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Assessment of Autonomic Dysfunction in Iraqi Patients with Parkinson's Disease using the SSR

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Abstract

Background: Parkinson's disease (PD) is a progressive neurodegenerative disorder that affects both motor and non-motor functions, including the autonomic nervous system. Autonomic dysfunction, particularly involving sympathetic pathways, is a common but underdiagnosed feature in PD.

Objectives: This study aimed to assess autonomic dysfunction in Iraqi patients with Parkinson's disease using the Sympathetic Skin Response (SSR) test as a non-invasive neurophysiological tool.

Methods: A case-control study was conducted at Ghazi Al-Hariri Surgical Teaching Hospital, involving 40 patients diagnosed with PD and 40 age-matched healthy controls. All participants underwent SSR testing to evaluate latency and amplitude in the hands and feet under standardized conditions.

Results: PD patients exhibited significantly prolonged SSR latencies and markedly reduced amplitudes in both hands and feet compared to controls ($p < 0.001$). Clinically, the most frequent autonomic symptoms were constipation (87.5%), impotence (80.0%), and dysphagia (75.0%).

Conclusion: The findings indicate a clear disruption in sympathetic autonomic function among PD patients, as revealed by SSR testing. This highlights the importance of early autonomic assessment in PD to facilitate timely interventions and improve patient care.

Keywords: Parkinson's disease, Autonomic dysfunction, Sympathetic skin response, SSR, Neurophysiology

1. Introduction

Parkinson's disease (PD) ranks second to Alzheimer's disease among common neurodegenerative illnesses. The current diagnostic criteria depend on the presence of bradykinesia as the primary symptom, accompanied by either rest tremor, stiffness, or both [1, 2]. The presentation is typically complicated, encompassing other non-motor symptoms such as sleep disturbances and autonomic dysregulation [3]. Autonomic dysfunction in PD patients is commonly acknowledged and presents numerous forms that might impact the quality of life and overall well-being of patients [4, 5]. The non-motor manifestations of autonomic dysfunction include gastrointestinal motility issues (such as constipation), cardiovascular dysregulation characterized by orthostatic hypotension and tachycardia, urinary and sexual dysfunction, as well as disturbances in sweating and thermoregulation [6].

A large Chinese cohort study estimated that around 91.8% of people with Parkinson's disease exhibit autonomic dysfunction [7]. PD results from the loss of neurons in several brain regions, leading to both movement dysfunction and other impairments. The primary disease is the degradation of dopaminergic neurons in the substantia nigra pars compacta of the midbrain [8]. The disorder is defined by the buildup of Lewy bodies, which consist of insoluble aggregates of the protein alpha-synuclein. The extensive diversity in presentation arises from the pathological process's capacity to impact various neurons in the brain, indicating the participation of

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non-dopaminergic neurons [9, 10]. PD is expected to impact approximately 6.1 million individuals globally [11]. The prevalence grows with age; individuals over 65 exhibit a prevalence of 1–1.5%, which escalates to 4–5% by the age of 80 (Parkinson's Foundation) [12]. The rising incidence of Parkinson's disease, coupled with inadequate management of patients, necessitates a nuanced instrument for evaluating autonomic function.

2. Materials and methods

2.1. Pateinta and study design

This case-control study was undertaken at Ghazi Al-Hariri Surgical Teaching Hospital, which has a recognized affiliation with Baghdad Medical College. We have 40 patients with a confirmed diagnosis of PD according to the international criteria for Parkinson's and movement disorders. All patients are over the age of 40. All 40 individuals underwent clinical assessment, including a history and neurological examination, with particular emphasis on the symptoms of autonomic dysfunction during the history-taking process. Moreover, 40 subjects with a normal neurological examination and history comprise the control group. They were age-matched to the group affected by PD.

Then both of the groups were subjected to the SSR (sympathetic skin response), in which Specialized neurophysiology apparatus and patient preparation are essential for SSR recording. Considering that responses are affected by skin temperature and mental state, the patient ought to feel warm and at ease. The conventional method involves utilizing silver or silver chloride electrodes to obtain recordings from glabrous skin, which contains a high density of sweat glands. The palm and the forearm function as the active electrodes, while the dorsum of the foot or shin acts as the reference electrode. A grounding electrode is situated nearby. A comfortable temperature of approximately 32 °C is sustained, with distractions and noise minimized. Skin conductance inherently fluctuates; thus, to reduce extraneous stimulation, the environment is maintained tranquil and dim [13].

