinical indications for neuroimaging

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Table 1. List of red flags and their frequencies

Red flag	Frequency	Percentage
Onset of new or different headache	64	57.7
Nausea or vomiting	33	29.7
Worst headache ever experience	32	28.8
Progressive visual or neurological changes	20	18.0
Paralysis	15	13.5
Weakness, ataxia or loss of co-ordination	14	12.6
Drowsiness, confusion, memory impairment or loss of consciousness	13	11.7
Onset of headache after age of 50 years	12	10.8
Papilloedema	10	9.0
Stiff neck	6	5.4
Onset of headache with exertion, sexual activity or coughing	6	5.4
Systemic illness	5	4.5
Numbness	4	3.6
Asymmetry of pupillary response	2	1.8
Sensory loss	1	0.9
Signs of meningeal irritation	1	0.9

Primary versus secondary headache in children: a frequent diagnostic challenge in clinical routine.

Roser T¹, Bonfert M, Ebinger F, Blankenburg M, Ertl-Wagner B, Heinen F.

⊕ Author information

Abstract

A sensitive and specific triage of patients with primary or secondary headache is a major concern in evaluating pediatric headache patients. History and physical examination are the major tools for differentiating primary headache disorders from symptomatic headaches caused by defined pathologies. If the criteria of the International Headache Society for a primary headache disorder are met, no further investigations are necessary. However, physicians should be familiar with subtle signs in history and physical examination that raise suspicion of intracranial pathology. These features, also named "red flags" and "relatively red flags," are outlined in detail in this review. Any red flag should prompt neuroimaging. In case of relatively red flags, a more restrained approach can be appropriate depending on the individual setting. Excessive concerns of patients and parents regarding an underlying pathology can constitute an indication for neuroimaging. Offering neuroimaging implicates the important issues of incidental findings and of "false reassurance." These risks should be discussed with patients and parents before the investigation. In any pediatric headache patient, regular clinical reevaluations should be warranted, even if neuroimaging is normal. The value of clinical follow-up examinations for a reasonable and reliable assessment of the patients cannot be overestimated.



Anamnesi + Obiettività neurologica



Red Flags

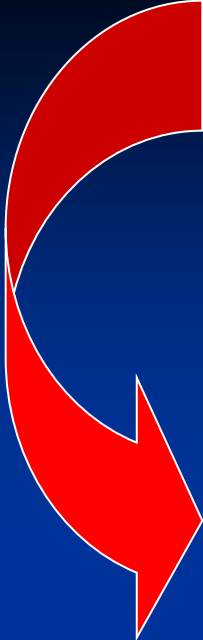
- Alterazione della coscienza e/o del comportamento
- Aumento della circonferenza cranica
- Soffi endocranici
- Paralisi dei nervi cranici
- Movimenti oculari abnormi e/o riflessi pupillari alterati
- Difetti del campo visivo, papilledema
- Deficit neurologici focali, deficit della deambulazione e/o di crescita
- Pubertà precoce o ritardata

Roser T1, Bonfert M, Ebinger F, Blankenburg M, Ertl-Wagner B, Heinen F.

Primary versus secondary headache in children: a frequent diagnostic challenge in clinical routine.

Neuropediatrics. 2013 Feb;44(1):34-9. doi: 10.1055/s-0032-1332743. Epub 2013 Jan 3.

Cefalea insidiosa

- 
- Cefalea da codice bianco (pregressa asintomatica) e senza segni di allarme!!!

Tutti riconoscibili???



CEFALEA

ESCI

ISTRUZIONI

COMPROMISSIONE DI UNA FUNZIONE VITALE

GRAVE DEFICIT NEUROLOGICO O SENSORIALE

RIALZO PRESSORIO (P.D. >100 ETA' <10 ANNI O >110 ETA' > 10 ANNI)

RIALZO PRESSORIO (P.D. $>80 \leq 100$ IN ETA' ≤ 10 ANNI OPPURE $>90 \leq 110$ IN ETA' > 10 ANNI)

CONVULSIONE RIFERITA < 12 ORE

CEFALEA INTENSA CON FEBBRE E/O VOMITO (≥ 3 EPISODI)

ANAMNESI POSITIVA PER RECENTE TRAUMA CRANICO < 12 ORE

ANAMNESI POSITIVA PER MALATTIA NEUROLOGICA O SHUNT V-P

DOLORE SEVERO (SCALA DEL DOLORE ≥ 8)

DOLORE LIEVE O MODERATO (SCALA DEL DOLORE ≤ 7)

VOMITO OCCASIONALE ≤ 2 EPISODI

ANAMNESI POSITIVA PER TRAUMA CRANICO MINORE > 12 ORE

CONVULSIONE RIFERITA > 12 ORE

CEFALEA PREGRESSA ASINTOMATICA



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journal homepage: www.elsevier.com/locate/ajem

The
American Journal of
Emergency Medicine

Original Contribution

Triage of children with headache at the ED: a guideline implementation study ☆☆☆★

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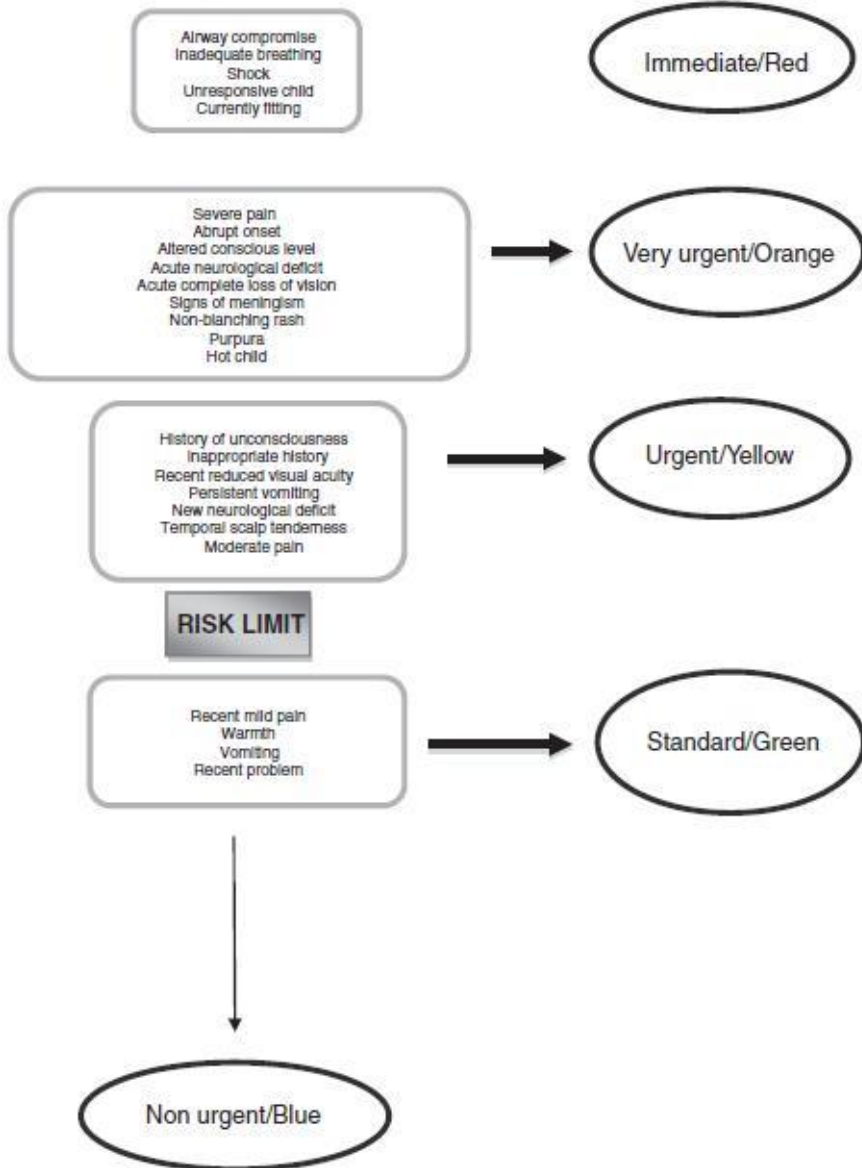
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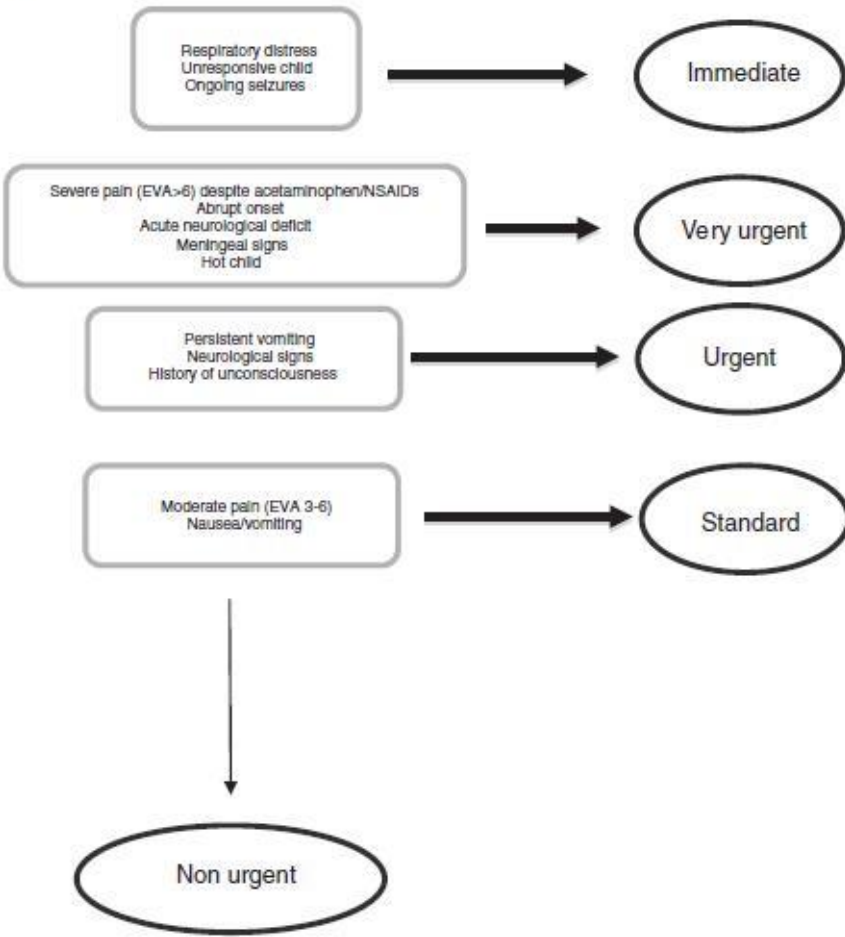
A

Headache



B

Headache



Materiali e metodi

Numero totale 132 pazienti con cefalea tra Gennaio e Dicembre 2013
UOC pediatria e Pronto Soccorso Pediatrico: PO Vittorio Emanuele, Catania

CODICI BIANCHI

I pazienti con cefalea associata ad un trauma cranico non sono stati inclusi

Cefalea non-life-threatening diseases

■ Cefalea primaria	69.4%
■ Infezioni ricorrenti tratto respiratorio	14%
■ Medicazioni croniche	1.8%
■ Disidratazione	1.3%
■ Ipertensione arteriosa	1.4%
■ Anemia	0.3%
■ Errori refrattivi	2.4%
■ Al. Denti	0.7%

Cefalea life-threatening

ICHD-II codes	%
■ Alterazioni cerebrali (Chiari,Dandy-Walker)	0.7
■ Tumori cerebrali	3
■ Pseudotumor cerebri	0.7

Da memorizzare che:

- Il 96% dei bambini hanno mostrato una cefalea benigna
- Il 4% hanno avuto una cefalea lifethreatening

- Le cefalee primarie sono state osservate nel
 - bambini in età scolare
 - + adolescenti

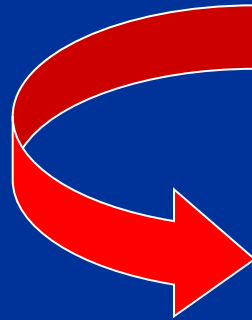
Falso mito !!!

Vomito non è sinonimo di Ipertensione Endocranica

- I sintomi associati non erano statisticamente associati con l'insorgenza di una cefalea benigna o lifethreatening
Il vomito è stato il più comune ed è stato segnalato nella diverse forme di cefalee con la stessa aliquota (30%).

