



Short Communication

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Stiff Person Syndrome- the forgotten cause of Depression

Anshul Arora*

Corresponding Author: Anshul Arora,

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I am honored and excited to present an exciting case during my rotation in the Department of geriatric Psychiatry.

Before getting into the case, I would like to introduce myself. I am Dr. Anshul Arora, a Board-certified Neurologist based in Germany.

During my last rotation in Psychiatry, I encountered several interesting patients, one of them on whom this case report is based on.

A Patient aged 50 was admitted because of progressive cognitive Impairment and depression. Over the years there was a decline of movement. The mood was reported depressed. The Patient would just be lying in bed without doing anything during the day. The appetite was decreased as well as a result he lost a sufficient amount of weight.

Apart from that there was cognitive decline with episodes of aggression during the evening.

From the past medical history chronic consume of Alcohol there was noted but was never proved.

On Examination the Patient was afebrile. He was alert to Person but not oriented to Place, Situation and time.

Furthermore, the mood looked depressed. The Affect was reduced. The drive decreased as well.

In the minimal Test the patient achieved 10 Points out of possible 30 points, which correlates with a severe care of

Upon neurological Examination the cranial nerves were intact. Hyperreflexia was noted. Signs after Babinski, Hoffmann, Chaddock as well Trömer were negative. The Gait displayed a reduced step length. Apart from that spasm and random myoclonic jerks were noted which worsened after tactile stimuli.

To rule out a cerebral Pathology a computer tomography of the head was done which yielded a slight general atrophy. Indications for an acute cerebral ischemia or intracranial bleeding could not be detected.

The laboratory analysis didn't show any pathological changes.

To exclude an infection of the central nervous system and to detect the markers for Dementia as well to exclude an encephalitis an analysis of the cerebrospinal fluid was done which ruled out an infection.

The further Evaluation yielded a positive Titer antibody against GAD.

To rule out a paraneoplastic syndrome a further evaluation of the thorax and abdomen with the aid of computer tomography was conducted which showed no indication of malignancy.

In view of the clinical and paraclinical findings the suspicion of Stiff Person Syndrome was raised.

We initiated a treatment consisting Methylprednisolone intravenous. Because of the cognitive impairment, the patient kept removing the intravenous line so that it had to be changed to Prednisolone per os.

In addition to the patient received Lorazepam and Baclofen.

After duration of 2 Weeks there were clinical improvements. The myoclonic jerks were reduced. The affect was much better. The hyposmia was reversible.

The Patient was transferred to neurological ward for further diagnostics.

Definition of Stiff Person Syndrome

Stiff person Syndrome also known as Stiff Man Syndrome is a rare neurological disorder of unclear cause characterized by progressive rigidity and stiffness. The Stiffness primarily affects the truncal muscles and is superimposed by spasms, resulting in postural deformities.

Pathogenesis:

In stiff person syndrome, spinal interneurons function to inhibit spontaneous discharges from spinal motor neurons, primarily through the action of glycine. However, this is only one inhibitory input for the motor pathway that includes GABA-mediated inhibition from the cortex, brain stem, and cerebellum. If GAD function is inhibited significantly, then GABA available for these functions is decreased and muscles become continuously stimulated by the motor neurons. Additional possible pathophysiologic etiologies in patients negative for GAD antibody include postsynaptic elements such as synaptophysin, amphiphysin, [2] gephyrin, [3] and GABA-transaminase.

In simple words

Treatment

Discussion & Conclusion

Having a patient of psychiatric disorder can be difficult. Not only because of the difficulty of the patient's condition but because of the difficulties coming along with it. Usually once a patient has been diagnosed with a psychiatric disorder it's difficult to overlook it. In other field there are Diagnosis of Exclusions which are concluded after a thorough work up where as patients with psychiatric clinical manifestations are concluded instantly without a in depth work up. Even when a patient is seeking justice, he/ she is innocent until proven otherwise but in psychiatric patients its mostly psychiatric disorder until proven otherwise.

Similar situation occurred with our patient. Because of his commodities he was subjected first as a patient with dementia with depression as a result of his presumed alcohol misuse, where as he actually had an autoimmune encephalitis. If an early detection would have helped his cognitive decline is a subject to imagination.

Over the years I have encountered several patients who actually had an underlying neurological disorder but were overlooked by their psychiatric manifestations.

The take home message is that a diagnosis of psychiatric disorder at any age should be a diagnosis of exclusion and not otherwise. Colleagues from different field need to be comfortable with patients with psychiatric manifestations rather than directing them to psychiatric ward. At the end we are solely there for the patient and should the patient should not be stigmatized for disorder he develops.

Reference

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