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Neurology

- No hay artículos sobre cefalea-migraña publicados este mes



Neurología

- [Importancia de la migraña crónica en una consulta general de neurología](#)

M. Gracia-Naya, R. Alarcia-Alejos, P.J. Modrego-Pardo

[REV NEUROL 2008; 46:577-581] PMID: 18465695 - Fecha de publicación: 15/05/2008



British Medical Journal (BMJ)

- No hay artículos sobre cefalea-migraña publicados este mes



JAMA

- No hay artículos sobre cefalea-migraña publicados este mes



New England Journal of Medicine

- No hay artículos sobre cefalea-migraña publicados este mes



Lancet

- No hay artículos sobre cefalea-migraña publicados este mes

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Headache Progress in Canada over the Decades

Werner J. Becker,
Headache: The Journal of Head and Face Pain Volume 48 Issue 5 Page 678-679, May 2008

Abstract

As elsewhere in the world, migraine and other headache disorders have always produced very significant disability amongst Canadians. Over the last 50 years, progress has been made by health professionals to improve the care received by patients with headache, and to reduce the headache-related burden carried by patients and their families. Milestones in this progress have included programs for better education for the public, for neurologists, and for other physicians about migraine. Highlights in the Canadian battle against migraine and other headaches include those listed below:

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The International Classification of Headache Disorders

Jes Olesen.

Headache: The Journal of Head and Face Pain Volume 48 Issue 5 Page 691-693, May 2008

Abstract

A set of related medical disorders that lack a proper classification system and diagnostic criteria is like a society without laws. The result is incoherence at best, chaos at worst. For this reason, the International Classification of Headache Disorders (ICHD) is arguably the single most important breakthrough in headache medicine over the last 50 years. The ICHD identifies and categorizes more than a hundred different kinds of headache in a logical, hierarchal system. Even more important, it has provided explicit diagnostic criteria for all of the headache disorders listed. The ICHD quickly became universally accepted, and criticism of the classification has been minor relative to that directed at other disease classification systems. Over the 20 years following publication of the first edition of the ICHD, headache research has rapidly accelerated despite sparse allocation of resources to that effort. In summary, the ICHD has attained widespread acceptance at the international level and has substantially facilitated both clinical research and clinical care in the field of headache medicine.

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Migraine Genetics: A Fascinating Journey Towards Improved Migraine Therapy

Michel D. Ferrari.

Headache: The Journal of Head and Face Pain Volume 48 Issue 5 Page 697-700, May 2008

Abstract

The study of migraine genetics promises to deliver significant changes to migraine therapy. Dr. Ferrari explores the emergence of the field and its importance to headache medicine.

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Fifty Years of Headache Care Evolution: From a Few Interested Doctors to Certified Specialists and Comprehensive Systems

Joel R. Saper

Headache: The Journal of Head and Face Pain Volume 48 Issue 5 Page 701-703, May 2008

Abstract

Headache care has evolved dramatically during the lifetime of the American Headache Society. Dr. Saper charts the most important developments and recognizes those instrumental in influencing current thought and practice.

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Functional Imaging in Primary Headache Disorders

F. Michael Cutrer,
Headache: The Journal of Head and Face Pain Volume 48 Issue 5 Page 704-706, May 2008

Abstract

Over the past two decades, the development of new functional neuroimaging techniques has improved our understanding of the brain events underlying several primary headache disorders. In migraine and cluster headache, the advent of these techniques has shifted the emphasis in pathophysiological research away from the vessel and back to the brain.

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Headache and Behavioral Medicine: A 50-Year Retrospective

Alvin E. Lake.

Headache: The Journal of Head and Face Pain Volume 48 Issue 5 Page 714-718, May 2008

Abstract

This article reviews all behaviorally oriented articles published in Headache from 1961(1:1) through the first 3 issues of 2008 and provides an analysis of trends in categories of articles by decade. A mean of 21.6% of all articles included significant attention to behavioral variables; this percentage was relatively stable from 1980 through 2008. The top 5 categories, accounting for 64% of all behavioral articles since the inception of Headache, were: behavioral treatment (19.2%), psychiatric comorbidity (14.0%), psychophysiology (11.2%), behavioral risk factors (9.8%), and psychobiological concepts (9.8%). There is an accelerating trend toward publication of articles related to psychiatric comorbidity, behavioral risk factors, and functional performance/disability.

