Holmes Tremor: A Therapeutic Challenge

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Editorial

Holmes Tremor (HT) is a rare form of combined tremor that occurs at rest, intention, and posture. This current definition of HT is derived from the Consensus Statement of the Movement Disorder Society on Tremor from 1998 [1]. It was first described by Benedict in 1889 as a consequence of mesencephalic infarct, and though was termed as “mesencephalic”, “rubral”, or “thalamic” tremor in the past, which are no longer in use [2]. Recently, this 3–4 Hz low frequency, irregular tremor which tends to occur at rest and worsens with posture and action is known as “Holmes Tremor” regarding to the description of Gordon Holmes in 1904. It is reported that there is an estimated latency of 2 weeks to 2 years from lesion to tremor onset [3].

It has been reported that HT arises from the lesions of brainstem, and/or cerebellar connections that interrupt the dentate–thalamic and the nigrostriatal tracts thus causing both an action and a rest tremor [2]. Since it is a symptomatic tremor, various aetiologies including structural disorders like cerebrovascular diseases, vascular malformations or tumours may underlie HT. However, there may be some cases without any demonstrable lesion, and a reversible case of HT has been reported due to spontaneous intracranial hypotension [3,4]. Although it is known as a challenging and complex tremor with a resistance to pharmacological agents, levodopa is reported as a first line drug to be tried, which may be helpful in many cases additionally, dopaminergic agonists as pramipexole, topiramate, levatiracetam, and onabotulinum toxin and injections can be helpful in some cases and worth trying [5-7].

Regarding to the drug-resistant feature of HT, and the limited therapeutic effect of the drugs described above, there is an ongoing search and interest for the functional surgery such as deep brain stimulation and thalamotomy in the treatment of HT [8-10]. There is a case-report revealing the beneficial effects of nucleus ventro Inter Medius (Vim) thalamotomy in a young patient with HT that resulted with a marked improvement in the rest and postural tremor of the patient. However, a mild kinetic tremor is reported to be remained [8]. A younger patient with HT due to thalamic abscess has been reported to improve after thalamic DBS by Peker and his colloquies [8]. Besides, DBS targeting pallidum has also shown to be effective in treating HT [10].

Since HT is a rare and complex tremor syndrome which is difficult to treat, it is a real therapeutic challenge for clinicians, neurologists, as well as the movement disorders specialists. It is important to keep in mind to investigate the possible underlying etiologies to adjust a proper algorithm for the treatment of HT. However, a symptomatic treatment is always crucial with the pharmacological agents that are reported to be more or less effective, and functional surgery options should be considered in cases of HT which are resistant to drugs.

References

