頭痛電子報第十二期

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【本期內容】

頭痛學組於９月２６日所舉辦「台北２００４頭痛研討會」已圓滿落幕，感謝大家的參與，本期內容亦收錄研討會摘要，提供給未能參加的會員參考。另有一項活動訊息，頭痛學組將邀請 American Headache Society 的主席，也是王署君醫師的老師，美國頭痛大師 Professor Silberstein 於 11 月 21 日星期天下午來台演講（台北）。詳細時間地點會再另行通知，敬請期待。

輔酶 Q10 能促進粒線體的功能，是人體產生能量的必需物，在美國一般當健康食品使用。有研究指出，偏頭痛可能與粒線體功能異常有關，所以輔酶 Q10 才被用作偏頭痛預防用藥的研究。本期內容首篇主要介紹輔酶 Q10 對偏頭痛預防之作用和療效，雖然輔酶 Q10 對於頭痛是否真的有所幫助，仍有待進一步的臨床研究證實，但也提供了一個治療偏頭痛的新方式。

第二篇談家長在面對孩童的頭痛時所應注意的事項，雖然本篇對象主要是針對一般家長，但其中提到小孩頭痛和成人之差異及治療方法，相信對醫師也有所助益，提供給大家參考。

輔酶 Q10（Coenzyme Q10）與偏頭痛

作者：-Todd D. Rozen MD

本文經同意轉譯自美國頭痛教育委員會頭痛通訊（Headache Newsletter of the American Council For Headache Education） **譯者：台北榮總神經內科賴冠霖醫師**

偏頭痛是個相當惱人的疾病，而預防偏頭痛不僅可以減輕頭痛發生的頻率、嚴重度和發作時間，更可以增進生活品質。而偏頭痛的預防用藥需要長時間的服用（數月至數年），因此，藥物的安全性和耐受性是相當重要的議題。截至目前為止，只有極少數的藥物對偏頭痛有預防療效。而這些藥物之中，也只有少部分沒有特殊的副作用。難怪有許多人對於用維他命、礦物質和其他日常飲食成分來作為偏頭痛的

用藥相當感興趣。舉例來說，有一項設計良好的研究指出，長期服用維他命 B2，可以有效預防偏頭痛。另外，有些研究也指出口服鎂離子或小白菊（feverfew）可以預防偏頭痛的發作（然而，有些研究卻指出沒有益處）。直到最近，輔酶 Q10 才被用作偏頭痛預防用藥的研究。

輔酶 Q10 是一種天然的成分，而且是身體糖解產生能量過程中，一個重要的必須元素。這個步驟發生在身體內數以兆計的細胞的能量工廠：*粒線體* 內。

由一些影像學的線索以及 DNA 的分析，我們認為，至少在某些病患身上，偏頭痛可能與粒線體功能異常有關。有偏頭痛的患者，不管在頭痛發作或是在發作間期，其腦內的能量代謝似乎有所缺陷。在平時，偏頭痛患者的腦內能量儲存量似乎就比較少，可能也是造成這些患者較易頭痛的原因。因此，如果偏頭痛的患者其粒線體的功能較差，若我們可以促進粒線體的生成，使細胞產生更多能量，或許可以改善偏頭痛。

輔酶 Q10 是在用來治療粒線體疾病的藥物中，最被廣泛研究的一種藥物。用在人類身上，它幾乎沒有什麼副作用。因為偏頭痛可能與粒線體功能缺失有關，而輔酶 Q10 可以增進粒線體的功能。因此，我們選用它來作為預防偏頭痛的藥物實驗。

在這項實驗中（費城的傑佛遜頭痛中心），32 名偏頭痛患者（26 名女性，6 名男性），於每日早上服用 150 毫克的輔酶 Q10。這項實驗為"開放性"（open-label）實驗，也就是每位患者皆知道自己服用輔酶 Q10，而無對照組。每位病患必須有一年以上的偏頭痛病史，且每月發作 2 到 8 次。

最後，共有 31 位患者完成這項研究。百分之六十一的患者，其每月頭痛天數有超過一半的減輕。在服用藥物前，這些病患的每月平均頭痛日數是七天；在服用藥物三個月後，病患的每月平均頭痛日數降至三天，達到統計學上的顯著差異。對每個患者而言，大部分的患者在服用藥物前，每月有高達五次的頭痛；而在服用藥物三個月後，大部分患者每月只有二到三次的頭痛（有統計學上的意義）。這些資料顯示，服用可能需要五到十二週的治療，才能達到完全的療效。另外很重要的一點是，這些服用輔酶 Q10 的患者並沒有特殊的副作用。