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A Knockout Punch: C. Miller Fisher's Migraine Accompaniments

William B. Young.

Headache: The Journal of Head and Face Pain Volume 48 Issue 5 Page 726-727, May 2008

Abstract

Occasionally patients in the stroke age-bracket over 40 years have unexplained transient cerebral ischemic attacks in association with normal cerebral angiograms. From this group, 120 have been collected in whom the transient episodes resembled the neurological accompaniments of migraine. According to symptoms, the patients were categorized as follows: visual accompaniments (patients with only ordinary scintillating scotoma were excluded), 25; visual and paresthesias, 18; visual and speech disturbance, 7; visual, and brain stem symptoms, 14; visual, paresthesias, and speech disturbance, 7; visual, paresthesias, speech disturbance, and paresis, 25; recurrence of old stroke deficit, 9; miscellaneous, 8. In establishing the diagnosis, angiography is advisable in all but classical cases. Typical of migrainous accompaniments are the build-up and migration of visual scintillations, the march of paresthesia, and progression from one accompaniment to another, characteristics that do not occur in thrombosis and embolism. Diagnosis facilitated when 2 or more similar episodes have occurred or migraine-like scintillations are present. Headache occurred in 50% of cases. Other cerebrovascular processes, coagulation disorders, and cerebral seizures must be ruled out.

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Drug-Induced Refractory Headache

Thomas N. Ward.

Headache: The Journal of Head and Face Pain Volume 48 Issue 5 Page 728-728, May 2008

Abstract

Two hundred patients who were taking daily symptomatic or immediate relief medications, often in excessive quantities, yet suffering from daily or near daily severe headaches were studied. One hundred and sixteen (58%) of them were also taking concomitant prophylactic medications and they were ineffective. Low-tyramine, low-caffeine dietary instructions and biofeedback training were given to all patients. The effect of continuing symptomatic medications, discontinuing symptomatic medications, and adding or changing prophylactic medications were studied in the various treatment groups. It is concluded that: (1) daily use of symptomatic or immediate relief medications results in chronic daily headache; (2) discontinuing daily symptomatic medication itself results in improvement of headache; (3) concomitant use of symptomatic medications nullifies the effect of prophylactic medications; (4) discontinuing daily symptomatic medications enhances the beneficial effect of prophylactic medications.

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A Pivotal Moment in 50 Years of Headache History: The First American Migraine Study

Stewart J. Tepper.

Headache: The Journal of Head and Face Pain Volume 48 Issue 5 Page 730-731, May 2008

Abstract

Objective.—To describe the magnitude and distribution of the public health problem posed by migraine in the United States by examining migraine prevalence, attack frequency, and attack-related disability by gender, age, race, household income, geographic region, and urban vs rural residence.

Design.—In 1989, a self-administered questionnaire was sent to a sample of 15,000 households. A designated member of each household initially responded to the questionnaire. Each household member with severe headache was asked to respond to detailed questions about symptoms, frequency, and severity of headaches.

Setting.—A sample of households selected from a panel to be representative of the US population in terms of age, gender, household size, and geographic area.

Participants.—After a single mailing, 20,468 subjects (63.4% response rate) between 12 and 80 Years of age responded to the survey. Respondents and nonrespondents did not differ by gender, household income, region of the country, or urban vs rural status. Whites and the elderly were more likely to respond. Migraine headache cases were identified on the basis of reported symptoms using established diagnostic criteria.

Results.—In total, 17.6% of females and 5.7% of males were found to have 1 or more migraine headaches per year. The prevalence of migraine varied considerably by age and was highest in both men and women between the ages of 35 to 45 years. Migraine prevalence was strongly associated with household income; prevalence in the lowest-income group (less than \$10,000) was more than 60% higher than in the 2 highest-income groups (greater than or equal to \$30,000). The proportion of migraine sufferers who experienced moderate to severe disability was not related to gender, age, income, urban vs rural residence, or region of the country. In contrast, the frequency of headaches was lower in higher-income groups. Attack frequency was inversely related to disability.