- Nel 67% dei casi la cefalea neurologica secondaria si associava a:

Febbre, deficit neurologici focali e disturbi comportamentali



Tutte i bambini affetti da una grave patologia di base neurologica aveva segni obiettivi neurologici, tra cui papilledema, movimenti oculari anomali.

Fattori Associati a cefalea benigna o lifethreatening

	<u>Benigne</u>	<u>Life-threatening</u>
Età	Scolare	Pre-scolare
<u>Esordio cefalea</u>	Graduale	Ingravescente/Rapid
<u>Localizzazione</u>	Uni/bil, frontal/temporal	Capace di loca Occipitale
Qualità dolore	descrizione/puls	Non abile descriz. Costrittivo
<u>Intensità dolore</u>	Lieve/Intenso	Molto Intenso
<u>Segno neurologici</u>	Nessuno	Deficit neurologici papilledema, atassia, Alt. coscienza

Headaches

Diagnosis and management of headaches in young people and adults

Clinical Guideline 150

Methods, evidence and recommendations

September 2012

QUANDO RICHIEDERE
RMN - ENCEFALO?

SINTOMI D'ALLARME

- Episodi più frequenti e di maggiore intensità
- Nausea e vomito soprattutto se mattutini
- Alterazioni del comportamento
- Accentuazione del dolore con i cambiamenti di postura
- Risvegli notturni per la cefalea
- Associazione con crisi epilettiche parziali
- Segni neurologici presenti

Delay in the diagnosis of paediatric brain tumours

in this age group approximates only 3/100,000 (0.003%) [12]. Uncritical imaging would result in a large number of normal neuroimaging studies. In our study, most children with headache as initial symptom showed additional signs/symptoms within a relatively short period. This is consistent with a large study of the Childhood Brain Tumor Consortium [12]. Analysing 3276 patients, less than 3% of children with headache and a brain tumour had no abnormality on neurological examination.

Although not yet proven, a shortened PSI may lead to the detection of smaller brain tumours that are easier to resect. The value of extensive tumour resection, which

Table 1 Frequency of initial signs and symptoms depending on age

Signs and symptoms	All (<i>n</i> = 252)	Age < 2 years (<i>n</i> = 50)	Age ≥ 2 years (<i>n</i> = 202)
Headache	35%	2%	43%
Nausea/vomiting	26%	18%	28%
Seizures	14%	20%	12%
Behavioural changes (irritability, mood, character, school)	10%	12%	9%
Ataxia	8%	8%	8%
Squint/diplopia	8%	6%	8%
Lethargy	5%	4%	5%
Hemiparesis/quadriparesis	5%	8%	4%
Head tilt	5%	12%	3%
Anorexia	3%	6%	2%
Growth failure	3%	—	3%
Sleep disturbance	2%	2%	2%
Polyuria/polydipsia	2%	—	3%
Visual loss	2%	2%	2%
Weight loss	2%	4%	1%
Facial nerve palsy	2%	4%	1%
Enlargement of the head	2%	8%	—
Cranial neuropathies other than III, IV, VI, VII	1%	—	1%
Gaze depression/separation of cranial sutures/bulging fontanelle	1%	4%	—
Dizziness	1%	—	1%
Nystagmus	1%	4%	—
Papilloedema	1%	—	1%
Amenorrhoea	0.5%	—	0.5%
Proptosis	0.5%	—	0.5%

- [Pediatr Int.](#) 2011 Dec;53(6):964-7. doi: 10.1111/j.1442-200X.2011.03493.x.
- **Primary headache: role of investigations in a cohort of young children and adolescents.**
- [Pavone P1](#), [Conti I](#), [Le Pira A](#), [Pavone L](#), [Verrotti A](#), [Ruggieri M](#).
- **Author information**
- **Abstract**
- **BACKGROUND:**
- We report a study conducted in children and adolescents who are affected by primary headaches. The aim was to establish the most useful investigations for diagnosing headaches.
- **METHODS:**
- The current study involved 300 consecutively hospitalized children and adolescents selected according to the criteria of the second edition of the International Classification of Headache Disorders. The following examinations were performed in all patients: full ophthalmologic; brain magnetic resonance imaging (MRI); electroencephalography; echocardiogram; and electrocardiogram. Dental, otorhinolaryngology, echocardiography of the supra-aortic trunks, abdominal ultrasound, and visual- and auditory-evoked potentials were carried out in patients according to the clinical signs associated with headache.
- **RESULTS:**
- In a large number of cases routine laboratory analysis and neurophysiologic investigations were within the normal value when neurologic examination was normal. Electroencephalography, ophthalmologic studies and cerebral MRI are advisable as they can reveal precocious pathologic events, even in the absence of evident or alarming clinical signs.
- **CONCLUSION:**
- As widely reported in the literature, most of these investigations may be of little clinical value, but the authors reasoned that electroencephalography, ophthalmologic investigations and a cerebral MRI may be noteworthy because such studies may reveal a precocious pathologic event which can change the prognostic value of the headache. In addition, negative results on cerebral MRI may relieve the anxiety of parents and in turn may positively influence the clinical course of headache in children and adolescents.

Primary headache: clinical data and results on association with other conditions. A pediatric point of view.

*Pavone Piero, MD; Conti Irene, MD; *Rizzo Renata, MD, PhD;
•Pulvirenti Alfredo, MD; Pavone Lorenzo, PhD, MD*

Co-morbidity with other conditions such as asthma and allergic disorders, sleep disorders, obesity resulted not significant. Our results on relationship between primary headache and epilepsy and celiac disease had been reported in previous studies. Concerning the behavioral disturbances we no found relationship between headache and ADHD, Tics, learning disability, OCD, but a significant results were obtained with anxiety and depression.

INTERPRETATION Primary headache in children is not associated with specific disorders such as asthma and allergic disorders, sleep disorder, obesity, as reported frequently in the literature particularly in adults.

On the contrary a significant association appear evident with anxiety and depression as reported in adult age.

Primary headaches in children: clinical findings on the association with other conditions. Int J Immunopathol Pharmacol. 2012 Oct-Dec;25(4):1083-91

- J Headache Pain. 2014 Sep 1;15:57. doi: 10.1186/1129-2377-15-57.
- **Clinical guidelines in pediatric headache: evaluation of quality using the AGREE II instrument.**
- Parisi P1, Vanacore N, Belcastro V, Carotenuto M, Del Giudice E, Mariani R, Papetti L, Pavone P, Savasta S, Striano P, Toldo I, Tozzi E, Verrotti A, Raucci U; “Pediatric Headache Commission” of Società Italiana di Neurologia Pediatrica (SINP).

- **BACKGROUND:**

- The Appraisal of Guidelines for Research and Evaluation (AGREE II) tool is a validated questionnaire used to assess the methodological quality of clinical guidelines (CGs). We used the AGREE II tool to assess the development process, the methodological quality, and the quality of reporting of available pediatric CGs for the management of headache in children. We also studied the variability in responses related to the characteristics of eleven Italian neuropediatric centers, showing similarities and differences in the main recommendations reported in CGs.

- **CONCLUSIONS:**

- CGs resulted definitely of low-moderate quality and non "homogeneous". Further major efforts are needed to update the existing CGs according to the principles of evidence based medicine.

- [Headache](#), 2015 Oct 31. doi: 10.1111/head.12709. [Epub ahead of print]
- **Depression as a mediator of the relation between family functioning and functional disability in youth with chronic headaches.**
- [Kaczynski K](#)1,2, [Gambhir R](#)1,2, [Caruso A](#)1, [Lebel A](#)1,3.
- **Author information**
- **Abstract**
- **OBJECTIVE:**
- This retrospective chart review examined a mediation model of parent and family functioning, childhood depression, and functional disability in youth with chronic headaches. Specifically, we evaluated whether depression mediates the relations between protective parenting and functional disability and between family functioning and functional disability.
- **BACKGROUND:**
- Children and adolescents with chronic and recurrent headache report elevated symptoms of depression. Children with chronic pain conditions, including chronic headaches, have also been found to originate from families with greater conflict, poorer cohesion, and lower organizational structure, and impaired family functioning is associated with greater disability in youth with chronic pain.
- **METHODS:**
- Three hundred and eighty-two patients ages 5-17 years who underwent a multidisciplinary evaluation at a tertiary pediatric headache clinic were included in this study. Participants completed a pain intensity rating, the Children's Depression Inventory, and the Functional Disability Inventory. A parent completed the Family Relationship Index and the Adult Responses to Children's Symptoms questionnaires. Structural equation modeling was used to examine a mediation model and several alternative models.
- **RESULTS:**
- Mediation was not supported, but an alternative model with both direct and indirect pathways provided excellent fit to the data: $\chi^2(1) = 0.745$, $P = .39$; comparative fit index = 1.00, root mean square error of approximation = 0.00 (CI: 0.00-0.17). Family functioning ($\beta = -0.19$, $P < .01$) and protective parenting ($\beta = 0.17$, $P < .01$) were associated with depression, but not disability. Depression was linked to disability ($\beta = 0.24$, $P < .01$). There was an indirect pathway from family functioning to depression to disability ($\beta = -0.05$, $P < .05$).
- **CONCLUSIONS:**
- Family context is an important variable to consider in youth with chronic headaches and disability. While many studies have identified family functioning and depressive symptoms as separately linked to functional impairment, to our knowledge, we are the first to demonstrate depression as an intermediary variable between family dysfunction and disability within the pediatric headache population.

- [Headache](#). 2015 Oct 31. doi: 10.1111/head.12701.
- **Pediatric inpatient headache therapy: What is available.**
- [Kabbouche M1](#).
- **Abstract**
- Status migrainosus is defined by the international classification of headache disorders (ICHD) criteria as a debilitating migraine lasting more than 72 hours. The epidemiology of status migrainosus is still unknown in adult and children, and frequently underdiagnosed. Children and adolescents often end up in the emergency room with an intractable headache that failed outpatient therapy. Six to seven percent of these children do not respond to acute infusion therapy and require hospitalization. It is imperative that more aggressive therapy is considered when patients are affected by a severe intractable headache to prevent further disability and returning the child to baseline activity. Multiple therapies are available for adults and children. Studies for acute therapy in the emergency room are available in adults and pediatric groups. Small studies are available for inpatient therapy in children and, along with available therapies for children and adolescents, are described in this review. A review of the literature shows growing evidence regarding the use of dihydroergotamine intravenously once patients are hospitalized. Effectiveness and safety have been proven in the last decades in adults and small studies in the pediatric populations.

Obiettività neurologica

- Nervi cranici (strabismo convergente ecc)
- Andatura regolare ?, a larga base di impianto ?
- ROT vivaci o aumento area riflessogena ?
- Disturbi del comportamento ?
- Difficoltà eloquio ?
- Romberg ?

- J Pediatr Gastroenterol Nutr. 2009 Aug;49(2):202-7. doi: 10.1097/MPG.0b013e31818f6389.
- **Headache in pediatric patients with celiac disease and its prevalence as a diagnostic clue.**
- Lionetti E1, Francavilla R, Maiuri L, Ruggieri M, Spina M, Pavone P, Francavilla T, Magistà AM, Pavone L.
- **Author information**
- **Abstract**
- **OBJECTIVES:**
- To establish the prevalence of headache in children with celiac disease (CD), the response to a gluten-free diet, and the prevalence of CD in children affected by headache.
- **METHODS:**
- This hospital-based study included 2 steps. In the retrospective part, 354 children with CD answered a questionnaire investigating the presence of headache before and after the gluten-free diet. The same questionnaire was administered to 200 healthy children matched for sex and age. In the prospective part, 79 children affected by headache were screened for CD by antitransglutaminase IgA. Diagnosis of CD was confirmed by duodenal biopsy; before starting a gluten-free diet patients underwent a brain positron emission tomography study. After 6 months of follow-up children were reevaluated for the presence of headache.
- **RESULTS:**
- Overall, 88 patients with CD complained of headaches before the diagnosis of CD as compared with 16 in the control group (24.8% vs 8%, $P < 0.001$). After the institution of a gluten-free diet, the headaches significantly improved in 68 children (77.3%), of whom 24 (27.3%) were headache-free during the study period. Four of 79 (5%) headache patients were found to have CD compared with 0.6% of the general population ($P = 0.005$). The brain positron emission tomography studies did not show any anomalies. During the follow-up, headaches improved in all 4 children with CD.
- **CONCLUSIONS:**
- We recorded -- in our geographical area -- a high frequency of headaches in patients with CD and vice versa with a beneficial effect of a gluten-free diet. Screening for CD could be advised in the diagnostic work-up of patients with headache.

