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Neurometabolic evidences for diabetes and intermittent explosive disorder: A case report

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Background: Despite studies suggesting that increased insulin resistance and higher fasting glucose levels may secondary lead to glucose hypo metabolism in specific brain regions which may contribute to the development of cognitive disturbances during diabetes1-2, the underlying mechanism of diabetes associated impulse control disorders is still unclear. Interestingly, a very recent study has demonstrated that impulse control disorders (especially intermittent explosive disorder) were significantly associated with the diabetes diagnosis. Thus, studies have reported that intermittent explosive disorder and border line personality are associated with an increase of glucose metabolism in the limbic system and a decrease in prefrontal regions supporting the role of dynamically interacting cortico-subcortical networks. Besides their key role as a pacemaker for cortical centers via purely motoric coordination, lentiform nucleus and pons have been also shown to play a significant role in emotional process in also involving reflexive emotional reactions. This is in line with previous evidences showing that gray matter volume reductions in neocortical regions may be specific to psychiatric disorders. These findings together suggest that a disturbance in connectivity between different brain regions, rather than abnormalities within the separate regions themselves, may be responsible for the clinical symptoms of intermittent explosive disorder.

Case: Here we describe a 36-year-old man, experience the aggressive outburst symptoms one year after he was diagnosed as type 2 DM. The patient describes severe physical and verbal violence precipitated by little provocation and fulfilled the DSM-IV diagnostic criteria for intermittent explosive disorder. On psychiatric examination, he was cooperative, alert, and fully oriented and displayed an appopriate emotional display. Psychiatric and neurological examinations were otherwise normal. The patient scored eight points of sum score on Modified Overt Aggression Scale. Mini-Mental State Examination and the original Beck's Depression Inventory revealed no abnormality. Detailed blood tests were found to be normal except significantly elevated HbA1c levels(13%). A magnetic resonance imaging scan of the brain performed on the admission day showed no abnormality. In contrast, reduced glucose uptake in PET was prominent on the left pontin area, left temporoparietal cortex and lentiform nucleus.

Discussion: In our case, functional abnormalities did not entirely parallel morphological changes, and were found in the temporoparietal and subcortical regions which appeared to be rather unaffected in MRI. This indicates that the reduced glucose uptake observed in the respective cortical regions may reflect secondary deficits due to diminished functions of emotional circuits involving the basal ganglia and brain stem and suggest that diabetic individuals may be vulnerable to lower cerebral glucose metabolism in regions implicated in the pathogenesis of intermittent explosive disorder. In conclusion, we present a typical presentation of presumed emotional incontinency in a 36-year-old man with diabetes and the correlation of the metabolic changes seen with his change in emotional state. This type of quantitative analysis can provide that information, unlike a subjective radiological evaluation limited with MRI and CT. A greater understanding of the functional activity of the underlying regions affected by diabetes and intermittent explosive disorder may help to provide insight into specific networks involved.

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