Conclusions.—A projection to the US population suggests that 8.7 million females and 2.6 million males suffer from migraine headache with moderate to severe disability. Of these, 3.4 million females and 1.1 million males experience 1 or more attacks per month. Females between ages 30 and 49 years from lower-income households are at especially high risk of having migraines and are more likely than other groups to use emergency care services for their acute condition.

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Headache and Mitochondrial Disorders

Noah Rosen.

Headache: The Journal of Head and Face Pain Volume 48 Issue 5 Page 733-734, May 2008

Abstract

We report on 2 patients who have a mitochondrial myopathy, encephalopathy, lactic acidosis, and recurrent cerebral insults that resemble strokes (MELAS). These 2, and 9 other, reported patients share the following features: ragged red fibers evident on muscle biopsy, normal early development, short stature, seizures, and hemiparesis, hemianopia, or cortical blindness. Lactic acidemia is a common finding. We believe that MELAS represents a distinctive syndrome and that it can be differentiated from 2 other clinical disorders that also are associated with mitochondrial myopathy and cerebral disease: Kearns–Sayre syndrome and the myoclonus epilepsy ragged red fiber syndrome. Existing information suggests that MELAS is transmitted by maternal inheritance. The ragged red fibers suggest an abnormality of the electron transport system, but the precise biochemical disorders in these 3 clinical syndromes remain to be elucidated.

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Change Mechanisms in EMG Biofeedback Training: Cognitive Changes Underlying Improvements in Tension Headache

Jeanetta C. Rains.

Headache: The Journal of Head and Face Pain Volume 48 Issue 5 Page 735-736, May 2008

Abstract

Forty-three college students suffering from recurrent tension headache were randomly assigned to 1 of 4 electromyographic (EMG) biofeedback training conditions. Although all subjects were led to believe they were learning to decrease frontal EMG activity, actual feedback was contingent on decreased EMG activity for half of the subjects and increased EMG activity for the other half. Within these 2 groups, subjects also viewed bogus video displays designed to convince them they were achieving large (high success) or small (moderate success) reductions in EMG activity. Regardless of actual changes in EMG activity, subjects receiving high-success feedback showed substantially greater improvement in headache activity (53%) than subjects receiving moderate success feedback (26%). Performance feedback was also related to changes in locus of control and self-efficacy. Changes in these 2 cognitive variables during biofeedback training were also correlated with reductions in headache activity following treatment, whereas changes in EMG activity exhibited during training were uncorrelated with outcome. These results suggest that the effectiveness of EMG biofeedback training with tension headache may be mediated by cognitive changes induced by performance feedback and not primarily by reductions in EMG activity.

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The role of cervical dysfunction in migraine: a systematic review

BA Robertson & ME Morris
Cephalalgia Volume 28 Issue 5 Page 474-483, May 2008

Abstract

This systematic review evaluates the strength of the evidence for the role of cervical musculoskeletal dysfunction in migraine. In this review, cervical musculoskeletal dysfunction will refer to the abnormal sensory afferentation from cervical region structures contained within the receptive field of the trigeminocervical nucleus. Electronic database searches using MEDLINE, PubMed and CINAHL were performed, and 17 studies investigating cervical musculoskeletal dysfunction in people with migraine were selected for review. The methodological quality of the included studies was assessed by two independent reviewers using a customized checklist. The review found that intersubject differences were inadequately reported and controlled, which resulted in grouping of participants with varying pathologies and symptoms. A diverse range of assessment procedures was used by the reviewed studies, which made comparison of their findings difficult. The assessment procedures were mainly used to quantify the degree of cervical musculoskeletal dysfunction, rather than to identify a cause and effect relationship between cervical structure and migrainous pain. Although animal study evidence proposes a role for cervical musculoskeletal dysfunction in migraine, this systematic review of the literature found that there is currently no convincing evidence to confirm this phenomenon in humans.

