NON-CERVICOGENIC HEADACHE

A CASE STUDY

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Abstract: A 40 year old overweight female presented with an intractable headache which had been present over a two week period. The patient had a history of cervicogenic headache which had responded to manipulation. Examination revealed elevated blood pressure and papilloedema. The differential diagnosis of papilloedema is discussed in respect to this case.

Index Terms: Chiropractic, cephalgia, headache, pain, papilloedema, hypertension malignant.

INTRODUCTION

Epidemiological studies suggest that between 66% (1) and 90% (2,3) of various populations suffer from headache. Other studies in the USA have shown that the most common reason for a visit to a general practitioner is headache. (4) Further, as has been stated by Vernon, 70% of all headaches have some cervical origin (5).

The statistics on Chiropractic practice would suggest that after low back pain the next most frequent cause of chiropractic consultation is neck pain and headache (6,7,8). The cervicogenic component in headache is found so commonly, in the experience of the authors, that it could be possible to visualise a degree of complacency about the cause of headache creeping into practice. It only takes one case to illustrate that such a scenario could have disastrous consequences.

PRESENTING COMPLAINT

A forty two year old overweight female presented to the clinic with unremitting headaches of some two weeks duration.

SIGNS AND SYMPTOMS

The patient reported that the headache was pounding in nature, and marginally worse towards the end of the day. The headache was felt over the whole head, but was more intense behind the eyes. No visual signs or symptoms were present. The pain was partially responsive to analgesics, both paracetamol and aspirin, but never completely faded. During the previous two weeks there had not been a waking moment when the headache was absent. The patient stated that she had felt that her blood pressure might be raised because there had been some additional stress at work recently. Her blood pressure had been taken at the time of initial examination, in 1989, and was within normal range (136/86). Due to a history of elevated blood pressure her local medical practitioner regularly monitored her blood pressure. It had been checked approximately 1 month prior to this consultation and she had been told it was normal. This was recorded on her chiropractic file.

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PREVIOUS HISTORY

The patient had a history of stress related headache over many years. Normally her headaches were of a gnawing nature, and were worse in the evening. They tended to originate in the suboccipital area and spread to one side of the head, usually the left. They had a tendency to last up to several days. There were no visual symptoms. These headaches would respond to analgesics, but if the headache was particularly bad, the relief would only be temporary. The headaches responded well to chiropractic care, which would give almost instant relief. Because of a programme of continuing care, the headaches prior to this treatment were rare and tended to appear only after trauma or prolonged stress.

FAMILY HISTORY

The patient's younger brother has a history of idiopathic deep vein thrombosis. Some five years prior to this incident he reported that he had a "metal mesh filter" inserted in his ascending aorta. This was done after he had experienced four major thrombotic episodes. This implant, he stated, was to prevent any clots from continuing to the heart. Aggressive anticoagulant therapy appears to have prevented any further thrombus formation since that time. No other family history was available at the time.

PHYSICAL EXAM

A complete orthopaedic and neurological examination was performed. The exam findings included; reduced right cervical rotation; reduced right lateral flexion;

weak right triceps (rated 3/ 5 for strength); weak right pectoralis major sternal (3/5); and right sided erector spinae tension. Standard cranial nerves screening tests were within normal limits. Maignes test was negative bilaterally, and all cervical reflexes were normal. Blood pressure was 190/120, left arm seated.

An opthalmoscopic examination was performed and, although it was difficult to visualise the right retina, the optic disc was obviously hyperaemic and had blurred edges. The blood vessels appeared engorged and tortuous. They were difficult to see through the edge of the optic disc. No haemorrhages were seen. The left retina proved impossible to adequately visualise.

DIFFERENTIAL DIAGNOSIS

The opthalmoscopic signs are consistent with a diagnosis of papilloedema (9)(10). The differential diagnosis of papilloedema is well given by Patten (11) and is summarised below:

- Raised intracranial pressure due to (types of space occupying) mass/lesion.
- Increased intracranial pressure due to circulatory block (being aquaductal stenosis, intraventricular tumour or 4th ventricle overflow block).
- Cerebral oedema. (being post traumatic, post anoxia, benign intracranial hypertension, poisoning by lead or Vitamin A. or steroid withdrawal).
- Raised C.S.F. protein or altered blood products (post sub arachnoid haemorrhage, meningitis, Guilaine -Barre, hypertrophic polyneuritis, or spinal cord tumours).
- Malignant hypertension.
- Metabolic disorders (hypercapnia, hypocalcaemia, malignant thyrotoxic exophthalmos).
- Disorders of circulation (types of thrombosis, polycythaemia, diabetes, hyperlipidaemia, Multiple myelomatosis, blockages of the retinal vessels, superior vena cava or vasculitis/temporal arteritis).

