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Footballer's Migraine

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Summary

Classical migraine, including incapacitating visual field defects, repeatedly developed in five young men immediately after blows to the head while playing football and in no other circumstances. A similar condition occurred in a professional boxer and an isolated attack in a boy footballer. Prophylactic treatment with ergotamine tartrate may not be wholly successful and it may be necessary to give up the sport. Any unitary theory of causation of attacks of migraine must account for prodromal symptoms immediately after head injury.

Introduction

Migraine may be a formidable obstacle to the enjoyment of life but is less well known as an occupational hazard and a threat to professional advancement. I have encountered classical migraine, including incapacitating visual field defects, occurring only when playing football and precipitated by head trauma, in five young men—two whole-time professionals, one a part-time professional, and two amateurs. Two, including the part-time professional, had given up the game on this account.

Case Reports

Case 1.—A whole-time professional footballer aged 22 consulted me in 1965 with a history of seven almost identical episodes in the previous six years. Although they varied in severity, the attacks followed a set pattern. All had occurred while playing football and all had been precipitated by a blow on the head. On a few occasions early in his career this had consisted of deliberate heading of the ball, but more recently symptoms had followed accidental blows either from the ball or from another player. Within two minutes he would develop tunnel vision, which naturally greatly reduced his effectiveness on the field and he would have to come off. The visual disturbance might last for as long as an hour and would later be accompanied by tingling in one or other hand, spreading to the face, and followed by severe generalized headache and vomiting. He had never experienced similar symptoms or indeed any form of headache in any other circumstances. There were no abnormal physical signs. His career obviously depended on his being able to sustain minor blows on the head without having to leave the field, and the prophylactic use of ergotamine tartate seemed justified. The taking of 1 mg before a practice match had no adverse effect and for the next year he took this dose before each match. This did not afford complete protection as in 1966 he developed a typical attack after a severe blow on the face, sufficient to crack two teeth. Recent postal follow-up by courtesy of the club doctor showed that he had not taken ergotamine for the past year and had been free of attacks. Personal follow-up by television shows that he has no inhibitions about heading a football.

Case 2.—An undergraduate of 22 was a keen footballer, but from the age of 15 he had experienced episodes of blurred vision, occurring only after heading the ball. He would immediately develop ill-defined gaps in the visual field and would continue playing only with difficulty. After the game he would feel sleepy and would then develop severe headache and nausea, lasting for several hours. These attacks occurred perhaps twice a year. Two months before I saw him he had been accidently struck on the head by a ball and this had been immediately followed by one of his usual attacks, although the visual defect was more pronounced than usual and he had to leave the field and was taken to a hospital casualty department. After this he found that in every game heading the ball would induce one of his attacks and he had to stop playing. He never experienced similar attacks in any other circumstances. Physical examination and skull *x*-ray picture were normal.

An isolated attack of classical migraine in young boys following a blow on the head at football is not uncommon, but I have been unable to ascertain whether this is the forerunner of ordinary migraine or is confined to minor injuries at football.

Case 3.—A boy of 12 was playing somewhat inexpertly in goal when he was struck on the side of the head by the ball. Within a few minutes he complained of blurred vision and a little later developed numbness of the right hand and difficulty with speech. As this improved, it was followed by severe headache and he was taken to a hospital casualty department by an alarmed games master. The almost equal alarm aroused in the casualty officer was allayed by the boy's rapid recovery and when I saw him a few days later he was quite well. He had no previous history of migraine and his subsequent progress is unknown.

The only other gainful occupation in which the head is necessarily and expressly exposed to trauma is boxing.

Case 4.—A part-time professional featherweight boxer aged 20 consulted me in 1968 because in the previous two months he had experienced four attacks of blurred vision and headache. These had all occurred while boxing and had immediately followed "a good punch" to the head. He thought that his vision would go black for a moment and he would then have tunnel vision, with the peripheral field "like being under water." After about 30 minutes this would be followed by severe generalized headache. All these attacks had occurred while sparring and not during a fight. He had never had similar symptoms at any other time. There were no abnormal physical signs. I advised him to give up boxing as I thought it unlikely that he would achieve much success and he ran the risk of severe punishment. He was reluctant to accept this advice and recent attempts at postal follow-up have been unsuccessful.