CEFALEA-PROFILASSI

Non farmacologica

- Igiene del sonno
- Dieta
- Sport

Il Fattore Dieta nella cefalea Pediatrica

Millichap JG, Pediatric Neurology 2003

“ Fattori dietetici potrebbero intervenire in fasi del processo emicranico influenzando il rilascio di serotonina e norepinefrina, causando vasocostrizione e vasodilatazione o diversamente stimolando direttamente i gangli del nervo trigemino, i nuclei del tronco, il talamo, la corteccia....”

Formaggio (tiramina)

Agrumi (amine fenoliche)

Cioccolato (feniletilamina)

Insaccati, hot dog (nitriti)

Derivati del latte (caseina)

Cucina asiatica (glutammato monosodico)

Grassi e cibi fritti (Acido linoleico ed oleico)

Caffeina

Coloranti e conservanti (tartrazina, solfiti)

Dolcificanti (aspartame)

Vino, birra (istamina, tiramina, solfiti)

Digiuno (ipoglicemia)

CEFALEA- PROFILASSI

Indicazioni

- 3-4 episodi significativi al mese
- 1 episodio significativo di lunga durata (< 3 gg)

Migraine in Children and Adolescents

A Guide to Drug Treatment

Mirja L. Hämäläinen

Department of Pediatric Neurology, Hospital for Children and Adolescents, Helsinki University Central Hospital, Helsinki, Finland

Table II. Principles of migraine treatment

Migraine cannot be cured, but migraine attacks can be controlled and relieved with adequate treatment, which includes:

- getting enough sleep and eating regular meals
- keeping a headache calendar or diary
- avoiding 'triggers' if possible and if they are known:
 - avoid staring at glaring or flickering lights
 - wear sunglasses in bright sunlight
 - drink plenty of fluids
 - do not head the ball in soccer
 - keep a regular sleep schedule
 - do not drink excessive amounts of cola drinks, coffee or tea
 - maintain a healthy diet
- taking symptomatic treatment for a migraine attack, such as ibuprofen or paracetamol (acetaminophen) during visual aura or sumatriptan nasal spray within 30 minutes of the headache starting. It is important that an adequate dose is taken early enough
- if there is no response to symptomatic treatment, further investigations are indicated and prophylaxis (i.e. preventive treatment) can be considered. Monotherapy is recommended with agents such as β -adrenoceptor antagonists, amitriptyline, topiramate, valproic acid (sodium valproate) or flunarizine

CEFALEA INFANTILE

Profilassi degli attacchi

- Flunarazina (Flunagen): Ca-antagonista 5mg/die
 - Propanololo (Inderal): beta-bloccante 2-4 mg/Kg/die
 - Ciproeptadina (Periactin): antag. serotonina 0.25-1.25mg/Kg/die
 - Amitriptilina (Laroxyl): antidepressivo 10-25 mg/die serale
 - Topiramato
 - Valproato
 - Gabapentin
- | | | |
|--|---------------------|-----------------|
| | } Anticonvulsivanti | 1-10mg/Kg/die* |
| | | 20-40 mg/Kg/die |
| | | 10-40 mg/Kg/die |

*Lewis D et Al – Pediatrics 2009

CEFALEA INFANTILE

Terapia degli attacchi:

- Ibuprofene (FANS): 10 mg/Kg/die
- Paracetamolo: 10-15 mg/Kg/dose
- Sumatriptan nasale (Agonista 5-Ht): 5-20 mg/dose

Evidenze di efficacia delle Profilassi Non Farmacologica delle Cefalee Primarie

Terapie psicologiche del dolore cronico e ricorrente nel bambino e nell'adolescente

Cochrane Library 2003

Sussistono evidenze molto buone a sostegno dell'efficacia di terapie psicologiche, quali la relaxation therapy e la terapia cognitivo – comportamentale nei bambini e negli adolescenti con cefalea cronica

Psychological treatment of recurrent headache in children and adolescents – a metha-analysis

Trautmann E Cephalalgia 2006

Biofeed-back, Relaxation Therapy e Terapie cognitivo-comportamentali hanno portato ad un miglioramento clinico, ma occorrono ancora studi controllati per dimostrarne l'efficacia.

Trattamento comportamentale breve

Brief neurologist-administered behavioral tretment of pediatric episodic tension-type headache, Andrasik F et al. Neurology 2003

Un trattamento con sedute settimanali per otto settimane hanno dimostrato miglioramenti evidenti fino ad un anno di follow-up

CONCLUSIONE

- Attenta, accurata anamnesi (genitori e paziente)
- Esame obiettivo neurologico con fondo oculare
- Differenziare cefalea primaria e secondaria
- Differenziare le cefalea in base ai codici in PS

11° Congresso Nazionale medico-infermieristico

SIMEUP

Società Italiana di Medicina di Emergenza ed Urgenza Pediatrica

l'urgenza... di sapere
sul... in pronto soccorso... in re...



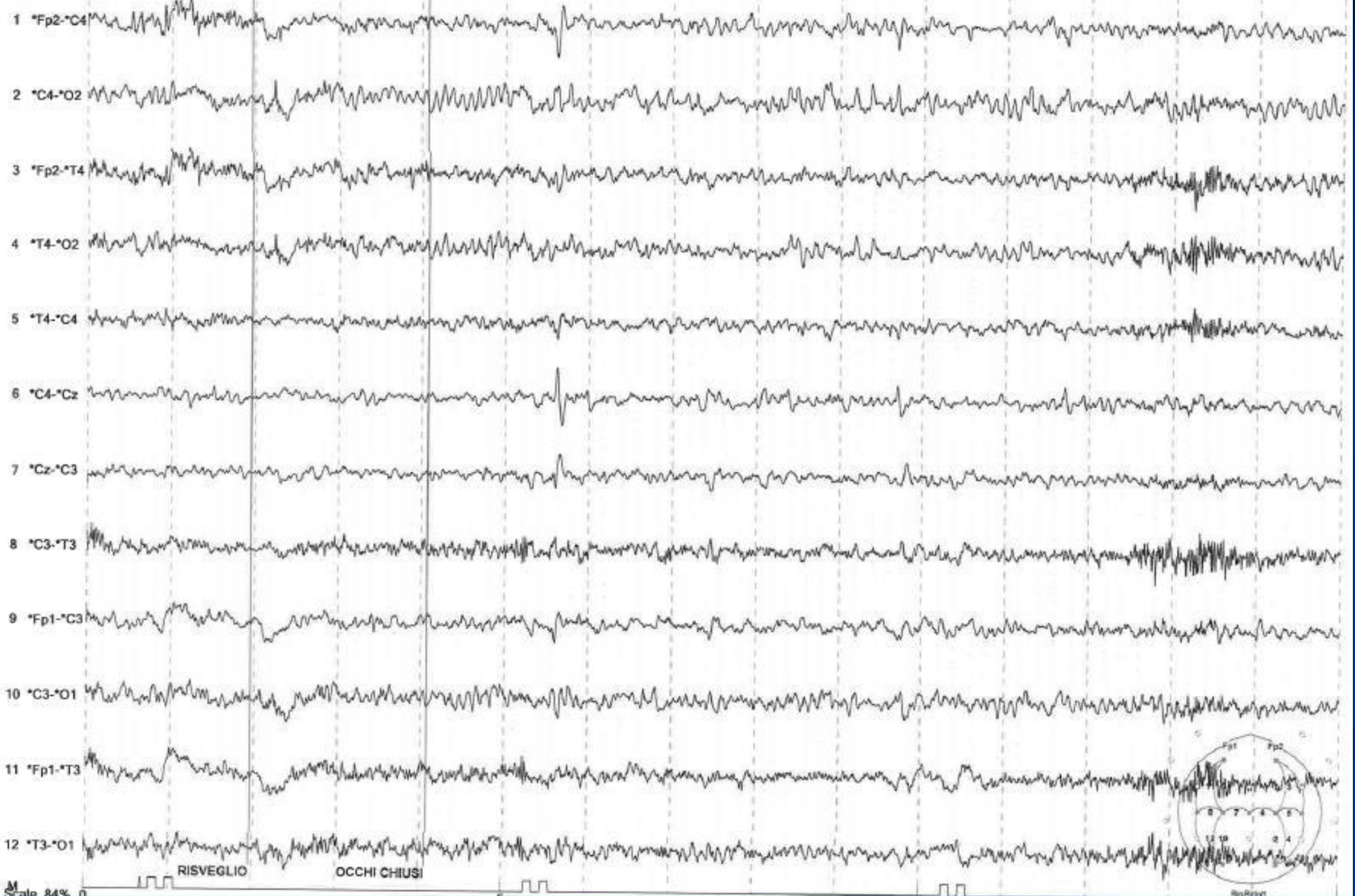
Caso clinico

- Riccardo 8 anni
- Cefalea tensiva da 2 anni
- A 7 anni RMN encefalo: ndr
- Fondo oculare:

Lieve edema papilla bilateralmente in follow up dall'oculista con campo visivo nella norma

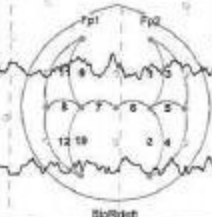
Test diagnostici

- RMN encefalo (fase angio) e midollo
- PEU-PEV
- EEG





SONNO



Che pensare ?

Puntura Lombare

- Pressione liquorale: 480 mmH₂O (norma per i bambini sono generalmente considerati fra i 180 e i 200 mmH₂O, misurati con puntura lombare con paziente in decubito laterale)

Allora siamo di fronte a:

Ipertensione cerebrale benigna

1. Sintomi e/o segni attribuibili ad aumento di pressione endocranica o papilledema
2. Pressione liquorale elevata durante puntura lombare in decubito laterale;
3. Normale composizione liquorale liquor
4. no alterazione RMN
5. no altre cause di ipertensione liquorale accertate come uso di farmaci e/o integratori



ELSEVIER

Brain & Development 35 (2013) 561–568

**BRAIN &
DEVELOPMENT**

Official Journal of
the Japanese Society
of Child Neurology

www.elsevier.com/locate/braindev

Original article

Clinical spectrum of the pseudotumor cerebri in children: Etiological, clinical features, treatment and prognosis

Hüseyin Per^{*}, Mehmet Canpolat, Hakan Gümüş, Hatice Gamze Poyrazoğlu,
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Received 27 May 2012; received in revised form 15 August 2012; accepted 16 August 2012

Abstract

Objective: Pseudotumor cerebri (PTC) is a clinical condition characterized by signs and symptoms of increased intracranial pressure, such as headache and papilledema. Our aim was to investigate the etiological and clinical features of pseudotumor cerebri (PTC) in children. **Materials and method:** We performed a comprehensive analysis of epidemiology, diagnostic work-up, therapy, and clinical follow-up in 42 consecutive patients. **Results:** Totally 42 patients diagnosed with PTC [27 (64.3%) females and 15 (35.7%) males] were included in the study. The average age of the symptoms onset was 10.79 ± 3.43 years (range from 12 months to 17 years). Obesity was found in eleven (26.2%) of them. Two of the patients had familial mediterranean fever, two of them had posttraumatic PTC. The following diseases were one patient, respectively; mycophenolate mofetil-induced PTC, hypervitaminosis A induced PTC, corticosteroid induced withdrawal due to nephritic syndrome, use of oral contraceptives, Guillain–Barre syndrome, urinary tract infection, varicella-zoster virus infection and dural venous sinus thrombosis associated with otitis media. The most common symptom was headache, recorded in 76.2% of the patients. All patients were treated medically. Three patients in our group also required a ventriculoperitoneal shunt. **Conclusion:** Pseudotumor cerebri is an avoidable cause of visual loss, both in adults and children. Pre-pubertal obese girls are more common. Medical therapy appeared to be successful in treating pediatric PTC in most patients. Nevertheless, despite adequate treatment, children can rarely experience loss of visual field and acuity; thus, prompt diagnosis and management are important.

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Author Manuscript

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Update on the pathophysiology and management of idiopathic intracranial hypertension

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³Department of Neurological Surgery, Emory University, Atlanta, GA 30322

DATI EPIDEMIOLOGICI RELEVANTI PER PATOFSIOLOGIA

- Sesso femminile con obesità
- Farmaci (tetracycline and its derivatives, cyclosporine, lithium, nalidixic acid, nitrofurantoin, oral contraceptives, levonorgestrel, danaxol, and tamoxifen)
- Obstructive sleep apnea (OSA)

Idiopathic intracranial hypertension as an initial presentation of systemic lupus erythematosus.

