**POSTER NUMBER**

8

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**ABSTRACT CATEGORY**

Clinical Research

**ABSTRACT TITLE**

Beyond Increased Muscle Tone: Co-occurrence of Stiff Person Syndrome and Idiopathic Parkinson’s Disease

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OBJECTIVE

Here, we report an intriguing case of a 71-year-old man who presented with a 2-year history of gait instability, bilateral leg stiffness, and loss of hand dexterity. To our knowledge, this is the first reported evidence of co-occurrence between Stiff Person Syndrome (SPS) and idiopathic Parkinson’s Disease (iPD).

BACKGROUND

SPS is a rare neuroimmunological disorder that presents with increased rigidity, triggered muscle spasms, and hyperlordosis. PD has been reported to co-occur with autoimmune conditions; however, significant overlapping symptomatology can pose diagnostic challenges.

METHODS

Initial examination showed bradykinesia and symmetrically increased tone in all extremities, hyperreflexia, and shuffling gait with short stride length. Imaging investigations were unremarkable, including MRI brain and cervical spine, DaT-SPECT brain, and PET CT brain. Laboratory analysis revealed elevated cerebrospinal fluid GAD-65 levels (0.41 nmol/L) and serum GAD-65 antibodies (>250 IU/ml), leading to the diagnosis of SPS. The patient initially received intravenous immunoglobulin (IVIG) therapy, followed by rituximab due to inadequate response.

Over time, the patient exhibited more pronounced asymmetrical parkinsonism. He developed action and resting tremors (right>left), micrographia, and hypophonic speech. Bradykinesia and rigidity were predominantly worse on the right side. A Syn-One biopsy was subsequently performed which revealed abnormal phosphorylated deposition in the cervical region, leading to initiation of carbidopa-levodopa.

RESULTS

Approximately 6% of GAD-65 antibody-positive cases have been reported to exhibit signs of hemiparkinsonism. While previously reported cases relied on clinical examination and DaT-SPECT [2], this unique case highlights the importance of obtaining further objective evidence, such as skin biopsy, in challenging diagnoses.

CONCLUSION

This case suggests that SPS might trigger an autoimmune cascade that accelerates phosphorylated alpha-synuclein accumulation, a hallmark of synucleopathies like iPD. Increased T cell reactivity suggesting underlying autoimmunity has been observed up to 10 years before iPD motor symptoms. Further research is essential to elucidate the potential link between SPS and synucleopathies.