The post-viral syndrome: a review

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SUMMARY. The post-viral syndrome is described and its aetiology is discussed. Many features of the syndrome point to hysteria and altered medical perception as causes but much evidence for organic disease is also presented. Current interest focuses on recent or persisting infection with Coxsackie viruses. A balanced view of the syndrome as a mixture of organic and psychiatric dysfunction is offered. Widely differing estimates of incidence are quoted, possibly owing to varying medical awareness of the syndrome. Many drug therapies have been tried without success and management of the post-viral syndrome is hampered by the reluctance of patients to accept psychiatric support once the diagnosis is known. Many names have been proposed for the syndrome, some implying a purely physical or purely psychogenic aetiology: post-viral syndrome is suggested as the most appropriate term. Increased awareness of the syndrome will lead to an increase in its diagnosis in general practice: the role of the Myalgic Encephalomyelitis Association in promoting a combined psychiatric and organic view of the disease among sufferers is emphasized.

Introduction

POST-VIRAL syndrome is the current name for an illness with almost as many names as symptoms. It is also referred to as myalgic encephalomyelitis and as epidemic neuromyasthenia, and has been called Akureyri disease, Iceland disease and Royal Free disease in the past.[1] The illness occurs both sporadically and in epidemics. Females are affected up to 10 times more often than males and all-female institutions, such as nurses homes, are favoured sites of epidemics.[2]

Symptoms and course of illness

The syndrome typically follows an upper respiratory tract infection from which the sufferer fails to make a full recovery, complaining of a multitude of symptoms which may persist for months or even years. The cardinal symptom is profound muscular fatigue[3] and this is often accompanied by muscle pain, headache, paraesthesiae, dizziness, urinary frequency, cold extremities, bouts of sweating and fainting attacks.[4] Other symptoms are poor memory, lack of concentration, sleep disturbance, mild expressive and receptive dysphasia, hyperacusis and emotional lability.[5] Clinical examination usually shows no abnormalities, nor do routine laboratory investigations. The diagnosis is therefore one of exclusion.

The illness follows one of three courses: many patients recover completely, in others there is a relapsing and remitting course and in some there is chronic illness.[6] Relapses are precipitated by undue physical or mental stress: patients who rest adequately in the early stages are said to have the best chance of an early, complete recovery without relapse.[4,5]

Aetiology

In 1978 an international symposium on myalgic encephalomyelitis concluded that this is a specific disease entity of viral origin and is not psychogenic.[7] This conclusion was not universally endorsed, and it was also suggested that it is a syndrome with a variety of causes. Persistant viral infection and virus-mediated damage to the immune system are the principal causes postulated.[8] Many viruses have been implicated, notably the Coxsackie viruses,[9] but also varicella, influenza and Epstein-Barr viruses.[10] Other hypotheses include disruption of glycolysis in nerve and muscle fibres[11] and injury to the dorsal raphe nucleus[12] owing to virus infections.

Evidence for hysteria

Doubt has been cast on the organic nature of the disease. Hysteria may account for many of the cases,[13] and altered medical perception may result in the grouping together of many dissimilar illnesses as one specific entity.[14] The symptoms have changed in character over the years, and current definitions differ markedly from the acute illness with paralytic manifestations described in early reports.[7] Altered medical perception may be responsible for this grouping together of dissimilar epidemics as examples of one single disease.

A reappraisal of the Royal Free epidemic of 1955 highlights factors which point to hysteria as an underlying cause of the illness: the frequency of the disorder among women, the similarity of symptoms to those reported in outbreaks of mass hysteria, the glove and stocking distribution of sensory loss, the normal results of laboratory investigations and the reproducibility by normal subjects of claimed abnormal electromyographic test results.[2] Ramsay has recently admitted that 92 of the original 292 cases during the Royal Free epidemic were excluded from the subsequent report because their symptoms were considered to be hysterical in origin.[15] Other outbreaks have been studied in retrospect and similar conclusions have been reached.[13]

The disease has high predilections for medical personnel[3] and for those in social classes 1 and 2[16] and only rarely occurs in more than one member of a family;[17] these facts may be seen as further evidence of its hysterical nature. One report of an epidemic emphasizes the need to limit the number of laboratory investigations since investigation may increase the severity of the symptoms.[18] An increased incidence of pre-existing neurosis among sufferers has also been reported.[14]

Evidence for organic disease

However compelling the evidence for an hysterical basis may be, there is further, equally compelling, evidence of organic disease. Some patients do have frank neurological signs[6] and some routine laboratory tests may be abnormal.[3,19] A recent study[8] demonstrated abnormal lymphocyte function, a reduction in the number of suppressor and helper T-cells and the presence of circulating immune complexes. Muscle biopsies showed necrosis and type II fibre predominance. Single-fibre electromyography has demonstrated abnormal jitter potentials[20] and nuclear magnetic resonance has revealed abnormally early intracellular acidosis during exercise.[21]

The role of the Coxsackie viruses has been studied by several groups. Behan and colleagues found antibodies to Coxsackie viruses in 70% of the 50 cases studied.[8] Calder and colleagues found 46% of 140 cases to be Coxsackie antibody positive, compared with 25% of 100 controls.[22] McCartney and colleagues found specific immunoglobulin (Ig)M Coxsackie antibodies in 31% of 118 patients.[23] These results have been interpreted as implying recent or persisting infection with these viruses.