然而，由於該實驗並無對照組，我們必須更小心的加以判讀這項研究的結果。任何藥物都必須經過設計良好的實驗，證實其效果優於安慰劑，我們才能相信它真的有療效。和安慰劑互相比較是非常重要的，因為有一部分的患者（大約百分之三十），單靠安慰劑即有明顯的療效。

經由這些初步的研究，輔酶 Q10 可以是某些偏頭痛患者的首選用藥。然而，這個結論還須經由有安慰劑對照組的實驗加以驗證。另外，輔酶 Q10 用來預防偏頭痛的最適合劑量仍是未明。輔酶 Q10 可以用在任何年齡層，而不用擔心有顯著的副作

用。雖然輔酶 Q10 對於治療頭痛到底是否有所助益，仍需進一步的研究證實，但它提供了一個新的治療偏頭痛的方式（增加腦內的能量代謝）。

雖然十分安全，輔酶 Q10 卻相當昂貴。以上述實驗的劑量來看，一個月的費用便需要 18 到 50 美金。（若以 1:33 的匯率換算，折合台幣約 594-1650 元）由於價格的差異很大，如果你想自己買來服用的話，最好貨比三家。另外，因為輔酶 Q10 目前只是健康食品，而不是美國食品及藥物管理局正式認可的藥物，所以不同的品牌可能有不同的效度和純度。消費者實驗者（Consumer Lab） 曾報導過：在 29 種不同的品牌中，有一種品牌的濃度只有標示上的百分之十七。在該網站上（www.consumerlab.com）（英文網站），消費者可也找到一些通過該實驗室檢驗的品牌。

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孩童的頭痛：家長能做些什麼

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本文經同意轉譯自美國頭痛教育委員會頭痛通訊（Headache Newsletter of the American Council For Headache Education） **譯者：台北榮總神經內科梁仁峰醫師**

雖然大多數新診斷的偏頭痛是在青少年階段，小到二、三歲的孩子還是可能會有偏頭痛。由於這些偏頭痛的症狀跟一般成人的偏頭痛並不完全相像，小孩子的偏頭痛可能過了許多年都無法得到正確的診斷，直到典型的症狀（像噁心、嘔吐、怕光、怕吵）出現。

如何找尋頭痛的線索，對父母而言是很重要的，因為兒童跟青少年也會頭痛以至於無法正常的生活。孩子可能無法描述他們的感受，而藉由一些背景知識、小心的觀察和創造力的問題，將可以對您孩子的頭痛有些概念，並尋求正確的醫療協助。

小孩子頭痛有什麼不同？

雖然兒童確實可能得到偏頭痛，但相較於大人的偏頭痛，有些差異值得注意：

1. 小孩的偏頭痛較短暫，常僅持續一兩個小時，多數十二小時內會結束。
2. 發作頻率較低，可能一個月只有一次，甚至幾個月才發作一次。
3. 他們的頭痛可能在幾個月到幾年的發作期後消失。
4. 兩側前額疼痛比單側多，隨著年紀的增長，頭痛越來越偏向單側。
5. 孩子們可能會有週期性的嘔吐、腹痛而沒伴隨頭痛，諸如此類週期性的症狀可能

是孩童時期的偏頭痛症候群。不過最好還是先看腸胃科醫師，排除消化道的疾病。

6. 小孩子可能不會告訴您典型的偏頭痛症狀，像是怕光或是怕吵。

一起來解謎

您的孩子可能有偏頭痛嗎？醫生和家長們所面臨的挑戰主要是如何適當的評估孩子們的感受。假如您直接問類似「你對光或聲音敏感嗎？」之類的問題，孩子可能根本不知道您在說什麼。

「噁心」常是一個小孩子難以正確理解的名詞。如果您問孩子：「你噁心嗎？」他們可能會一臉迷惑的看著您。即使問得簡單一點，像是：「你有反胃的感覺嗎？」他們可能還是不懂您在說什麼。

藉由觀察孩子們的行為，能辨認出其頭痛的特性。舉例來說，觀察他們是否躲到安靜的地方，或是話突然比平常少的多；注意他們何時恢復正常的活動力；問他們想不想吃些東西，特別是他們最喜歡的東西。如果他們拒絕了，可能是正在噁心。假如臉色蒼白又不吃東西，可能是偏頭痛正在發作的線索。