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Guidelines for controlled trials of prophylactic treatment of chronic migraine in adults

S Silberstein, P Tfelt-Hansen , DW Dodick, V Limmroth (, RB Lipton, J Pascual & SJ Wang.
Cephalalgia Volume 28 Issue 5 Page 484-495, May 2008

Abstract

In 1991 the Clinical Trials Subcommittee of the International Headache Society (IHS) developed and published its first edition of the Guidelines on controlled trials of drugs in episodic migraine because only quality trials can form the basis for international collaboration on drug therapy, and these Guidelines would 'improve the quality of controlled clinical trials in migraine'. With the current trend for large multinational trials, there is a need for increased awareness of methodological issues in clinical trials of drugs and other treatments for chronic migraine. These Guidelines are intended to assist in the design of well-controlled clinical trials of chronic migraine in adults, and do not apply to studies in children or adolescents.

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Familial hemiplegic migraine type 1 shows no hypersensitivity to nitric oxide

JM Hansen, LL Thomsen, J Olesen & M Ashina
Cephalalgia Volume 28 Issue 5 Page 496-505, May 2008

Abstract

Familial hemiplegic migraine type 1 (FHM-1) is a dominantly inherited subtype of migraine with aura and transient hemiplegia associated with mutations in the CACNA1A gene. FHM-1 shares many phenotypical similarities with common types of migraine, indicating common neurobiological pathways. Experimental studies have established that activation of the nitric oxide–cyclic guanosine monophosphate (NO–cGMP) pathway plays a crucial role in migraine pathophysiology. Therefore, we tested the hypothesis that CACNA1A mutations in patients with FHM-1 are associated with hypersensitivity to NO–cGMP pathway. We included eight FHM-1 patients with R583Q and C1369Y mutations and nine healthy controls, who received intravenous infusions of 0.5 $\mu\text{g kg}^{-1} \text{min}^{-1}$ glyceryl trinitrate (GTN) over 20 min. We recorded: headache intensity on a verbal rating scale; mean flow velocity in the middle cerebral artery (VmeanMCA) by transcranial Doppler; diameter of the superficial temporal artery (STA) by Dermascan. One patient reported migraine without aura 5 h after start of the GTN infusion. No aura was reported. The AUCheadache in the immediate phase was more pronounced in patients than in controls ($P = 0.01$). In the 14 h following GTN infusion, there was no difference in the AUCheadache between patients and controls ($P = 0.17$). We found no difference in the AUCVmeanMCA ($P = 0.12$) or AUCSTA ($P = 0.71$) between FHM-1 patients and controls. None of the control persons reported migraine-like headache. FHM-1 patients do not show hypersensitivity of the NO–cGMP pathway, as characteristically seen in migraine patients with and without aura. This indicates that the pathophysiological pathways underlying migraine headache in FHM-1 may be different from the common types of migraine.

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Cerebrospinal fluid and serum neuron-specific enolase in acute benign headache

M Casmiro, E Scarpa, P Cortelli & L Vignatelli
Cephalalgia Volume 28 Issue 5 Page 506-509, May 2008

Abstract

We determined the cerebrospinal fluid (CSF) and serum neuron-specific enolase (NSE) concentrations in 19 patients with acute benign headache. All patients had normal neurological examination, CSF and head computed tomography scan. The final diagnoses were: primary thunderclap headache (n = 7), primary exertional headache (n = 3), primary cough headache (n = 1), migraine without aura (n = 4), headache unspecified (n = 2), probable infrequent episodic tension-type headache (n = 1), headache attributed to hypertensive crisis without hypertensive encephalopathy (n = 1). A group of 108 healthy subjects served as controls. CSF NSE concentration was 14.16 ng/ml [95% confidence interval (CI) 11.86, 16.47] in the headache sample (controls 17.19 ng/ml, 95% CI 16.23, 18.15). Serum NSE concentration was 7.50 ng/ml (95% CI 5.20, 9.80) in the headache sample (controls 8.45 ng/ml, 95% CI 7.67, 9.23). CSF/serum ratio was 2.81 (95% CI 2.21, 3.40) in the headache sample (controls 2.23, 95% CI 2.03, 2.42). Acute benign headache is not associated with neuronal damage as estimated by means of CSF and serum NSE concentration.