A balanced view

If the evidence for hysteria seems convincing and the evidence for organic disease seems irrefutable, what then is the true nature of this illness? Authors on the subject fall into two camps, and they declare their support for the psychogenic hypothesis or for the organic hypothesis. Might not the truth lie somewhere in between? Hysterical cases do occur during outbreaks,[24] functional overlay occurs[15] and psychiatric symptoms are common.[25] General practitioners should keep an open mind and should consider whether each case contains elements of both organic and psychiatric dysfunction.

Many patients with the post-viral syndrome have psychological symptoms, including emotional lability, inability to concentrate, depression and hysterical overlay of physical symptoms.[25] These psychological disturbances may persist for many years and may be severe: cases of depression requiring treatment with electroconvulsive therapy have been described.[26] Schizophrenia, paraphrenia and anxiety states have also been reported[25,26] and psychiatric illness may rarely be the first manifestation of the illness.[27]

Epidemic or sporadic?

There are many reports of epidemics of the post-viral syndrome but little information is available on the sporadic form. Some authors consider that sporadic cases are rare and that most cases occur during epidemics. However, this low incidence of sporadic cases may be due to a lack of awareness of the syndrome: Keighly and Bell found 20 cases in a population of 2500 during 14 months[17] and Calder and colleagues found 140 cases in a population of 10 000 in just six months.[22] The incidence of the illness has been estimated to be about 3 in 100 000,[8] though the figure has been placed as high as 1 in 1000.[28]

Mass hysteria is clearly not a feature of sporadic cases, but altered medical perception may be, since many reports of sporadic cases are by authors with experience of epidemics.[13] Sporadic cases will usually present late or may remain undiagnosed for years. They may simply be regarded as examples of the 'fat folder' syndrome.

The Myalgic Encephalomyelitis Association

A self-help group, the Myalgic Encephalomyelitis Association exists to assist sufferers, to disseminate information about the disease and to sponsor research into its aetiology. The Association states that there are some 80 000 to 120 000 cases of myalgic encephalomyelitis in the United Kingdom and over 500 000 cases worldwide,[29] but data to support these figures have not been provided. The objectives of the research are to create a 'definitive test' and to find a cure for the disease.[29] Recent publicity of Coxsackie virus antibody research findings has resulted in claims in the national press that the definitive test has now been found,[30] but these may be premature since relatively few patients are positive to Coxsackie B.[22] Unfortunately perhaps, in propagating information about its research involvement, the Myalgic Encephalomyelitis Association provides patients with straws to grasp at in their search for cures.

Management

Should patients be 'labelled' with the diagnosis of post-viral syndrome at all? Behan[31] suggests that many patients with psychiatric illness are mis-labelled with this diagnosis. Although it may be heartening to have a firm diagnosis of organic disease,[16] this may make people reluctant to accept psychiatric treatment. Whether the initial process is organic or hysterical there is probably a psychiatric disturbance, either pre-existing or secondary to the disease, in most patients.[25] Many would benefit from psychiatric help, and a blinkered view that the disease is purely organic might be harmful. The diagnosis of post-viral syndrome should probably only be made in the absence of pre-existing neurotic traits.

Research into the use of IgG injections has been reported,[15] and patients may request such treatment arbitrarily despite the absence of completed clinical trials. The current vogue is for treatment with oral anti-fungal agents: although no trial results have yet been published, details for self-treatment with drugs such as amphotericin were given in the Myalgic Encephalomyelitis Association's Autumn 1986 newsletter.[32]

Rest is the mainstay of treatment, and further treatment is symptomatic: analgesics, anxiolytics and antidepressants may be required. Carbamazepine has been advocated for the treatment of severe muscle aches and pizotifen for headaches.[19] Some patients find that caffeine or alcohol cause a worsening of their symptoms and avoidance of these substances should then be counselled.[19] The condition has been treated in the past with steroids, azathioprine, plasma exchange, inosine pranobex, acyclovir, interferon and sulphasalazine, all without significant improvement over placebo treatment.[33] Perhaps unsurprisingly, given the lack of success so far with conventional medicine, patients turn to less conventional treatments such as exclusion diets, dietary supplements and allergy treatments.

Nomenclature

Just as in the debate over aetiology there are opposing views on nomenclature: myalgic encephalomyelitis is the banner of the 'organic' camp,[7] while others have suggested myalgia nervosa as an appropriate name for a disease with an hysterical cause.[13] The American term epidemic neuromyasthenia has been opposed on the grounds that the disease process bears little resemblance to that of the myasthenias.[7] Post-viral syndrome describes the illness without implying anything about aetiology and is therefore the preferable term.

Conclusion

The post-viral syndrome is a mixed-bag of organic and psychiatric disease, and finding a definitive test to prove a viral aetiology will not lessen the psychiatric symptoms of its sufferers. With the increasing activity of the Myalgic Encephalomyelitis Association, general practitioners must expect to see, diagnose and treat more cases in the coming years. If the Association comes to accept the need for a mixed physical and psychiatric approach to the disease, regardless of aetiology, it may be able to play a much more positive role in promoting the return to health of its members.

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