看醫生的時候，能期待些什麼 ？

若您的小孩被診斷為偏頭痛，您和孩子必須知道，你們面對的是一個良性，而非危險的病。許多病例說明這是一種可以治療的疾病。一般而言，不需做太多測驗或檢查來建立偏頭痛的診斷。然而，由於每個患者狀況不同，醫師可能會安排進一步的評估。譬如您或孩子有不尋常的過去病史或異常的測驗結果，醫師可能會安排其他檢驗以排除腦瘤或其他嚴重進行性的疾病。

治療這些孩童

治療的方針主要是依據頭痛影響日常生活的程度。如果頭痛只是輕微的，如僅造成一小時的缺課，或是只有少數的相關症狀，只要讓他們適當的休息，和保證症狀一會就過去也許就足夠，必要時也可以使用輕微的止痛藥。

然而，假如是中度到嚴重的頭痛，而導致生活受到影響，譬如疼痛持續四到六個小時，痛到沒辦法上學或參加其他日常活動，醫師可能就會採取較積極的評估和治療。這時候非類固醇類止痛藥可能會派上用場。假如孩子吃了止痛藥後一到二小時還是沒有緩解，可能得考慮偏頭痛專用的藥物，像是翠普登(triptans)。這些藥物可以在頭痛發作後的二至四小時服用以緩解頭痛，最好是在一至二小時內服用。

翠普登類藥物的劑型包括錠劑、鼻噴劑和針劑。雖然有些報告顯示這些藥物對孩童和青少年頭痛的效果，但目前為止這藥物還沒被美國 FDA（食品藥品管理局）核准用於 18 歲以下的青少年或兒童。

如果頭痛一週超過兩次，那麼家長們應該考慮預防性的治療，也就是天天吃藥以預防偏頭痛的發作。關於這方面用藥最好和熟悉的醫師詳細討論，因為這個年齡

層的預防用藥研究資料還不是很充分。

重要的是，記住別讓孩子服用止痛藥一週超過兩天。假如您的小孩覺得需要更多的劑量，可能代表他的頭痛並不單純。如果是成人會開始慢性每日頭痛，這些族群的人也有這個可能。

非藥物的策略

一些非藥物的方法是有相當幫助的，也應該使用在每個頭痛的兒童上，試試看下面幾招：

1. 建立規律的生活作息，確定每天晚上固定時間睡覺。
2. 減少咖啡因的攝取量為每天一次或是更少，這些咖啡因可能來自軟性飲料，例如冰紅茶。
3. 確定喝了足夠的水，特別是夏天。
4. 確定有規律的運動。
5. 幫助保持適當的體重。
6. 若生活形態改變沒有顯著的效果，可考慮其他系統化的治療方法，像是生物回饋和壓力處理技巧等等。

到目前為止，可以試的最有效的非藥物性方法是教育。讓您的孩子們知道他們遇到了顯著的問題，而醫師們知道這些問題的原因，而且有辦法控制得宜。此外，由於這個年齡層的相關研究結果越來越多，未來的希望也越來越多。

尋找正確的醫師

當醫師治療孩子的頭痛遇到困難時，可以考慮尋求第二意見更換治療方式看看。當大多數的藥物可以讓頭痛在一、二個小時內停止，或是大大的降低，實在沒有道理讓這些可憐的孩子痛到六、七個小時。一般而言，如果給予正確的治療，小孩跟青少年的反應一般都不錯。即使目前沒有有效的療法，在可預見的未來還是會有更多的選擇。

台北２００４頭痛研討會摘要

Cluster headache

台北榮總神經內科 傅中玲醫師

Cluster headaches are among the most painful of all headaches. The signature is a pattern of periodic cycles of headache attacks, which may be one of the following:

Episodic (occurring regularly for weeks to months, followed by long pain-free periods), or Chronic (occurring without sustained breaks).

Cluster symptoms tend to occur during spring, autumn or both and they most often occur at night. Over half of cluster headache patients experience warning symptoms, long before an attack. They are called *prodromal*, or *premonitory*, symptoms. They include the following: strange tingling sensations around the eye, nose, or neck, nasal congestion or runny nose, excessive tearing, feeling restless or depressed. When the actual attack occurs, symptoms typically escalate rapidly (within about 15 minutes) to intense levels. The pain is typically described as severe, stabbing or boring. It is virtually always on one side, although it may occur on the opposite side in other attacks or even within the same attack. Pain does not worsen with movement (as it often does with migraine headaches). Other symptoms include the following: excessive tearing, feelings of intense restlessness and agitation, facial sweating, and nausea.