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Validation of the revised Patient Perception of Migraine Questionnaire (PPMQ-R): measuring satisfaction with acute migraine treatment in clinical trials

M Kimel, R Hsieh, J McCormack, SP Burch & DA Revicki
Cephalalgia Volume 28 Issue 5 Page 510-523, May 2008

Abstract

This study was aimed to evaluate in clinical trial settings the psychometric properties of the revised Patient Perception of Migraine Questionnaire (PPMQ-R), a satisfaction measure for acute migraine treatment. The PPMQ-R was administered 24 h post dosing in 1304 migraineurs randomized to two identical Phase 3, single-attack trials. Reliability, concurrent and construct validity and known-groups validity were evaluated using Cronbach's α , Pearson correlations and analysis of variance, respectively. PPMQ-R scale and Total scores (Efficacy, Functionality and Ease of use) showed very good internal consistency reliability (α 0.84–0.99). Efficacy, Functionality and Total PPMQ-R scores showed large, inverse relationships with migraine pain severity, number of migraine symptoms and work ability ($r = -0.62$ to -0.75 ; all $P < 0.0001$). All scales discriminated among migraine pain severity levels (all $P < 0.001$). The PPMQ-R has sufficient evidence of validity and reliability for measuring patient satisfaction, an important benchmark of quality and effective care.

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Bath-related thunderclap headache: a study of 21 consecutive patients

S-J Wang, J-L Fuh, Z-A Wu, S-P Chen & J-F Lirng
Cephalalgia Volume 28 Issue 5 Page 524-530, May 2008

Abstract

We consecutively recruited 21 patients (all women, mean 54 ± 8 years) with bath-related thunderclap headache (BRTH). Thirteen of them were in menopause, two had just ceased hormonal therapy, and one was at 3 months postpartum. Bathing was the initial trigger for thunderclap headaches in nine patients (43%). Many patients ($n = 15$, 71%) had other non-bath-related attacks. Most patients ($n = 18$, 86%) reported that the headache occurred immediately when water was sprayed over their body, with warm water (52%) as the most common. During the disease course [mean 14 days (6–34)], the mean number of BRTH was 5.1 ± 3.6 attacks. Nineteen patients (90%) changed bathing habits to prevent attacks. Thirteen patients (62%) had magnetic resonance angiography vasoconstrictions, and two of them (15%) developed reversible posterior encephalopathy. None of the patients without vasoconstrictions had this complication. Nimodipine was effective in stopping further attacks in 84% (16/19) treated patients. No relapse was reported at a mean follow-up of 30 months. BRTH occurred exclusively in women and predominantly in middle age. Deficiency or fluctuation of female sex hormones may play a role. About 60% patients showed cerebral vasospasms, fulfilling the diagnosis of reversible cerebral vasoconstriction syndrome and indicating a risk of posterior encephalopathy.

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Patent foramen ovale and migraine: a quantitative systematic review

TJ Schwedt, BM Demaerschalk & DW Dodick
Cephalalgia Volume 28 Issue 5 Page 531-540, May 2008

Abstract

Initial studies indicate an increased prevalence of patent foramen ovale (PFO) in migraineurs with aura, and an increased prevalence of migraine and migraine with aura in persons with PFO. Retrospective analyses of PFO closure suggest clinically significant improvements in migraine patterns. The aim of this study was to examine the prevalence of migraine in patients with PFO, the prevalence of PFO in migraineurs, and the effect of PFO closure on migraine. We conducted a quantitative systematic review of articles on migraine and PFO that met inclusion criteria, then reviewed, appraised, and subjected them to data extraction. Of 134 articles identified, 18 met a priori selection criteria. The estimated strength of association between PFO and migraine, reflected by summary odds ratios (ORs), was 5.13 [95% confidence interval (CI) 4.67, 5.59], and between PFO and migraine with aura the OR was 3.21 (95% CI 2.38, 4.17). The grade of evidence was low. The association between migraine and PFO was OR 2.54 (95% CI 2.01, 3.08). The grade of evidence was low to moderate. Six studies of PFO closure suggested improvement in migraine, but had a very low grade of evidence. The low-to-moderate grade of evidence from observational studies supports an apparent association between PFO and migraine. Although PFO closure seemed to affect migraine patterns favourably, the very low grade of available evidence to support this association precludes definitive conclusions.