Maloney K.

+ Author information

Abstract

A 14-year-old girl with no known illness presented with a several week history of headaches and vomiting. The patient also reported having joint pain and swelling to the wrists and knees. She had no prior history of headaches, use of hormonal contraception or other medications, recent weight changes or family history of autoimmune disease. Blood pressure temperature, height and weight were normal. She was alert, there was alopecia, cervical lymphadenopathy, symmetrical synovitis to the wrists, bilateral papilloedema and cranial nerve VI palsy. Laboratory investigations revealed a normochromic normocytic anaemia, leucopenia and lymphopenia. Serum chemistries were normal. CT of the brain was normal. Lumbar puncture revealed an opening pressure of greater than 300 mm H₂O; cerebrospinal fluid (CSF) analysis was normal. HIV antibodies were non-reactive. Despite treatment with acetazolamide she developed somnolence. Hence MR venography was performed which showed no evidence of cerebral vein thrombosis. Further investigations revealed a positive direct coombs test, positive antinuclear antibodies (ANA) positive antidouble-stranded DNA (dsDNA) and false positive VDRL. Complement levels were reduced. Anti-Smith, anticardiolipin antibodies and lupus anticoagulant were negative.

[Thrombosis of the cerebral venous sinuses in the differential diagnosis of idiopathic intracranial hypertension in young obese females: is it a real problem?].

[Article in Spanish]

Cabrera-Naranjo F¹, Gonzalez-Hernandez A, Laqoa-Labrador I, Fabre-Pi Ó.

⊕ Author information

Abstract

INTRODUCTION: Idiopathic intracranial hypertension (IIH) typically presents in young women with obesity or a recent weight increase. The differential diagnosis of IIH includes thrombosis of the cerebral venous sinuses (TCVS), which can also present as an isolated intracranial hypertension syndrome. We review the frequency with which patients with a typical IIH profile presented TCVS as their diagnosis.

PATIENTS AND METHODS: The study consisted in a retrospective review of all the admissions due to intracranial hypertension syndromes in our centre between 2000 and 2011. The cases selected were those with a normal cerebrospinal fluid study and computerised axial tomography scan of the head that presented as an isolated intracranial hypertension syndrome; those who manifested a focal neurological picture, however, were excluded. From the patients that were included, a subgroup made up of females between 16 and 35 years of age with a body mass index of above 25 were selected.

RESULTS: A total of 37 cases were obtained. Of these, 35 (94.6%) were cases of IIH and two (5.4%) were TCVS. The time elapsed between the onset of symptoms and diagnosis was less than seven days in both cases of TCVS (100%) and in two cases (5.4%) of IIH.

CONCLUSIONS: Up to 5.4% of patients with a typical IIH profile that present with an intracranial hypertension syndrome can present TCVS. The presence of prothrombotic factors and a high D-dimer can suggest this possibility, although there is still a need for well-established parameters that allow decisions to be made in emergencies in the absence of any chance of performing an urgent MR phlebography scan.

Indian J Pharmacol. 2013 Jan-Feb;45(1):89-90. doi: 10.4103/0253-7613.106444.

Pseudotumor cerebri in a child treated with acitretin: a rare occurrence.

Sarkar S¹, Das K, Roychoudhury S, Shrimal A.

⊕ Author information

Abstract

Pseudotumor cerebri (PTC) is a rare neurological disorder characterized by increased intracranial pressure in absence of any intra-cranial space-occupying lesion. It is mostly due to impairment of drainage of CSF from arachnoid villi. Clinically pseudotumor cerebri presents with headache, diplopia, nausea, vomiting, papilloedema and if treatment is delayed, may lead to blindness. Females of childbearing age group, endocrinal abnormalities and ingestion of certain drugs have been reported to be associated with pseudotumor cerebri. However, it's occurrence in relation to acitretin ingestion has not been reported on pubmed database. Here we present a case where significant temporal association of acitretin intake with PTC was found in a child who was being treated with this medication for recalcitrant pustular psoriasis. The case is reported for its rarity in occurrence and associated significant morbidity including visual loss if not diagnosed and treated immediately. According to Naranjo ADR Causality scale of adverse drug reaction, the association of PTC due to acitretin in our case was probable.

Criteria Diagnostici

Diagnostic criteria for pediatric PTC.	
Modified dandy criteria for diagnosis of PTC [3,4]	Diagnostic criteria for pediatric PTC adapted from Rangwala [5]
<ol style="list-style-type: none">1. Signs and symptoms of increased intracranial pressure2. No localizing findings on neurological examination3. Normal MRI/CT brain scans with no evidence of central venous sinus thrombosis4. Increased intracranial pressure over 250 mm H₂O and normal cerebrospinal fluid composition5. No other identified cause of intracranial hypertension	<ol style="list-style-type: none">1. Pre-pubertal2. Symptoms or signs of generalized intracranial hypertension or papilledema. Normal mental status3. Documented elevated intracranial pressure Neonates: >76 mm H₂O Age less than 8 with papilledema: >180 mm H₂O Age 8 or above or less than 8 without papilledema: >250 mm H₂O4. Normal CSF composition except in neonates who may have up to 32 WBC/mm³ and protein as high as 150 mg/dl5. No evidence of hydrocephalus, mass, structural, or vascular lesion on MRI, with and without contrast, and MR venography. Narrowing of the transverse sinuses is allowed6. Cranial nerve palsies allowed if they are of no other identifiable etiology and improve with reduction in cerebrospinal fluid pressure or resolution of other signs and symptoms of intracranial hypertension7. No other identified cause of intracranial hypertension

CLINICA

Clinical features	Etiopathology/imaging correlate
Common	
Postural headache	Traction on pain sensitive structures due to downward displacement
Nausea, vomiting	Meningeal irritation/tonsillar descent
Posterior neck pain	Meningeal irritation
Less common	
Diplopia	Stretching of III/VI cranial nerve
Changes in hearing, hyperacusis, tinnitus, a sensation of "fullness"	Stretching VIII cranial nerve ^[3,17]
Giddiness, vertigo	Altered intralabyrinthine pressure
Visual blurring	Stretching of the optic nerve/chiasm ^[18]
Facial numbness	Stretching of VII nerve
Radicular symptoms involving the upper limb	Stretching of cervical nerve roots
Rare	
Coma	Hind brain herniation ^[19]
Parkinsonism	Mid brain compression
Dementia	Frontal and temporal cortices compression due to downward brain "sagging"
Quadriplegia	Prolonged engorgement of spinal epidural plexus
Hypopituitarism	Pituitary hyperemia

The optic disc is minimal in children with idiopathic intracranial hypertension.

[Dai S¹](#), [Trimboli C](#), [Buncic JR](#).

⊕ Author information

Abstract

This study sought to characterize the optic disc morphology, particularly the cup-to-disc ratio of the optic nerve head in children with idiopathic intracranial hypertension. The medical charts and digital optic disc photos of children with confirmed diagnosis of idiopathic intracranial hypertension were reviewed retrospectively. The optic disc area, cup area, and cup-to-disc ratio were measured digitally using VISUPAC software, and the mean values of those parameters were compared to the published norms. Of children with idiopathic intracranial hypertension, 83% had absence of the physiological cup of the optic disc, compared to 10% of children in the general population of the same age. The median disc area was 2.2 mm², and median cup area was 0.0mm², compared to the published norms of 2.69 mm² and 0.44 mm², respectively. There is very significantly high prevalence of small optic disc cups in children with idiopathic intracranial hypertension, with the cup being absent on majority of cases in our patient cohort. This may signal an underlying systemic predisposition to the development of intracranial hypertension.

KEYWORDS: idiopathic increased intracranial pressure; optic disc size; pseudotumor cerebri

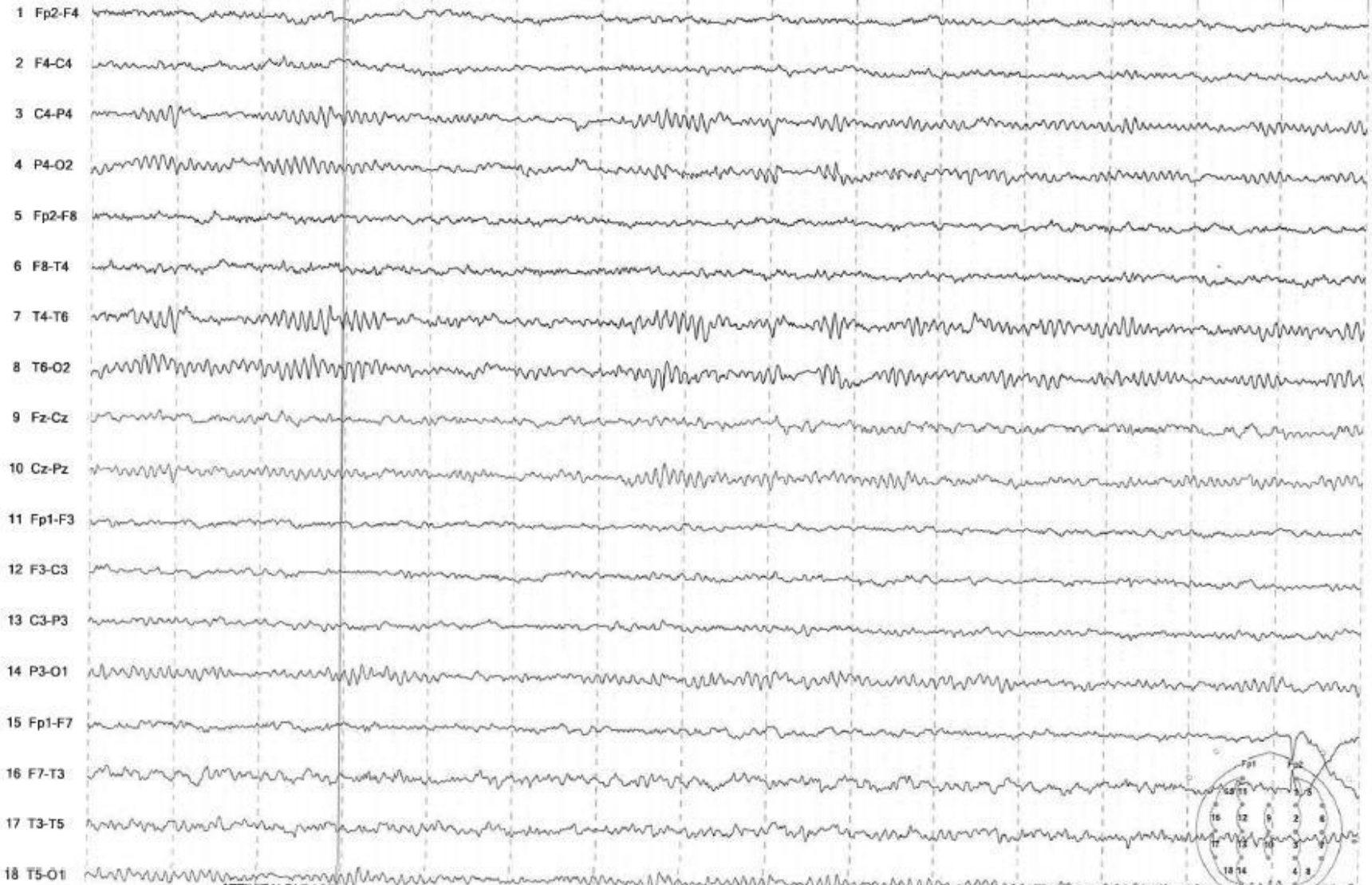
Dopo 3 mesi di Acetazolamide

- Clinicamente: non cefalea
- EEG ?