Headache attacks tend to occur with great regularity at the same time of day. About 75% occur between 9 at night and 10 in the morning. Peaks have also been reported between 1 PM and 3 PM. A single cluster attack is usually brief but extremely painful, lasting about one to three hours. During an active cycle, sufferers can experience these attacks as infrequently as one every other day to several attacks a day. Cycles of such daily or near daily attacks typically occur over the course of a week to a year--most often in spring and autumn. Usually a patient has one or two cycles per year that each last one to three months.

Evidence now strongly suggests that abnormalities in the *hypothalamus*, a complex structure located deep in the brain, may play a major role in cluster headaches. Advanced imaging techniques have revealed that a specific area in the hypothalamus is asymmetrical in these patients and is activated during a cluster headache attack. The most important nervous cluster is the *suprachiasmatic nuclei (SCN),* which appears to help coordinate the body's activities (sleep/wake) with the environment (dark/light). Some studies support the idea that some failure in this biologic pacemaker may impair the pain control system and cause these terrible attacks.

Lifestyle factors, including smoking, alcohol abuse, and stress (in particular stressful work situations), appear to play a very strong role in this headache. Alcohol, in fact, can trigger an attack. A particularly high association exists between smoking and cluster headaches. Quitting smoking, however, is not associated with any fewer or less severe attacks.

The most effective treatments for a cluster attack are the following: oxygen inhalation and triptan drugs (injections of sumatriptan). Relief can occur in five to 10 minutes. Prevention of attacks during a cluster cycle is extremely important. The following are the most commonly used preventive agents: calcium-channel blockers (most often used for preventing cluster headaches), corticosteroids, lithium and antiepileptic medications.

Surgical intervention may be considered for patients with chronic cluster headaches that do not respond to treatments at all or when they have not gone into remission for at least a year. To date, surgery has limited success and can have distressing side effects. Deep brain electrical stimulation is showing promise, however.

Pathophysiology of tension-type headache

新光醫院神經科 陳威宏醫師

Tension type headache (TTH) is the most frequent primary headache, with a lifetime prevalence of 74% in general population. Despite its tremendous socioeconomic impact in modern society little is known about the underlying pathophysiology and treatment. For decades, it has been debated if the pain in TTH originates from myofascial tissues or from central mechanisms in the brain. The increased tenderness in TTH represents the activation of peripheral nociceptors. Decreased pain, thermal and electrical thresholds have been reported in chronic TTH patients, which probably represents a central misinterpretation of the incoming signals. Nitric oxide (NO) might play a role in the pathophysiology of chronic TTH. It has been demonstrated that NO synthetase inhibitor reduces headache and muscle hardness, whereas the NO donor glycerol trinitrate causes headache in patients with chronic TTH. Myofascial factors and peripheral sensitization of nociceptors play an important role in the episodic form, and central sensitization has been demonstrated in the chronic form. As chronic tension-type headache usually evolves from the episodic form, prevention and reversal of this central sensitization may be an important target for future pathophysiological studies and drug development.

Nonpharmacologic Treatments for Primary Chronic Headache

高雄長庚神經內科 李連輝醫師

As the mechanisms of chronic headache are so complicated with multiple factors. Edogeneous and exogeneous factors, environmental and genetic predisportions. So the treatment of chronic headache need to be multyiplicity: acute and prophylactic with pharmacologic and nonpharmacologic treatments.

I. Classification and causes of primary chronic hedacahe

II. Pathogenesis of chronic headache(multiple factors)

III.Treatments of Chronic headache: 1)Acute treatment 2) Prophylactics treatments 3) Pharmacologic treatments 4) Nonpharmacologic treatments

IV. Nonpharmacologic treamnents:

1. Migraine:

1) Common provacational triggers for migraine

2) Evidence- based guideline of nonpharmacologic treatments in migraine

2. Tension headache:

1) Conventional nonpharmacologic treatments: Psychologic, physical treatments

2) Unconventional nonpharmacologic treatments: Local therapy, manipulation of the neck, acupuncture, hypnosis, others

V. Demonstration of nonpharmacologic treatments(digital)

1) Biofeedback 2) Relaxation 3) Walking 4) Jogging 5) Taichi chikon(with tree)

VI. Strategy of chronic headache treatments in future

1) Pharmacoliogic and nonpharnacologic treatments

2) Team work for treat chronic headache patients

3) Research

Migrainous infarction

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王署君醫師

The diagnosis of migrainous infarction is based on the association of the abrupt onset of a neurologic deficit during a migraine attack with evidence of cerebral infarction on neuroimaging. Other causes of stroke must be excluded. Strict criteria for the diagnosis of migrainous infarction must be applied because migraine is common and patients with migraine may suffer from other causes of stroke. The diagnosis of migrainous infarction should be made only when patients with an established history of migraine suffer a cerebral infarction during a typical migraine attack (Rothrock et al 1988).