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Migraine and left-handedness are not associated. A new case-control study and meta-analysis

K Biehl, A Frese, M Marziniak, I-W Husstedt & S Evers
Cephalalgia Volume 28 Issue 5 Page 553-557, May 2008

Abstract

To investigate the possible association between migraine and left-handedness, we enrolled 100 patients with a diagnosis of migraine according to the International Headache Society diagnostic criteria and 100 age- and sex-matched control subjects into a case-control study. Handedness was determined by the Edinburgh Handedness Inventory. There was no significant difference in the frequency or grade of left-handedness between the two groups. Additionally, we pooled our data with those from five similar studies, which did not alter the result. Thus, neither our study nor the meta-analysis support Geschwind and Behan's hypothesis of an association between migraine and left-handedness.

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Comparison of CGRP and NO responses in the human peripheral microcirculation of migraine and control subjects

M-L Edvinsson & L Edvinsson
Cephalalgia Volume 28 Issue 5 Page 563-566, May 2008

Abstract

Calcitonin gene-related peptide (CGRP) and nitric oxide (NO) are two molecules shown to have a role in migraine pathophysiology. Our objective was to test the hypothesis that migraine subjects are particularly sensitive to these signal molecules. The cutaneous microvascular responses to endothelial and non-endothelial dependent dilators were tested using laser Doppler flowmetry in combination with iontophoresis. The blood flow responses to iontophoretic administration of the endothelium-dependent vasodilator acetylcholine (ACh), or to the endothelium-independent dilators sodium nitroprusside (SNP) and CGRP, and to local warming (44 °C) were compared in this controlled trial. The design was that of two arms: patients diagnosed with migraine without aura (n = 9) for >10 years were compared with nine healthy subjects matched for age and gender (seven female and two male, age range 30–60 years). Iontophoretic administration resulted in local vasodilation. ACh induced a relaxation of 1225 ± 245% (relative to baseline) in controls and 1468 ± 368% (P > 0.05) in migraine. The responses to SNP were 873 ± 193% in controls and 1080 ± 102% (P > 0.05) in migraine subjects. The responses to CGRP were 565 ± 89% in controls and 746 ± 675% (P > 0.05) in migraine patients. The responses to local heating which induced maximum dilation did not differ between the groups (1976 ± 314% for controls and 1432 ± 226% in migraine; P > 0.05). We conclude that there is no change in the microvascular responsiveness of the subcutaneous microvasculature in migraine.

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NEUROLOGÍA

Importancia de la migraña crónica en una consulta general de neurología

M. Gracia-Naya, R. Alarcia-Alejos, P.J. Modrego-Pardo
[REV NEUROL 2008; 46:577-581] PMID: 18465695 - Fecha de publicación: 15/05/2008

Introducción.

La migraña crónica es una cefalea primaria difícil de tratar que produce gran afectación de la calidad de vida del paciente. La clasificación internacional de cefaleas modificó los criterios de migraña crónica recientemente, por lo que existen pocos trabajos que analicen grupos con estos nuevos criterios. Objetivo. Analizar un grupo de pacientes con migraña crónica remitidos a una consulta de neurología general. Pacientes y métodos. Se seleccionaron los primeros 100 pacientes con migraña. Se establecieron y analizaron subgrupos de pacientes con migraña episódica, crónica o crónica con probable cefalea por abuso de fármacos según la clasificación de cefaleas de la Sociedad Internacional de Cefaleas (IHS) y su revisión del año 2006. Resultados. Del total de 738 nuevos pacientes, 100 (13,5%) sufrieron migrañas. De estos 100 nuevos pacientes con migraña, 42 (el 5,6% de la serie total) cumplieron los criterios de migraña crónica, y 15 pacientes con migraña crónica, los criterios de probable cefalea por abuso de fármacos. Antes de acudir a la consulta de neurología, sólo al 41% se le había diagnosticado migraña, un 38% no había recibido información sobre esta entidad, sólo el 17% tomaba triptanes como tratamiento sintomático, y un 23% había seguido tratamiento preventivo. Conclusiones. Destacamos la importancia de la migraña episódica y crónica en una consulta general de neurología, aplicando los criterios recientes de la IHS. Los pacientes con migraña crónica enviados a la consulta de neurología siguen, en un alto porcentaje, sin haber sido diagnosticados, ni informados, ni tratados correctamente, con un alto grado de automedicación y abuso frecuente de fármacos. Los tratamientos preventivos y triptanes en migrañas intensas se siguen utilizando poco en atención primaria.

