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Motor Deterioration Of Parkinsons Disease Due To Infection In A Geriatric Indevidual:A Diagnostic And Therapeutic Challenge.

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Abstract: *Clinicians are well aware of the fact that patients with Parkinson's disease may significantly deteriorate following systemic infection or, in its most severe case, may even develop an akinetic crisis. Although this phenomenon is widely observed and has a major impact on the patients' condition, the knowledge about the underlying mechanisms behind is still sparse. Possible explanations encompass changes in the pharmacodynamics of the dopaminergic drugs, altered dopamine metabolism in the brain, alterations in the dopaminergic transmission in the striatum or an enhancement of neurodegeneration due to remote effects of peripheral inflammatory processes or circulating bacterial toxins.*

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I. Introduction

Parkinson's disease (PD) is characterized by a progressive loss of dopaminergic neurons. The clinical picture encompasses motor and nonmotor symptoms with a slow deterioration over years.[1] A considerable number of patients, however, may experience a subacute worsening of their condition including their motor symptoms during systemic infections.[2] Not all patients completely recover from this deterioration, thus leading to a higher level of disability and the requirement of higher doses of dopaminergics afterwards.[2,3] In its most severe form, infections may trigger a life-threatening akinetic crisis featuring severe akinesia, cognitive and psychotic disturbances, fever, impaired consciousness, dysphagia, and speech problems. Patients then often become unresponsive to antiparkinsonian drugs.[4]Infections are furthermore often accompanied by delirium, in PD frequently presenting in its' hypoactive form.[5]

II. Materials And Methods

Sankar Adak a 70 yr old male and a diagnosed case of parkinsonsdisease,diagnosed 1year backand was on irregular medication presented withh/o fever for last 2 days along with severe orientaion.Patient was apparently normal 3days back,butfollwing this episode of fever the patient has become completely bedbound.On presentation, BP was 90 /60,pulse 120/minute,tachypnoea was present.

On examination patient had leadpipe and cogwheel rigidity on superior extremities, increase tone and resting tremor. Terminal neck rigidity was there but kernigs signwas negative.

Complete blood count was sent along with CRP..it showed neutrophilic leucocytosis and CRP was also raised.To rule out meningitis csf study was done which was normal.

The patient was on irregular medication for last 10 days (antiparkinsonian drugs) and along with that this recent episode of fever made his condition worse.Suspecting it as a case of motor deterioration of parkinsons disease due to underlying infectionbroad spectrum antibiotics along with levodopa carbidopa combination was started,which resulted in remarkable improvement in motor symptoms within 24 hours.

III. Discussion

The view of previous studies on this issue has often been blurred by focussing on reasons for subacute deterioration of PD as a whole, particularly in the context of hospitalizations.[7,8] There is only one study by Umemura *et al.* who systematically analyzed the impact of systemic infections alone in a retrospective case–control study.[2] Of 80 PD patients with systemic inflammation, 26 had experienced prolonged and sustained motor deterioration afterward. High body temperature and delirium were significantly associated with motor deterioration, whereas other factors like patient's age, disease duration, pre-existing Hoehn and Yahr stage, and

the presence of dementia were not predictive in this regard. Beyond this study, evidence is limited in this field. One major gap, besides the considerable impact of infections itself, is the lack of understanding of pathophysiological mechanisms underlying infection-related PD deterioration, underscoring the need for further studies. The aim of this viewpoint is to provide some pathophysiological considerations, which could be integrated for future studies. We have divided these under the following possibilities: (1) altered medication intake; (2) altered dopamine metabolism and receptor signaling; (3) enhancement of neurodegeneration by peripheral inflammation.

IV. Conclusion

In theory, several different mechanisms may be involved in symptom deterioration of PD with systemic infections. Possible explanations are altered dopamine metabolism, insensitivity to dopamine at receptor level, enhancement of ongoing neuroinflammatory processes in PD, or altered drug intake. The available literature does not permit final conclusions on the underlying mechanisms, so that further studies in this field are therefore warranted. A long-term follow-up of PD patients including biomarkers and imaging assessments is probably the best approach to elucidate the relevant molecular pathways and to identify predictive risk factors.And in geriatric population who are at increased risk of acquiring infection and progressive neurodegeneration,prompt diagnosis and appropriate management of motor worsening of parkinsons disease may bring down the morbidity and mortality due to PD.

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