Case report

Neurocognitive deficits in an adult road traffic accident victim

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Summary

Road traffic accident (RTA) is very common phenomenon in our country and brain injury due to RTA is most common consequence. As there is brain injury, there might be psychiatric manifestation too. A case of neurocognitive disorder due to RTA had been illustrated here. Diagnosis was made by his complaints of forgetting things, self-talking, poor self-care and slowness. Clinician as well as psychiatrists should be aware of the scenario during clinical practice.

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Introduction

Developing countries like Bangladesh has known to have more disease burden.[1] There have been many cases of road traffic accidents (RTAs) resulting from low construction standards and poor road maintenance.[2] According to the latest WHO data published in 2020 RTAs Deaths in Bangladesh reached 25,023 or 3.50% of total deaths. The age adjusted Death Rate was 16.74 per 100,000 of population ranked Bangladesh 88 in the world.[3] Transportation in the country with two-wheelers accounted for the majority of traffic incidents.[4] Involvement in RTAs might put individuals at increased risk for a wide range of psychiatric disorders that had significant public health issues and the RTA even itself could trigger existing psychiatric disorder.[5]

The mental health disorders and the traumatic brain injury (TBI) associated with RTA could have an adverse effect on the neurocognitive functioning of the survivor. One study that was aimed at comparing the health status and quality of life between patients with whiplash and other injuries, the psycho-cognitive sequelae stood at 32.7%, 27.5% and 10.7% for severe TBI, mild TBI and other injuries respectively.[6] There are three cognitive changes that was found in people with trauma and they include: impaired memory, concentration difficulties and difficulties associated with finishing a task.[7]. Here exemplified a case of mild neurocognitive disorder of an adult male due to RTA.

Case summary

A 40-years-old, married, male presented in a psychiatry institute with the complaints of forgetting things for 4 years, self-talking and poor self-care for 2 years and slowness for 1 year. His onset was gradual and course was continuous. All his complaints started following a decompressive craniectomy done after an RTA 4 years back. The injury was moderate to severe in nature and it caused him long time functional impairment in occupational, familial, spiritual, personal and social domain slowly. Though there was marked anterograde amnesia, it was not noticed early by family members and he was also not concerned about it. There were some behavioral and sleep problems. That time he was diagnosed as schizophrenia on the basis of history. Then he was given antipsychotics like fluphenazine decanoate injection 4 weekly and olanzapine, he was not improving and deteriorated everyday with onset of new problematic symptoms. In this period, he was treated in psychiatry outpatient department (OPD) of a medical college hospital, but compliance was poor. Improvement was never more than 20%. Thereafter, they visited OPD of National institute of mental health and prescribed antidepressant and antipsychotics. Despite multiple treatment, he never improved more than 40%. On mental state examination, apathetic look was noticed with reduced eye blinking and psychomotor retardation, decreased speech rate and volume was documented. Mood was reactive but low. Marked cognitive impairment was noticed in attention, abstract reasoning and intelligence. Recent memory was impaired. Patient didn't think he had any problem. On nervous system examination, speech comprehension seemed impaired with intact fluency and repetition. Mini mental state examination (MMSE) score was 16. Wechsler Abbreviated Scale of Intelligence (WASI) score came extremely low. In MRI of brain, there was two important impression

we had found which was: chronic infracts with encephalomalacic and gliotic changes in left temporal lobe (post traumatic). Evidence of craniotomy noted at left parieto-temporal bones with overlying soft tissue scar. His treatment was started with memantadine hydrochloride and clonazepam. Psychoeducation was given to family members regarding nature of the disease, structuring the environment, structuring the daily activities as a part of occupational therapy was also offered.

Discussion

This case highlighted several important issues and challenges. Diagnosis of this patient was one of them. The mental health disorders and the TBI associated with RTA could have an adverse effect on the neurocognitive functioning of the survivor. One study that was aimed at comparing the health status and quality of life between patients with whiplash and other injuries, the psycho-cognitive sequelae stood at 32.7%, 27.5% and 10.7% for severe TBI, mild TBI and other injuries respectively.[6]

There were three cognitive changes that were found in people with trauma and they include: impaired memory, concentration difficulties and difficulties associated with finishing a task. Three published studies had prospectively assessed for cognitive deficits. These had implicated verbal memory, sustained attention, verbal learning, visuospatial memory and reaction-time proficiency deficits and trended toward poorer cumulative learning and verbal fluency.[6] Trauma had been associated with deficits in the areas of verbal memory and learning, executive functioning, working memory and attention in adults.

In this case, patient was presented with the complaints of forgetting things for 4 years, self-talking and poor self-care for 2 years and slowness for 1 year. Then he was diagnosed as schizophrenia on the basis of history and was given antipsychotics like fluphenazine decanoate injection 4 weekly and olanzapine, he was not improving and deteriorated everyday with onset of new problematic symptoms. In hospital setting, after careful history, mental state examination and applying psychometric scale we found loss of consciousness, post traumatic amnesia and disorientation after trauma. Prominent evidence of cognitive decline after the RTA was found. Decreased speech rate and volume was documented. Cognitive deficit was hampering his day-to-day functioning along with night time worsening of symptoms. Patient tried to give answers though recent memory was impaired gradually. On nervous system examination, speech comprehension seemed impaired with intact fluency and repetition. MMSE score was 16 and WASI score came extremely low. On the basis of these symptoms, diagnosis made was major neurocognitive disorder due to traumatic brain injury.

Conclusion

An effective evaluation seems to be pivotal in the management of mild cognitive disorder due to road traffic accident which demands proper attention of the clinicians including psychiatrists. More extensive epidemiological studies are recommended to highlight diagnosis as well as management of this condition.

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