WORKING DIAGNOSIS

Due to the history of increased blood pressure, the length of time that the headache had been present, the family history, and the observed papillioedema, a diagnosis of thrombus / space occupying lesion was assumed, and as this was not amenable to chiropractic care, and was regarded as a potentially life threatening condition, referral was the only course of action possible.

TREATMENT

The patient was immediately referred to her local medical practitioner who performed a full blood exam. Upon seeing the negative results of these tests, the G.P. arranged an urgent consultation with a local opthalmologist. The ophthalmologist conducted another retinal exam. A neurological consultation followed that afternoon. This led to admission to a major teaching hospital and CT scan with angiogram that evening.

Apart from a finding of increased spinal CSF pressure, nothing of further significance was discovered. The patient was presumed to have some form of benign intracranial pressure presumably due to aquaductal blockage. The investigations are still proceeding.

MECHANISMS LEADING TO THE SYMPTOM COMPLEX

According to Demeyer (12) and Anderson (13), papilloedema is a visual symptom of a compression of the central vein where it transverses the subarachnoid space in the optic nerve sheath. The compression is a direct consequence of any major increase in the intracranial pressure. This subsequently increases the pressure around the optic nerve, causing the ophthalmic vein to collapse and obstruct the retinal veins. These distend causing oedematous fluid to leak into the nerve fibres as they cross the disc to enter the optic nerve, causing blurring of the optic papilla (optic disc) when viewed through an ophthalmoscope.

Each of the differential diagnoses can lead to an increase in the intracranial pressure. The working diagnosis of space occupying lesion/thrombus is an obvious cause of this increased pressure. This type of lesion increases the pressure in the limited space of the cranium by increasing the space taken by the tissues, either by blocking the fluid drainage away from the brain, or by adding to the mass of tissue present. This compresses the brain and causes the pressure headache and papilloedema. Other symptoms often associated with an increase in intracranial pressure include vomiting, blot and dot haemorrhages in the eye, visual disturbances, (especially an increase in the size of the blind spot in the visual field), raised systolic blood pressure, slow pulse, and possibly a diminished consciousness state (13).

While the patient had increased blood pressure, and was in the peak incidence age for malignant hypertension, (40 - 50 in females) (13), there was a complete lack of visual signs and symptoms, and there was no sign of renal involvement. While a diagnosis of an early case of aggressive malignant hypertension can not be discounted, this disease usually has a slower course, with an onset of up to 6 months (13). The regular blood pressure monitoring, which showed

no abnormal readings in the months prior to the episode, suggested that a more aggressive process was acting in this case.

Some of the causes of cerebral oedema could be removed due to the lack of trauma noted in the history. There was no sign of behavioural changes or strange neurological symptoms, which might have indicated lead poisoning, or one of the polyneuritis diseases. The two week onset removes sudden large haemorrhage as a cause, although it does not remove the possibility of a slow steady leak from an intracranial bleed - for example from a case of idiopathic thrombocytic purpura. However, no other signs of a bleeding disorder were noted.

A possible mechanical aetiology for a headache lasting two weeks could be a cervicogenic headache with or without a migrainous component. This aetiology is postulated to have a component of raised intracranial pressure (11) and might mimic the early stages of space occupying lesion. A cervicogenic origin for this headache could be from a convergence within the trigeminocervical nucleus which Bogduk states leads to a "pain perceived as arising in the head, but whose actual source lies not in the head but in the cervical spine." (14). Thus mechanical problems in the upper cervical spine might cause a perceived pain in the head, or might cause pressure on the autonomic nervous system leading to a change in intracranial pressure, either or both of which might lead to the headache reported.

However, two weeks of unremitting headache not really responsive to analgesics, with very high blood pressure, and no visual symptoms, even in a patient with a chronic headache history is atypical of a mechanical diagnosis. This therefore warrants further investigation.

CONCLUSION

All of the differentials left, after the elimination process outlined, required an immediate pathological work up. Referral in this case offered the safest outcome for the patient and the quickest method of obtaining it.

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