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Discussion

In these patients the diagnosis of migraine is indisputable, but, largely because of the widespread and erroneous belief that complicated migraine is associated with vascular anomalies, the occurrence of such incapacitating symptoms only after blows on the head has in each patient given rise to fears of some more serious condition. The complete absence of physical signs and of sequelae did not suggest that elaborate investigations were needed.

There are few references to similar symptoms. Whitty (1967) mentioned the case of a patient who had migraine induced by boxing in his youth, and Graham (1968) referred to "youngsters with classical migraine who develop a characteristic attack when hit on the head in a football match," but presumably not while attempting to head the ball, which under American rules would result in more comprehensive injuries.

Migraine induced in this way is not common among footballers and is not mentioned in standard texts on sporting injuries. Inquiry at two Football League clubs did not reveal any further examples, but it cannot be known how often a promising career in football or boxing is given up on this account. In the professional footballer, threatened with the loss of an enjoyable and lucrative occupation, attempts at prophylactic treatment are clearly justified, but in my limited experience only doubtfully successful. A boxer subject to visual field defects could scarcely be encouraged to pursue so dangerous a career.

I found that in the traditional paramedical lore of the football trainer there is some confusion between "concussion" and headache. The ball weighs from 14 to 16 ounces (400 to 450 g) at the beginning of the game, and although the modern waterproof ball does not increase in weight by the 20% common with the leather ball on a wet pitch, the impact, even when travelling at the quite ordinary speed of 30 miles (48 km) an hour, is not inconsiderable. The footballer is trained to meet the ball, from whatever direction it comes and whatever its intended destination, in a restricted area in the midline in the frontal region. The neck muscles are contracted and the force of the contact may be increased by a sharp movement of the head. It is claimed that correct heading of the ball avoids all ill effects but in a recent 15-minute demonstration by five professionals arranged for my benefit one finished up with a headache.

My patient (Case 1), a most skilful player, was certain that it was the accidental unexpected blow that was most likely to produce an attack. The implications of migraine prodromata induced by minor head injury have been discussed by Haas and Sovner (1969), who drew attention to their occurrence in children and to the understandable but needless alarm that results. They inclined to the view that the blow caused traction on the arteries at the base of the brain, with subsequent spasm. It is reasonable to suppose that an unexpected blow to the side of the head would be more likely to cause arterial distortion than the anticipated and minutely controlled impact of deliberate skilled heading. Other mechanisms and factors may be involved, including violent physical exertion, but the time relations clearly implicate the trauma as the immediate trigger. Any unitary theory of the cause of the migraine attack should therefore take into account migraine occurring immediately or within a minute or two after minor head trauma and at no other time.

My thanks are due to Mr. Denis Follows, Secretary of the Football Association, for information and to Mr. Kenneth Fish, of Oxford United Football Club, for a practical demonstration of heading a football.

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MEDICAL MEMORANDA

Unusual Presenting Symptoms in Neuroblastoma

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Although neuroblastoma is one of the commonest tumours in children (Willis, 1962) the diagnosis is often made late in the disease when there are already widespread metastases. This is because the presenting symptoms are so variable (Thurman and Donaldson, 1967), and in the early stages of the disease they are often non-specific. Careful history-taking often discloses that the child has had several weeks or even months of vague ill health sometimes associated with intermittent abdominal pain or variable bone pains, which may be accompanied by a limp.

Neuroblastoma in the mediastinum may cause compression of the superior vena cava or lead to collapse of a lung from pressure on a main bronchus. Occasionally Horner's syndrome may result from pressure on the cervical sympathetic chain. Ptosis or unequal pupils in a baby are not very obvious signs and do not at once suggest a diagnosis of mediastinal tumour.

Abdominal neuroblastoma arising in the adrenal gland or from the sympathetic chain may cause only a small impalpable tumour when there are already widespread bone metastases present. Where there is a large abdominal tumour this has usually infiltrated surrounding structures by the time of diagnosis and may have extended through the intervertebral foramina causing neurological signs.

The more generalized symptoms may be due to the leucoerythroblastic anaemia caused by bone marrow infiltration by malignant cells. The child may be pale, lacking in energy, and failing to gain weight. Occasionally secretion of pressor amines by the tumour itself may cause hypertension with headaches and chronic diarrhoea.

Two cases that presented with particularly unusual symptoms are reported.

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