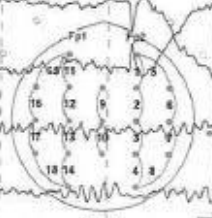
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100uV
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Patt. Long G ACFill ON Refer. OFF Reset OFF

Data: 25/01/2013 ID: 1234568015
Nome Riccardo Di Bella



ATTIVITA' DI BASE



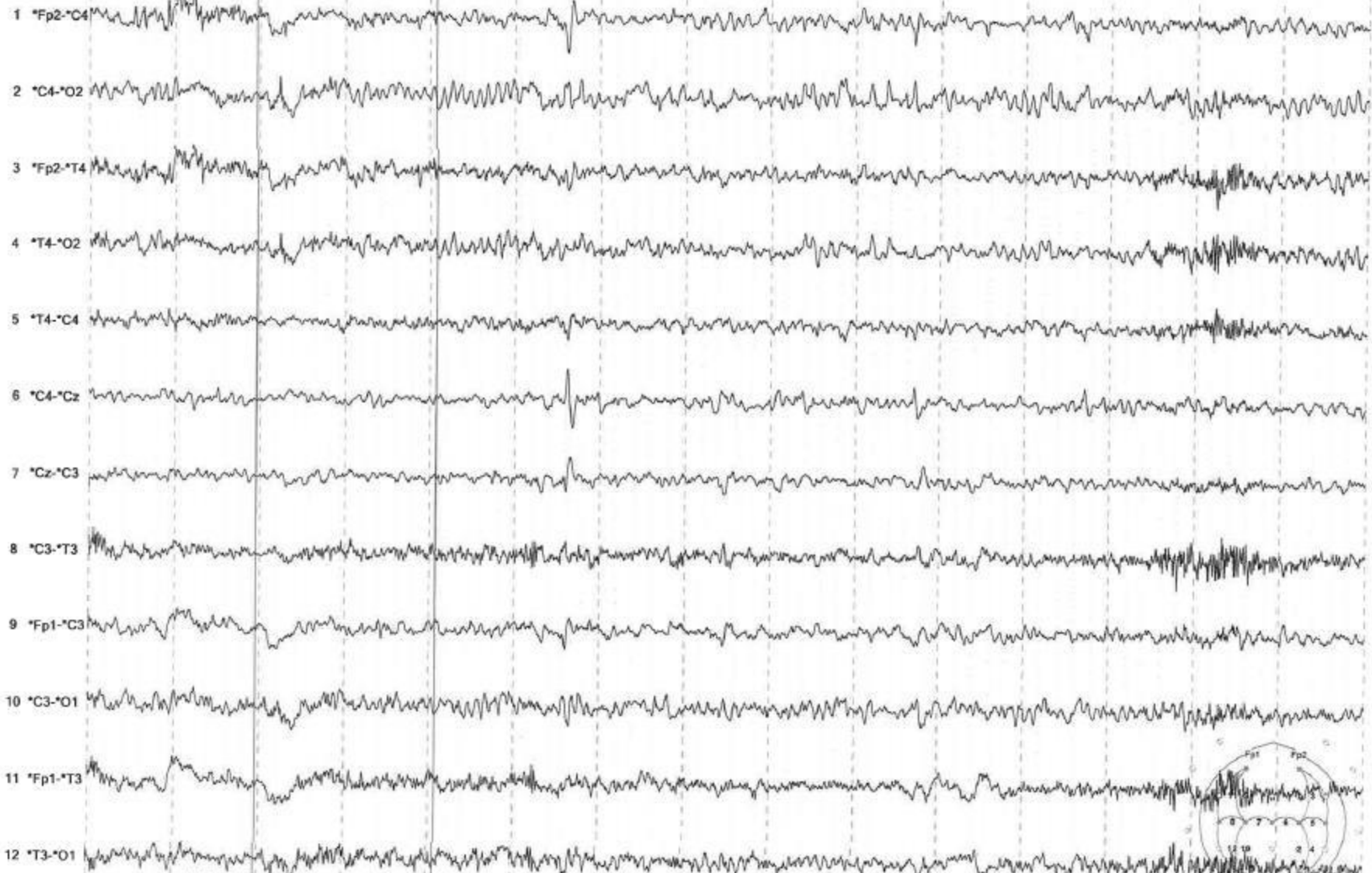
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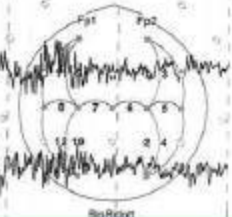
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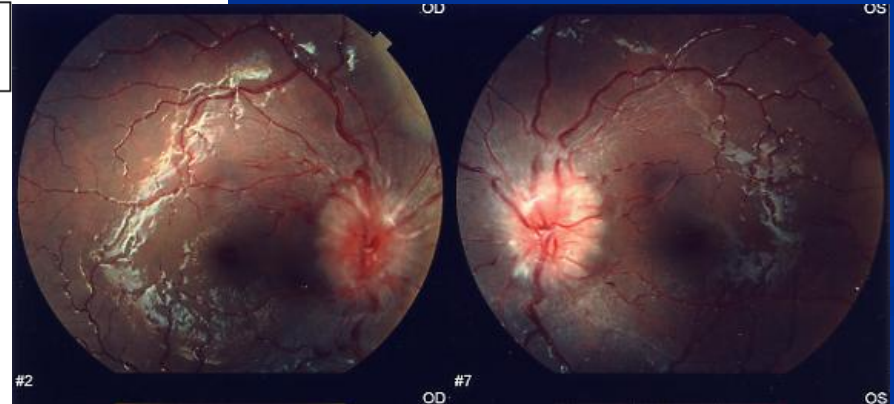
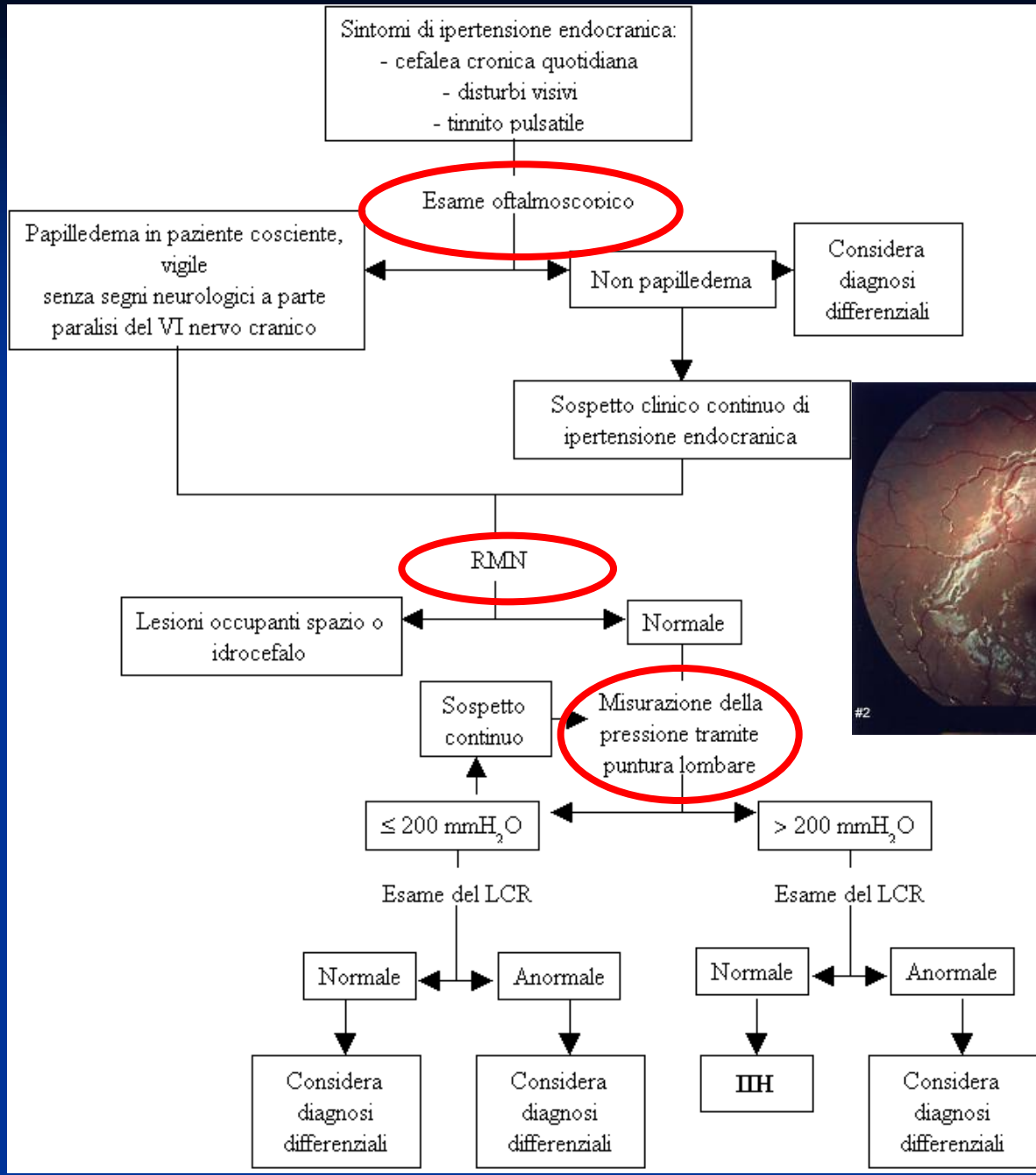
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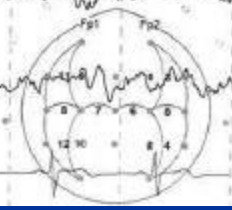
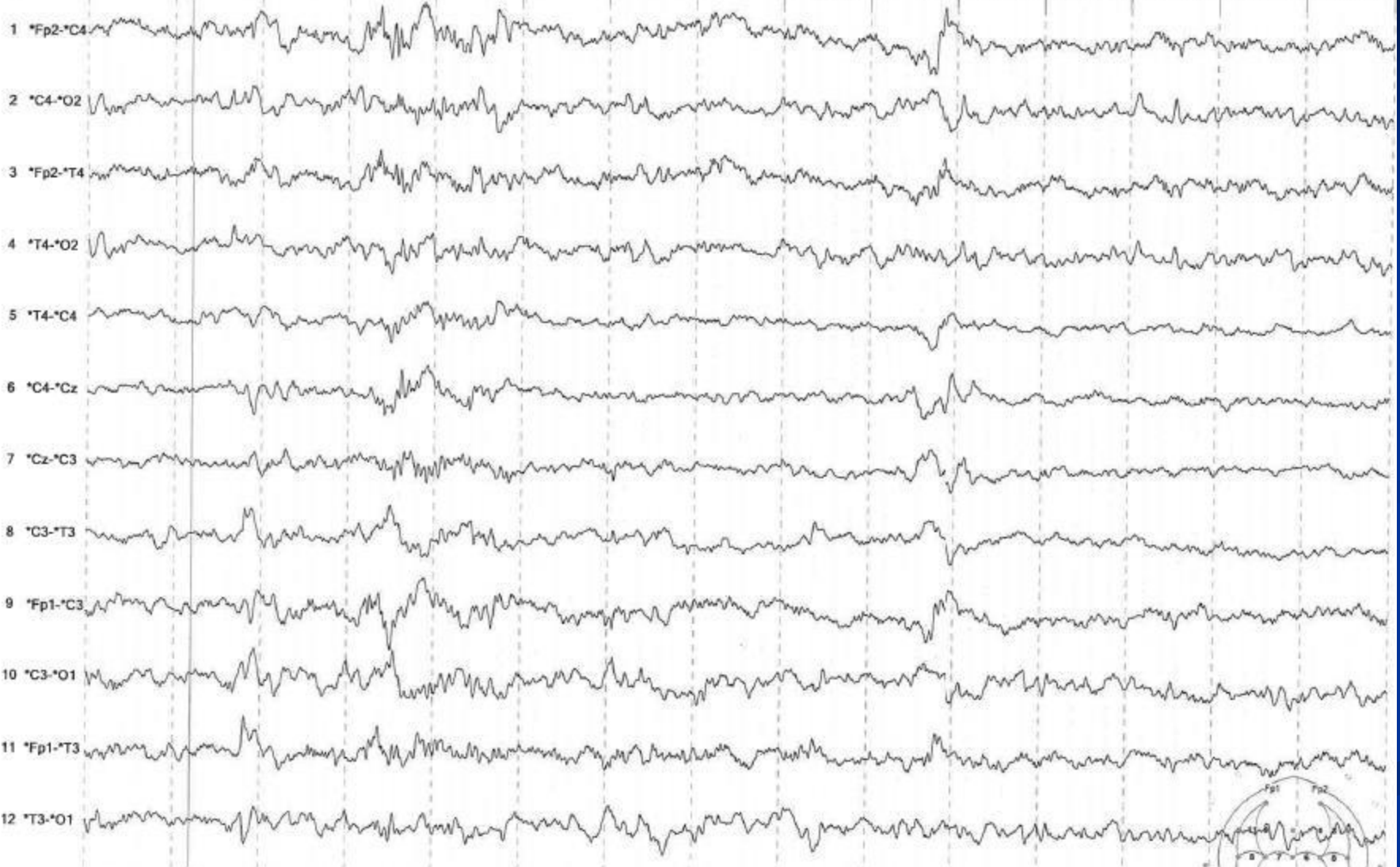
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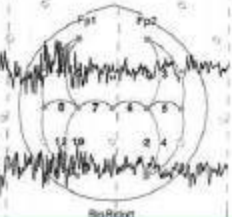
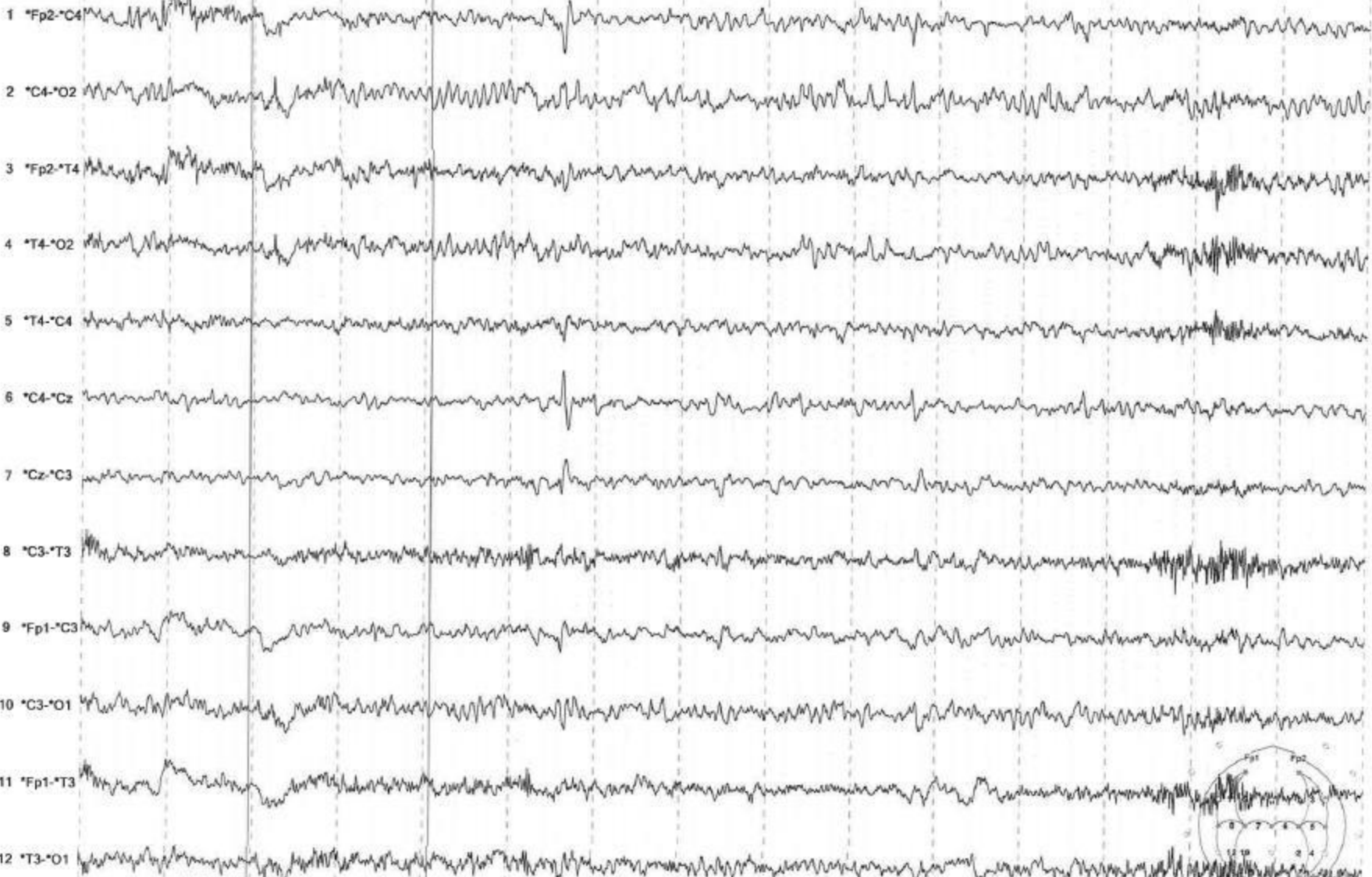