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Ficha Técnica Menatriptán

El principio activo de **MENATRIPTAN** es frovatriptán.

Cada comprimido con cubierta pelicular contiene 2.5 mg de frovatriptán (DCI, como succinato).

Lactosa y otros excipientes, c.s.

1. QUÉ ES MENATRIPTAN Y PARA QUE SE UTILIZA

MENATRIPTAN se presenta en la forma farmacéutica de comprimidos con cubierta pelicular. Cada envase contiene 2 comprimidos.

MENATRIPTAN es un medicamento que pertenece a la familia de los agonistas selectivos del receptor de la 5-hidroxitriptamina (5-HT₁).

MENATRIPTAN se utiliza para el tratamiento agudo del dolor de cabeza de las crisis de migraña con o sin aura (sensación subjetiva pasajera que precede a la migraña, que varía de una persona a otra y que afecta a la audición, la visión, etc.).

MENATRIPTAN debe usarse para tratar una crisis de migraña, pero no para prevenir dicha crisis.

2. ANTES DE TOMAR MENATRIPTAN

No tome MENATRIPTAN en los siguientes casos:

- si Ud. es alérgico a frovatriptán o a cualquiera de los demás excipientes de **MENATRIPTAN**,
- si Ud. tiene una historia anterior de infarto de miocardio,
- si Ud. padece de enfermedades cardiovasculares, si tiene riesgo de enfermedades cardiovasculares (mujeres en edad postmenopáusia, varones de más de 40 años, es fumador o está siguiendo una terapia de sustitución de nicotina), o si tiene una historia anterior de enfermedades cardiovasculares tales como angina de pecho,
- si Ud. ha padecido anteriormente algún accidente cerebrovascular,
- si Ud. padece de hipertensión arterial severa o moderada, o de hipertensión arterial leve no controlada,
- si Ud. padece alguna enfermedad hepática,
- simultáneamente con otros medicamentos también usados en el tratamiento de la migraña (ergotamina y derivados de la ergotamina (incluida metisergida), sumatriptan y otros agonistas de 5-hidroxitriptamina (5-HT₁)).

Tenga especial cuidado con MENATRIPTAN:

Si usted está tomando alguno de los siguientes medicamentos:

- medicamentos inhibidores de la monoaminoxidasa (Medicamentos para la depresión)

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- preparaciones que contengan hierba de San Juan.
- inhibidores selectivos de la recaptación de serotonina (citalopram, fluoxetina, fluvoxamina, paroxetina, sertralina).
- metilergotamina
- anticonceptivos orales

Toma de MENATRIPTAN con alimentos y bebidas:

No se han descrito interacciones.

Embarazo:

Consulte a su médico o farmacéutico antes de tomar un medicamento.

No se recomienda tomar **MENATRIPTAN** durante el embarazo.

Lactancia:

Consulte a su médico o farmacéutico antes de tomar un medicamento.

No se recomienda tomar **MENATRIPTAN** durante la lactancia.

Conducción y uso de máquinas:

Este medicamento, al igual que la migraña, puede causar somnolencia. Por ello no resulta

recomendable realizar tareas que requieran concentración y habilidad mientras se está bajo este tratamiento o bajo los efectos de una crisis de migraña.

Toma de otros medicamentos:

Informe a su médico o farmacéutico si está tomando, o ha tomado recientemente cualquier otro medicamento, incluso los adquiridos sin receta.

Debe informar a su médico si está tomando o ha tomado algún medicamento, especialmente para el tratamiento de la migraña, enfermedad cardiovascular, depresión, o medicamentos para dejar de fumar.