The diagnostic criteria of migrainous infarction were just revised by the International Classification of Headache Disorders, second edition, as follows (coded as 1.5.4): (1) the present attack in a patient with 1.2 migraine with aura is typical of

previous attacks except that 1 or more aura symptoms persists for more than 60 minutes; (2) neuroimaging demonstrates ischemic infarction in a relevant area; (3) the headache is not attributable to another disorder (Headache Classification Subcommittee of the International Headache Society, 2004).

The exact cause of migrainous infarction is still not certain. Based on studies using cerebral angiography during attacks, the most important underlying mechanism for the stroke is believed to be carotid or vertebral arterial spasm resulting in a critical degree of cerebral hypoperfusion (Featherstone 1986; Rothrock et al 1988; Sanin and Mathew 1993). However, this may not always be true, so other factors should be considered. The incidence of migrainous infarction is rare, according to the strict diagnosis proposed by International Headache Society. Henrich and colleagues reported that the incidence rate of first migrainous infarction was 3.36 per 100,000 per year (Henrich et al 1986). However, in the absence of other stroke risk factors, this estimate was reduced to 1.44 per 100,000 people per year. In a recent study of the Barcelona Stroke Registry, Arboix and colleagues reported that the group of patients with migrainous infarction accounted for only 0.6% of all first-ever acute strokes, 0.8% of ischemic strokes, 12.8% of ischemic strokes of unusual etiology, and 13.7% of ischemic strokes in young adults 45 years of age or younger (Arboix et al 2003). Because distinct diagnostic criteria are lacking, migrainous infarction should be considered a diagnosis by exclusion.

The diagnosis demands a well established history of migraine and exclusion of other conditions that cause stroke. Computed tomography, MRI (including diffusion-weighted image, perfusion-weighted image, magnetic resonance spectroscopy, and MRA), cerebral arteriography, transcranial Doppler evaluations, and transesophageal echocardiography evaluations should be performed when possible. Lumbar puncture and blood studies to help exclude vasculitis should also be carried out. Prophylactic treatment of migrainous infarction in patients with prolonged aura includes platelet antiaggregants or calcium channel blockers. Aspirin administration should be considered as a prophylactic treatment against migrainous infarction. Patients who have anticardiolipin syndrome may need anticoagulants. Calcium channel blockers are recommended for patients at risk for migrainous infarction. Nimodipine reduces cerebral vasoconstriction, and several studies have demonstrated the potential effectiveness of calcium channel blockers in the prophylactic treatment of migraine headaches (Meyer 1985).Ergotamine, triptans, and serotonergic medications may initiate or worsen intracranial vasospasm and dysautoregulation in patients with migrainous infarction. These drugs should be withheld in patients with prolonged aura. Peripheral beta-blockers, such as propranolol, should also be withheld, since they may worsen intracranial vasoconstriction.

In general, the long-term prognosis in patients with migrainous infarction is good. Milhaud and colleagues, in a prospective stroke registry, found that the outcome at 1

month was favorable in more than 70% of migraineurs with ischemic stroke (Milhaud et al 2001). Arboix and colleagues reported the mean length of hospital stay of 9 consecutive patients with migrainous infarction was 9.75±6.2 days. No patients died during hospital stay, and 67% were symptom-free at discharge (Arboix et al 2003).

CASES DEMONSTRATON

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The classification and diagnostic criteria of *Migraine with aura*, *Persistent aura without infarction* and *Migrainous infarction* have some changes in ICHD-II. There are 6 subforms of *Migraine with aura*. The identification of the symptom of motor weakness is the first step in their differentiation. The past history of *Migraine with aura* is essential in diagnosis of *Persistent aura without infarction* and *Migrainous infarction*. Similar aura, but lasting more than one week, is mandatory in *Persistent aura without infarction*. On the other hand, when the similar aura symptom persisted more than 60 minutes with a relevant positive finding in neuroimaging, then we can therefore consider the diagnosis of *Migrainous infarction.* Clinical cases were demonstrated for the audience to practice these criteria.