RISVEGLIO OCCHI CHIUSI









RISVEGLIO OCCHI CHIUSI

ATTENZIONE!

MEDICINA

SE FAI SESSO TI VIENE IL MAL DI TESTA

Una volta erano in tanti a dire: se di sera non faccio sesso mi viene il mal di testa. Altri aggiungevano di non riuscire a prendere sonno. Una volta, quando erano aperte le case di tolleranza, era facile provvedere, oggi è più problematico. Comunque niente paura: secondo i medici è esattamente il contrario perché ci sono uomini che durante l'amplesso rischiano di procurarsi una forte emicrania, che aumenta in parallelo con l'aumentare dell'eccitazione (ma perché le donne no?). In questi casi si diventa rossi paonazzi, il cuore batte troppo forte, insomma qualcuno ci può restare secco. Quante volte abbia-

GIORGIO POLI

mo sentito di una persona che muore durante l'atto sessuale?

Spiega il prof. Pinessi: «Si tratta di una crisi emicranica scatenata dall'aumento di pressione arteriosa e della frequenza cardiaca. Nella metà dei casi interessa uomini emicranici e più frequentemente gli ipertesi e quelli che soffrono di cefalea da sforzo. Questa cefalea va indagata bene perché nel 3-4% dei soggetti può essere causata da un piccolo aneurisma cerebrale».

Mamma mia. Questo tipo di studi medici fa sorgere paure che una volta non esistevano. Oggi se

fai sesso devi stare attento non solo alle più banali malattie veneree, ma anche a non beccarti l'Aids per cui i giovani vanno in giro con i risultati delle analisi cliniche da dove risultano essere «negativi».

Se riesci ad evitare con le dovute precauzioni il rischio del contagio, e già sei fortunato, ora devi stare attento pure all'emicrania. Perché se vai troppo forte puoi entrare in crisi di cefalea, se non proprio in crisi cardiaca. Ma allora un uomo che deve fare, provvedere da solo come consigliava quel parroco di un paesino siciliano?

ALTRO SERVIZIO PAG. 13



EMICRANIA

Forme cliniche

- E. senza aura
- E. con aura :
 - classica
 - complicata
- E. varianti

Cefalea di Tipo Tensivo Episodica Sporadica/ Frequente

Criteria diagnostici

- A. **Almeno 10 episodi che si verificano in media < 1 giorno al mese (< 12 giorni all'anno)** **Almeno 10 episodi che si verificano in media > 1 giorno ma meno di 15 giorni al mese per almeno 3 mesi (> 12 e < 180 giorni all'anno)** e che soddisfino i criteri B-D
- B. **La cefalea dura 30' a 7 giorni**
- C. **La cefalea presenta almeno due delle seguenti caratteristiche:**
 - 1. **Localizzazione bilaterale**
 - 2. **Qualità gravativo-costrittiva (non pulsante)**
 - 3. **Intensità lieve o media**
 - 4. **Non è aggravata dall'attività fisica di routine**
- D. **Si verificano entrambe le seguenti condizioni:**
 - 1. **Assenza di nausea e vomito (può manifestarsi anoressia)**
 - 2. **Può essere presente fotofobia o fonofobia, ma non entrambe**
- E. **Non attribuita ad altra condizione o patologia**

Quando una cefalea di tipo tensivo si verifica per più di 15 giorni al mese per più di 3 mesi → **Cefalea Tensiva Cronica**



EMICRANIA CON AURA

■ Classica

■ Complicata:- e. emiplegica

- e. oftalmoplegica
- e. dell'arteria basilare
- stato confusionale
- s. di Alice nel paese delle meraviglie



EMICRANIA EMIPLEGICA

Si manifesta con sintomi motori e sensitivi che coinvolgono un emilato.

In alcune forme familiari a trasmissione AD si riscontra una mutazione del gene del canale del cloro *CACN LA4* (cromosoma 19p13)



E. Oftalmoplegica

Sintomi: dolore unilaterale, orbitale con paralisi parziale o completa del 3° nervo cranico.

Strabismo, ptosi, diplopia, midriasi possono essere presenti.



Emicrania dell'arteria basilare

Più frequente nel sesso femminile.
Sintomi: deficit bilaterale del campo visivo, visione ristretta, offuscata.
Si associano atassia, disartria, vertigini, perdita di coscienza.



Stato confusionale

Si manifesta con irrequietezza ,
agitazione, sensorio alterato, ridotta
risposta agli stimoli dolorosi.



S. di “Alice nel paese delle meraviglie”

Le immagini appaiono distorte, con relazioni spaziali e temporali alterate con micropsia, metamorfopsie, allucinazioni olfattorie, uditive e gustative.

Ischemia del lobo parietale



EMICRANIA

Varianti





Il rapporto S. periodiche-Eemicrania è basato sui seguenti elementi:

- Familiarità positiva per emicrania
- Evoluzione delle s. periodiche in emicrania dell'adulto

Emicranie complicate - Emicranie varianti

D.D. con Crisi epilettiche

1) Dati anamnestici:

- Descrizione dettagliata degli episodi
- Fattori scatenanti
- Pattern psichico
- Familiarità

2) EEG



CEFALEA SECONDARIA

Cause Neurologiche

- Trauma cranico
- Emorragie endocraniche
- Trombosi
- Meningo-encefaliti
- Tumori endocranici
- Idrocefalo
- Pseudotumor cerebri

Cause non neurologiche

- Malattie sistemiche
- Malattie vascolari
- Malattie infettive
- Malattie endocrine
- Malattie oculari
- Malattie dentali
- Malattie muscolari
- Malattie articolari
- Malattie del collo
- Malattie del torace
- Malattie del sistema circolatorio
- Malattie del sistema respiratorio
- Malattie del sistema digerente
- Malattie del sistema urinario
- Malattie del sistema riproduttivo
- Malattie del sistema circolatorio periferico
- Malattie del sistema circolatorio cerebrale
- Malattie del sistema circolatorio vertebrale
- Malattie del sistema circolatorio cranico
- Malattie del sistema circolatorio cervicale
- Malattie del sistema circolatorio toracico
- Malattie del sistema circolatorio addominale
- Malattie del sistema circolatorio pelvico
- Malattie del sistema circolatorio periferico
- Malattie del sistema circolatorio cerebrale
- Malattie del sistema circolatorio vertebrale
- Malattie del sistema circolatorio cranico
- Malattie del sistema circolatorio cervicale
- Malattie del sistema circolatorio toracico
- Malattie del sistema circolatorio addominale
- Malattie del sistema circolatorio pelvico