MENATRIPTAN no se debe tomar simultáneamente con otros medicamentos usados para el tratamiento de la migraña (especialmente ergotamina, derivados de la ergotamina (incluida metisergida), sumatriptan u otros agonistas del 5-HT₁). En caso de tener que hacerlo, debe dejarse transcurrir un mínimo de 24 horas después de la toma de estos medicamentos y la administración de frovatriptán. A la inversa, se recomienda esperar 24 horas después de la administración de frovatriptán antes de administrar una medicación tipo ergotamina.

3. CÓMO TOMAR MENATRIPTAN

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Siga estas instrucciones a menos que su médico le haya dado otras indicaciones distintas.

Recuerde tomar su medicamento.

MENATRIPTAN se toma por vía oral. Los comprimidos deben tragarse enteros, con la ayuda de un poco de agua.

MENATRIPTAN debe tomarse tan pronto como sea posible después del comienzo de una crisis de migraña, pero también es efectivo si se toma algo más tarde.

MENATRIPTAN no debe tomarse nunca a modo de prevención antes del inicio de una crisis de migraña.

La toma de **MENATRIPTAN** está recomendada para personas de más de 18 años y menos de 65.

La dosis recomendada para el tratamiento de la crisis de migraña es un comprimido (2.5 mg de frovatriptán).

Si reaparece la migraña después de un alivio inicial, se puede tomar una segunda dosis (un comprimido con 2.5 mg de frovatriptán), siempre que se deje un intervalo mínimo de 2 horas entre ambas dosis.

Si el paciente no nota ningún alivio con la primera dosis de frovatriptán, no se debe tomar una segunda dosis para la misma crisis.

La dosis diaria total no debe ser superior a dos comprimidos al día (5 mg de frovatriptán al día).

Si Ud. toma **MENATRIPTAN** de forma muy frecuente (toma repetida durante muchos días seguidos) está realizando un uso incorrecto de este medicamento, y puede provocar con ello un aumento en las reacciones adversas. Además, un uso excesivo de fármacos antimigrañosos puede dar lugar a dolor de cabeza diario crónico.

Si estima que la acción de **MENATRIPTAN** es demasiado fuerte o débil, comuníquese a su médico o farmacéutico.

Si Ud. toma más MENATRIPTAN del que debiera:

Si accidentalmente Ud. ha tomado más **MENATRIPTAN** del que debiera, consulte inmediatamente a su médico o farmacéutico, o consulte al Servicio de Información Toxicológica en España, teléfono 91 562 04 20.

4. POSIBLES EFECTOS ADVERSOS

Al igual que todos los medicamentos, **MENATRIPTAN** puede tener efectos adversos.

Los efectos adversos aparecidos tras la toma de frovatriptán son transitorios, generalmente entre leves y moderados, y desaparecen espontáneamente. Algunos de los síntomas que se indican como efectos adversos pueden estar asociados a la crisis de migraña.

Las reacciones adversas observadas son:

- Vértigo, dolor de cabeza, sensación de hormigueo, entumecimiento o pinchazos en brazos y piernas.
- Náuseas, sequedad de boca, problemas digestivos.

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- Fatiga, sensación de cambio de temperatura, dolores torácicos (dolor u opresión en el tórax que puede llegar a ser intenso e irradiarse hacia la garganta) (ver abajo).

- Somnolencia
- Dolor muscular
- Enrojecimiento

A los pocos minutos de tomar el medicamento puede producirse una sensación de opresión o dolor torácico, a veces intenso, con la posibilidad de que se extienda a la garganta. Si ello ocurre, acuda rápidamente a su médico y no tome ninguna dosis adicional de este medicamento.

Si aprecia efectos adversos no mencionados en este prospecto, comuníquese a su médico o farmacéutico.

5. CONSERVACIÓN DE MENATRIPTAN

Mantenga **MENATRIPTAN** fuera del alcance y de la vista de los niños.

No conservar **MENATRIPTAN** a temperatura superior a 30°C .

Caducidad

No utilizar **MENATRIPTAN** después de la fecha de caducidad indicada en el envase.

Este prospecto ha sido aprobado en Agosto 2002

Frovatriptán fue desarrollado por Vernalis Limited

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