Factors were measured. The main parts of SSR are the delay and the amplitude of the deviation from the baseline. Latency is usually defined as the time between the stimulus artifact or the start of an idea and the first deflection or the peak of the first major phase. Most of the time, amplitude is measured from peak to peak between the first positive and negative deflections of a biphasic pattern. Two types of waveforms can be seen: the "P-type," which starts out deflecting up, and the "N-type," which starts out deflecting down. Since SSR gets used to things quickly, the stimuli are spread out just right. Changes must be constantly monitored to be sure they are caused by real reactions and not just computer tricks. When testing SSR, the lack of a response after enough stimulation is often seen as an abnormal result. Clinical neurophysiology books show how responses usually look and how often they happen [14].

2.2. Ethical approval

The study was approved by the scientific committee "Institutional Review Board" (IRB) at Baghdad Medical College, consented to by the Al-Rusafa Health Directorate Ethical Committee (No. 20241023 on Nov 22, 2024).

3. Results

Table 1 presents the demographic characteristics and disease duration among participants in the study, comparing individuals with PD to a control group. Both groups comprised 401 individuals. The mean age of the PD group was slightly higher than that of the control group (60.4 ± 6.7 years vs. 57.7 ± 5.7 years), with a marginally significant difference ($p = 0.050$). Regarding sex distribution, males constituted 57.5% of the PD group and 52.5% of the control group, while females represented 42.5% and 47.5%, respectively; however, this difference was not statistically significant ($p = 0.647$). The mean duration of Parkinson's disease among affected individuals was 5.2 ± 2.8 years.

Table 2 summarizes the clinical features related to autonomic dysfunction observed among the PD patients included in the study (N = 401). The most prevalent symptom was constipation, reported in 87.5% of patients, followed by impotence (80.0%) and dysphagia (75.0%). Other common manifestations included excessive sweating (65.0%), postural hypotension (62.5%), and intolerance to heat and cold (57.5%). Less frequently reported symptoms were dizziness (52.5%) and sphincter dysfunction (25.0%).

Table 1. Description of study demographics and duration of PD.

Characteristic	Control, N = 40[1]	PD, N = 40[1]	P-value[2]
Age (year)	57.7 ± 5.7	60.4 ± 6.7	0.050
Sex			
Male	21 (52.5%)	23 (57.5%)	0.647
Female	19 (47.5%)	17 (42.5%)	
Duration (year)	—	5.2 ± 2.8	

[1]Mean ± SD
[2]Welch Two Sample t-test

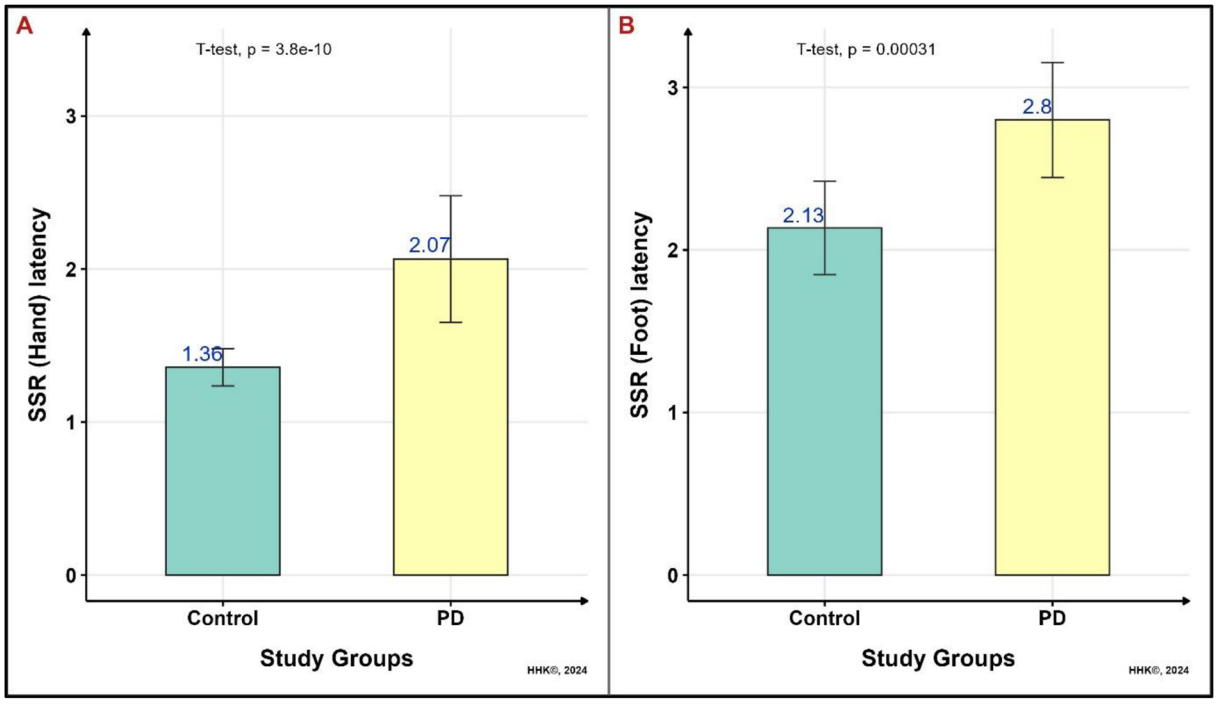

Fig. 1. Error plot showing the mean SSR latency in both study groups.