CASO CLINICO

V.F., 12 anni, M

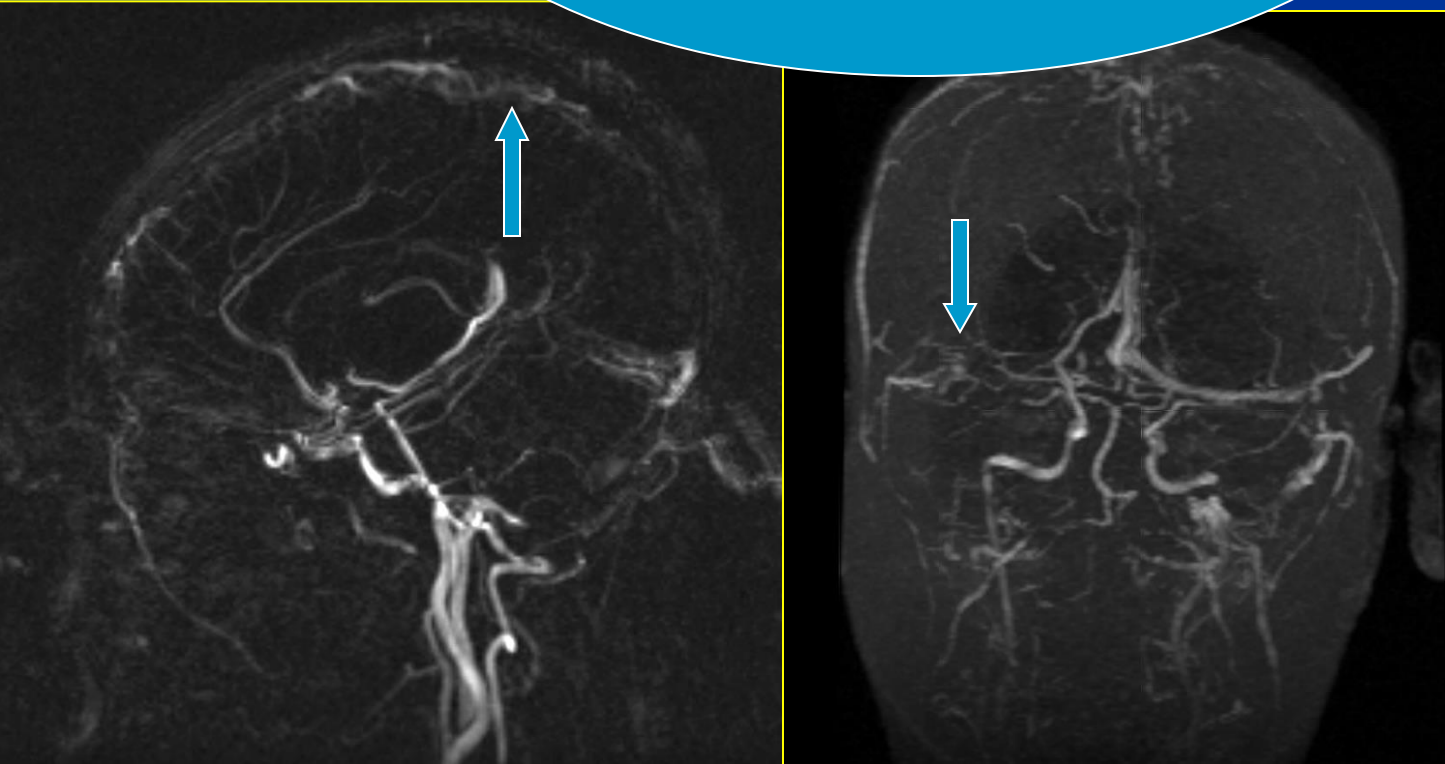
Da 3 settimane cefalea ingravescente di media entità al vertice

Da 1 settimana acufeni, diplopia

E.O.: aspetto lievemente disteso, FC 100 batt/min

Laboratorio: deficit

TROMBOSI DEI SENI VENOSI CEREBRALI



ANGIO-RM
VENOSA:
Mancata
visualizzazione
del seno sagittale
superiore e del
seno trasverso-
sigmoideo di dx



ITER DIAGNOSTICO

- Anamnesi familiare e personale
- Decorso temporale della cefalea
- Esame obiettivo generale
- Esame neurologico

Negativo

- Visita oculistica
- Es. ematochimici

Positivo

- Es. ematochimici
- Visite specialistiche
(ORL, fundus)
- TC; RMN; AngioRM
- Ecocolordoppler
- Esame liquor

QUANDO RICHIEDERE
RMN - ENCEFALO?



SINTOMI D'ALLARME

- Episodi più frequenti e di maggiore intensità
- Nausea e vomito soprattutto se mattutini
- Alterazioni del comportamento
- Accentuazione del dolore con i cambiamenti di postura
- Risvegli notturni per la cefalea
- Associazione con crisi epilettiche parziali
- Segni neurologici presenti



Delay in the diagnosis of paediatric brain tumours

in this age group approximates only 3/100,000 (0.003%) [12]. Uncritical imaging would result in a large number of normal neuroimaging studies. In our study, most children with headache as initial symptom showed additional signs/symptoms within a relatively short period. This is consistent with a large study of the Childhood Brain Tumor Consortium [12]. Analysing 3276 patients, less than 3% of children with headache and a brain tumour had no abnormality on neurological examination.

Although not yet proven, a shortened PSI may lead to the detection of smaller brain tumours that are easier to resect. The value of extensive tumour resection, which



ALTRE FORME DI CEFALEA

Cefalea tensiva: crisi di maggior durata e minore intensità, dolore di tipo gravativo e a cerchio. Stato depressivo-ansioso spesso presente

Cefalea a grappolo: crisi subcontinue intervallate da brevi periodi di benessere



**“ESISTE UN RAPPORTO
EMICRANIA / EPILESSIA ?”**



EMICRANIA-EPILESSIA

CO-MORBIDITÀ: lo stesso individuo può presentare, in periodi diversi, sia crisi emicraniche che crisi epilettiche.



EPILESSIA - CEFALEA

La cefalea può presentarsi nel corso di una crisi epilettica come:

- Aura
- Episodio critico (dolore improvviso, breve, lancinante)
- Manifest. post-convulsiva:
 - E. general. di lunga durata
 - E. parziali (EBPR-EBPO)

Caso clinico

D.M.S. 8 aa.

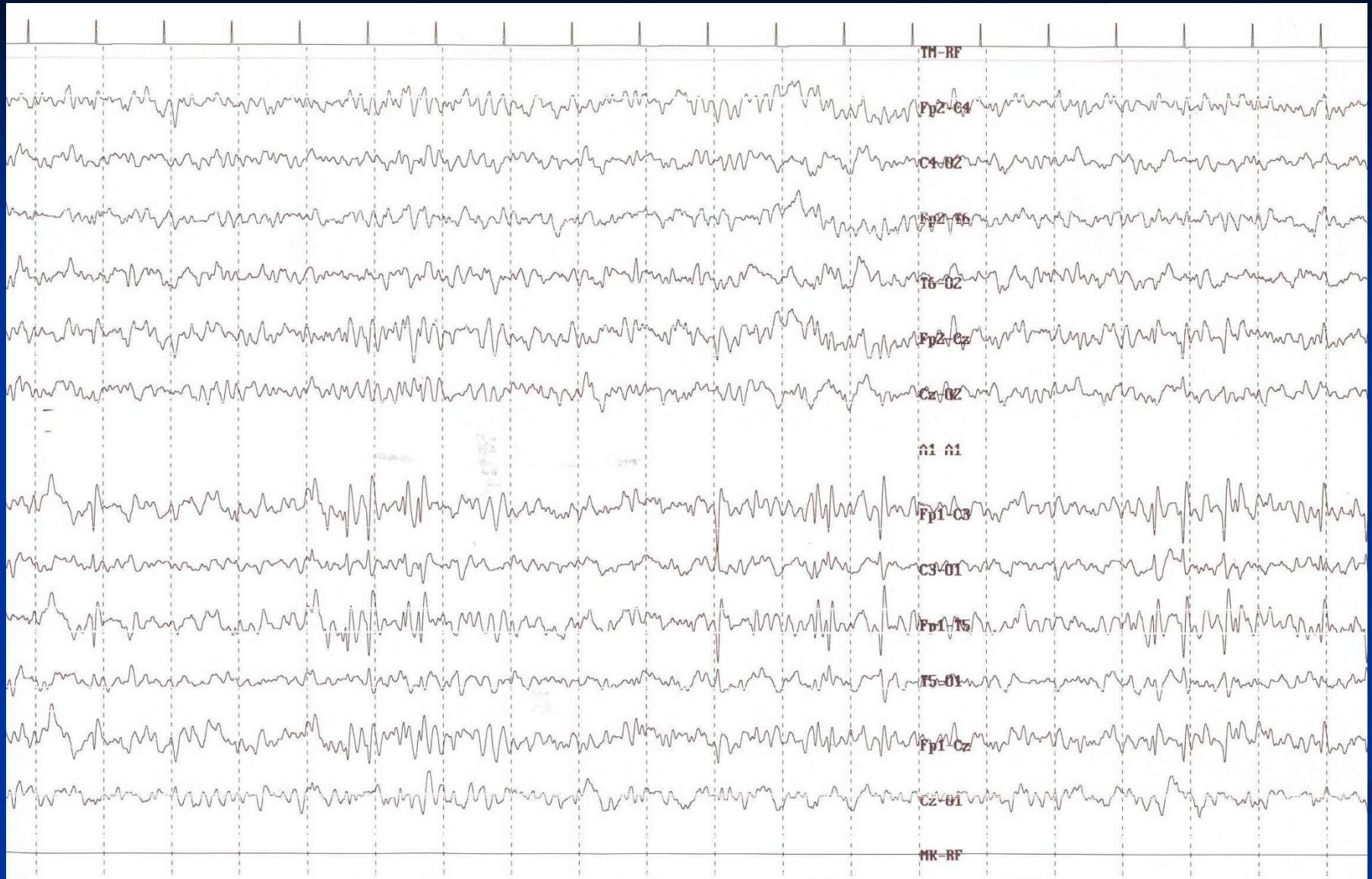
Da circa un anno episodi di cefalea frontale, durata 1-2 ore, frequenza 2-3 episodi al mese, seguita da vomito, a risoluzione spontanea.

La cefalea è prevalentemente pomeridiana.

Visita oculistica: nella norma norma

EEG in veglia: Complessi p/o sulle regioni C-T dell'emisfero di sinistra. Tali grafoelementi risultano a tratti visibili sulle medesime regioni dell'emisfero controlaterale.

EEG in sonno: Complessi p/o sulle regioni C-T dell'emisfero di sinistra. Tali grafoelementi risultano a tratti visibili sulle medesime regioni dell'emisfero controlaterale.





EMICRANIA NELL'INFANZIA PROGNOSI





CEFALEA-PROFILASSI

Valutare attentamente:



CEFALEA CRONICA GIORNALIERA

Classificazione

Emicrania trasformata(e.cronica): e. episodica divenuta persistente.

Cefalea cronica tipo tensivo:

Nuova persistente cefalea giornaliera:

Emicrania continua

CEFALEA CRONICA GIORNALIERA

(Cefalea cronica non progressiva)





E. DEL BAMBINO V S. E. DELL'ADULTO

- Durata più breve
- Bifrontale
- Unilaterale

CEFALEA INFANTILE

Profilassi degli attacchi

- Flunarazina (Flunagen): Ca-antagonista 5mg/die
 - Propanololo (Inderal): beta-bloccante 2-4 mg/Kg/die
 - Ciproeptadina (Periactin): antag. serotonina 0.25-1.25mg/Kg/die
 - Amitriptilina (Laroxyl): antidepressivo 10-25 mg/die serale
 - Topiramato
 - Valproato
 - Gabapentin
- | | | |
|--|---------------------|-----------------|
| | } Anticonvulsivanti | 1-10mg/Kg/die |
| | | 20-40 mg/Kg/die |
| | | 10-40 mg/Kg/die |

CEFALEA INFANTILE

Terapia degli attacchi:

- Ibuprofene (FANS): Nureflex 10 mg/Kg/die
- Paracetamolo: Tachipirina 10-15 mg/Kg/dose
- Sumatriptan nasale (Agonista 5-Ht): Imigran 5-20 mg/dose

Eemicrania

Cortical spreading depression

↑ H⁺ e K⁺

Liberazione di NO e Glutammato

↓ Flusso Cerebrale regionale
+
Attivazione della porzione caudale
del Ganglio Trigeminale

