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Short Communication

Augmentation of Management of Depression and Epilepsy: A Novel Strategy

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Depression and epilepsy both are highly prevalent disorders globally and a strong comorbidity between the two appears to be existing since ancient times in view of Hippocrates writing around 400 BC "melancholics ordinarily become epileptics, and epileptics, melancholics".

Depression is the most commonly observed comorbid psychiatric disorder in epilepsy. In patients with epilepsy (PWE), the prevalence of depression reported is found to vary from 20 - 55% [1]. On the other hand, there is a definite increase in the risk of epilepsy in patients with depression or with a previous history of attempted suicide [2]. Another study [3] has reported an association of 4- to 6-fold greater risk of developing epilepsy in patients with a history of depression suggesting a possible bi-directional relationship between depression and epilepsy or the presence of common pathogenic mechanisms that facilitate the occurrence of one in the presence of the other.

There is also evidence of even sharing of similar neural networks in depression and epilepsy which researchers claim as a possible representation of an epiphenomenon [4], the evidence of a common neurocircuitry emerging from voxel based morphometry (VBM) studies.

Depression can commonly cause sleep deprivation that in turn may lead to increased incidence of seizures; conversely, epilepsy that is still associated with a strong social stigma can lead to depression both through biological mechanisms and its psychosocial impact [5].

The incidence of treatment resistant depression (TRD) and drug resistant epilepsy (DRE) that fail to respond satisfactorily to drug-medication is 10 - 30% and 30% respectively. In the author's opinion, a significant contribution to the high incidence of TRD and DRE could be attributed to the bidirectional relationship (or comorbidity) that depression and epilepsy have been found to exhibit with each other, not to mention the possibility of aggravating one another. Unfortunately, this relationship has not been explored to the extent it necessitates especially from clinical and management point of view. This is further confounded by the fact that the medication drugs of the two disorders can potentially interfere with each other leading to possible suppression or modification of their expected efficacy. To exemplify, some antidepressants are known to lower the epileptic threshold and increase seizure frequency.

A significant and inadvertent possibility is treating either disorder in isolation with an oversight or oblivion to the possibility of its comorbidity, in which case the management could be incomplete and compromised if the comorbid disorder is overlooked and not treated simultaneously.

Therefore, the author postulates mandatory investigation of concomitant comorbidity before initiation of definitive treatment of either depression or epilepsy and strongly recommends invariable incorporation of deliberation on possible concomitant comorbidity in their management to yield best possible therapeutic efficacy and success.

The author speculates that the postulated strategy may aid in reducing the incidence of drug resistance in depression and epilepsy; albeit, well designed prospective studies are strongly recommended. Thus, the novel strategy of assessment of the bidirectional relationship or comorbidity prior to initiation of treatment of depression and epilepsy is likely to enhance the treatment of both disorders and at the same time also reduces the incidence of TRD and/or DRE.

Declarations of Interest

None.

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