Emicrania

Liberazione di polipeptidi vasoattivi intestinali



Vasodilatazione cerebrale



Stravasamento di proteine dai vasi Durali



Flogosi dei vasi durali di natura neurogenica



Eccitazione dei recettori del dolore



DOLORE

Eemicrania

Cortical spreading depression

↑ H⁺ e K⁺

Liberazione di NO e Glutammato

↓ Flusso Cerebrale regionale
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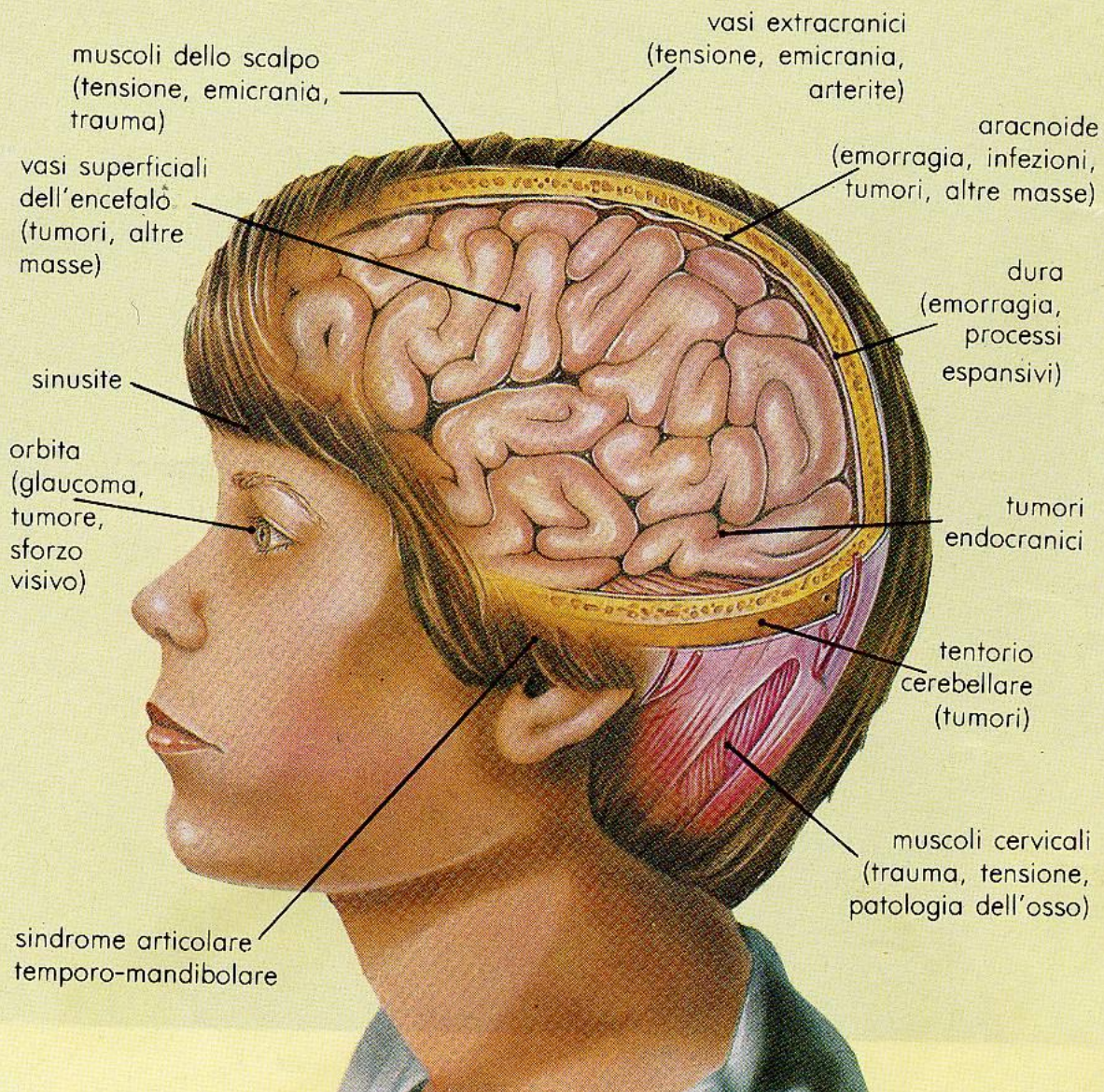
Eccitazione dei recettori del dolore



DOLORE

↓ Flusso Cerebrale regionale
+
Attivazione della porzione caudale del Ganglio Trigeminale







CEFALEA

Trasmissione del dolore

- A. Vasi intracranici sopratentoriali attraverso il n. trigemino
- B. Vasi intracranici sottotentoriali attraverso 3 nervi cervicali



EMICRANIA

➤ Poligenica, multifattoriale.

Coinvolto il gene P/Q tipo canale del calcio $\alpha 1 A$ sub-unità (CACNA1A) localizzato sul cromosoma 19 p13



ITER DIAGNOSTICO

- Anamnesi familiare e personale
- Decorso temporale della cefalea
- Esame obiettivo generale
- Esame neurologico

Negativo

- Visita oculistica
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- Esame liquor

CEFALEA-PROFILASSI

Valutare attentamente:

CEFALEA-PROFILASSI

Non farmacologica

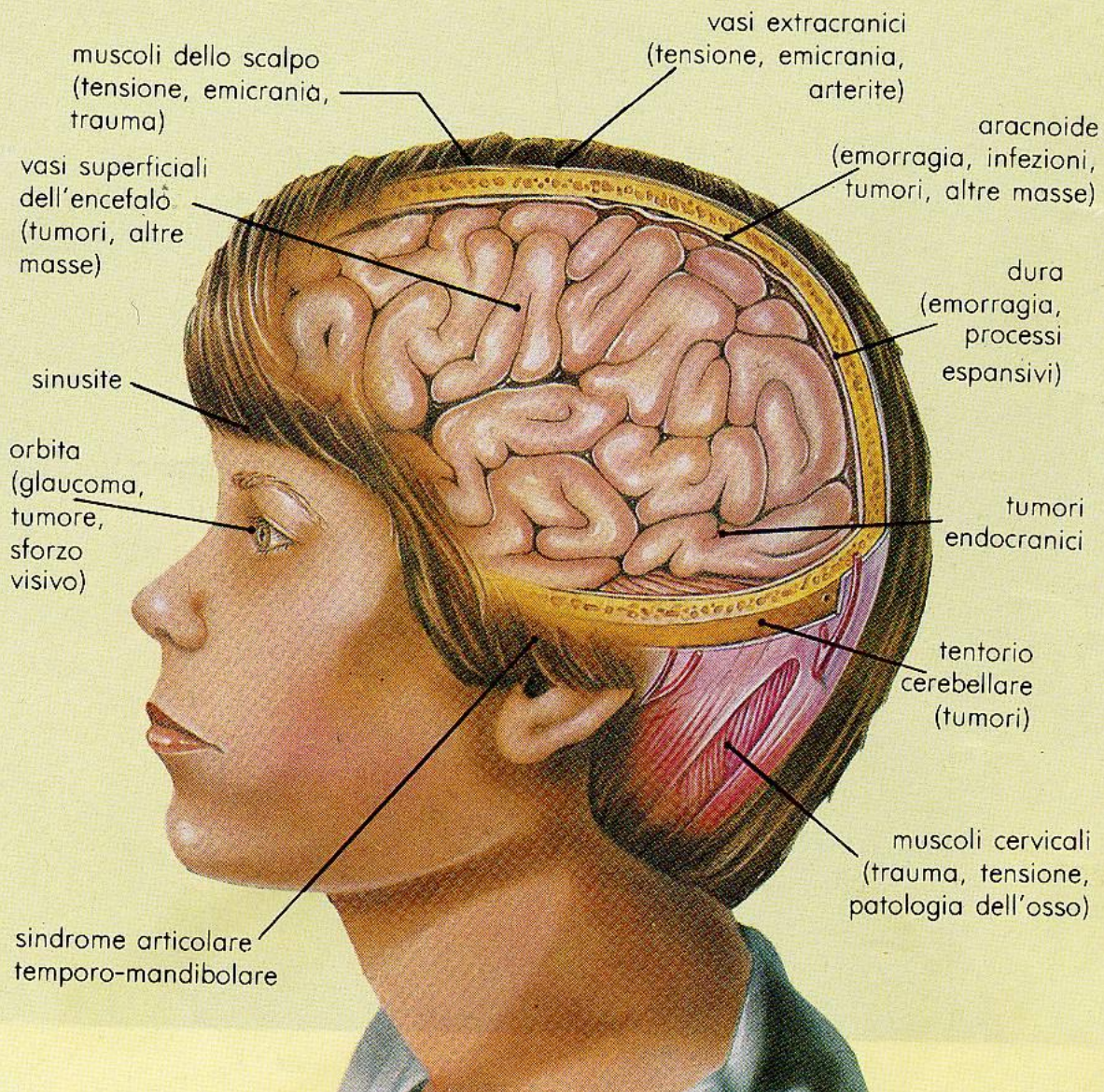
CEFALEA- PROFILASSI

Indicazioni

CEFALEA-PROFILASSI FARMACOLOGICA

01

02



L'EEG è fondamentale nel distinguere episodi epilettici da quelli non epilettici, ma con significativi limiti

Un EEG interictale normale NON esclude la diagnosi di epilessia



Eur J Pediatr (2002) 161: 663–667
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ORIGINAL PAPER

Milana Dobrovoljac · Heinz Hengartner
Eugen Boltshauser · Michael A. Grotzer

Delay in the diagnosis of paediatric brain tumours

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© Springer-Verlag 2002

Abstract The pre-diagnostic period of 252 children (median age 6.3 years, range 0–16.9 years) with primary brain tumours was assessed to analyse their clinical presentation and reasons for any delay in diagnosis. The median pre-diagnostic symptomatic interval (PSI) was 60 days (range 0–3010 days) with a parental delay of 14 days (range 0–2310 days) and a doctor's delay of 30 days (range 0–3010 days). Only 33% of brain tumours were diagnosed within the 1st month after the onset of signs/symptoms. PSI correlated significantly with patients' age and tumour histology, but not with gender, year of di-

Abbreviations *ICP* intracranial pressure · *PSI* pre-diagnostic symptomatic interval

Introduction

As a group, brain tumours are the most common solid tumours in children [5]. They differ from primary central nervous system tumours occurring in adults not only in histology, localization, management and prognosis, but also in clinical presentation [6]. At the onset of illness



Table 1 Frequency of initial signs and symptoms depending on age

Signs and symptoms	All (n = 252)	Age < 2 years (n = 50)	Age ≥ 2 years (n = 202)
Headache	35%	2%	43%
Nausea/vomiting	26%	18%	28%
Seizures	14%	20%	12%
Behavioural changes (irritability, mood, character, school)	10%	12%	9%
Ataxia	8%	8%	8%
Squint/diplopia	8%	6%	8%
Lethargy	5%	4%	5%
Hemiparesis/quadriparesis	5%	8%	4%
Head tilt	5%	12%	3%
Anorexia	3%	6%	2%
Growth failure	3%	—	3%
Sleep disturbance	2%	2%	2%
Polyuria/polydipsia	2%	—	3%
Visual loss	2%	2%	2%
Weight loss	2%	4%	1%
Facial nerve palsy	2%	4%	1%
Enlargement of the head	2%	8%	—
Cranial neuropathies other than III, IV, VI, VII	1%	—	1%
Gaze depression/separation of cranial sutures/bulging fontanelle	1%	4%	—
Dizziness	1%	—	1%
Nystagmus	1%	4%	—
Papilloedema	1%	—	1%
Amenorrhoea	0.5%	—	0.5%
Proptosis	0.5%	—	0.5%



Table 2 Frequency of signs and symptoms at diagnosis depending on age

Signs and symptoms	All (n = 252)	Age < 2 years (n = 41)	Age ≥2 years (n = 211)
Nausea/vomiting	60%	54%	61%
Headache	54%	5%	64%
Ataxia	46%	17%	52%
Hemiparesis/quadriparesis	35%	37%	35%
Squint/diplopia	35%	17%	38%
Papilloedema	35%	10%	39%
Seizures	21%	27%	19%
Behavioural changes (irritability, mood, character, school)	22%	29%	20%
Gaze depression/separation of cranial sutures/bulging fontanelle	15%	41%	10%
Cranial neuropathies other than III, IV, VI, VII	15%	15%	16%
Facial nerve palsy	14%	10%	15%
Head tilt	14%	20%	13%
Lethargy	13%	15%	13%
Visual loss	13%	15%	13%
Weight loss	12%	15%	12%
Nystagmus	11%	5%	12%
Decreased level of consciousness/drowsiness	9%	10%	9%
Enlargement of the head	9%	32%	5%
Visual field defects	6%	–	8%
Sleep disturbance	6%	10%	5%
Hemisensory loss	4%	–	5%
Anorexia	4%	10%	2%
Growth failure	4%	–	4%
Polyuria/polydipsia	3%	–	4%
Dizziness	2%	–	3%
Temperature regulation disturbance	2%	5%	2%
Parinaud syndrome	1%	–	1%
Amenorrhoea	1%	–	1%
Dysarthria	1%	–	1%
Proptosis	1%	2%	0.5%
Bulimia	1%	–	1%
Sexual precocity	1%	–	1%
Tinnitus	0.5%	–	0.5%



EMICRANIA NELL'INFANZIA

A

B

Eemicrania

Cortical spreading depression



↑ H⁺ e K⁺



Liberazione di NO e Glutammato



↓ Flusso Cerebrale regionale

+

Attivazione della porzione caudale
del Ganglio Trigeminale

Emicrania

Liberazione di polipeptidi vasoattivi intestinali



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DOLORE

Emicrania