Table 2. Description of the clinical features of PD patients.

Characteristic	N = 40[1]
constipation	35 (87.5%)
Impotence	32 (80.0%)
Dysphagia	30 (75.0%)
Sweating	26 (65.0%)
Postural hypotension	25 (62.5%)
Heat and cold intolerance	23 (57.5%)
Sphincter dysfunction	10 (25.0%)
Dizziness	21 (52.5%)

[1]n (%)

Table 3. Description of the sympathetic skin response latency and amplitude in both study groups.

Characteristic	Control, N = 40[1]	PD, N = 40[1]	P-value[2]
SSR (Hand) latency	1.36 ± 0.12	2.07 ± 0.41	**<0.001**
SSR (Hand) amplitude	0.75 ± 0.24	0.28 ± 0.11	**<0.001**
SSR (Foot) latency	2.14 ± 0.29	2.80 ± 0.35	**<0.001**
SSR (Foot) amplitude	0.36 ± 0.17	0.13 ± 0.12	**<0.001**

[1]Mean ± SD
[2]Welch Two Sample t-test

Table 3 details the comparison of sympathetic skin response (SSR) parameters—latency and amplitude—in both the PD and control groups. The results demonstrate a significant delay in SSR latency and a marked reduction in amplitude among PD patients. For the hand, SSR latency was significantly prolonged in the PD group (2.07 ± 0.41 seconds) compared to controls (1.36 ± 0.12 seconds), while the corresponding amplitude was significantly lower (0.28 ± 0.11 mV vs. 0.75 ± 0.24 mV; $p < 0.001$ for both). Similarly, in the foot, SSR latency was increased in PD patients (2.80 ± 0.35 seconds) compared to controls (2.14 ± 0.29 seconds), and the amplitude was again significantly diminished (0.13 ± 0.12 mV in PD vs. 0.36 ± 0.17 mV in controls; $p < 0.001$ for both comparisons).

4. Discussion

Our study demonstrated a significant alteration in sympathetic autonomic function among Iraqi patients with PD, as evidenced by prolonged latencies and reduced amplitudes in SSR recordings. These findings align with several previous studies that documented similar abnormalities in PD patients using SSR as a diagnostic modality [15, 16].

The observed prolongation in SSR latency and attenuation of amplitude among our patients indicate a dysfunction in both central and peripheral sympathetic pathways. Previous pathological studies have shown that alpha-synuclein aggregates—the hallmark of PD—are not limited to the substantia nigra but are also found in the intermediolateral cell columns of the spinal cord, hypothalamus, and sympathetic ganglia, all of which contribute to autonomic imbalance [17]. The study reveals significant differences in sympathetic skin response (SSR) parameters between patients with Parkinson's disease and healthy controls. Both latency and amplitude showed statistically significant abnormalities.

Specifically, PD patients exhibited prolonged SSR latency and reduced amplitude in both the hand and foot, reflecting peripheral autonomic dysfunction, particularly in postganglionic sympathetic fibers.

Compared to a large Chinese cohort where 91.8% of PD patients exhibited signs of dysautonomia, our findings support the global prevalence of autonomic dysfunction in PD but also reveal certain regional patterns. For instance, constipation and impotence were highly prevalent in our cohort, potentially due to cultural, dietary, or environmental factors affecting autonomic tone in the Iraqi population [7]. Other studies, such as Ma et al. have suggested that SSR abnormalities are more pronounced in advanced PD stages. Our results concur with this, as the mean disease duration was over 5 years, likely correlating with progressive autonomic deterioration [13].

Our findings are supported by Wang et al., who documented significantly delayed SSR latencies and diminished amplitudes for the upper and lower limbs of patients with Parkinson's, correlating these abnormalities with disease severity and subtype [18]. Similarly, a more recent study by Xie et al. highlighted that SSR testing, especially foot amplitudes, offers diagnostic value in distinguishing PD from multiple system atrophy, reinforcing the significance of autonomic assessment in early-stage PD [19].

Ke et al. also confirmed that SSR abnormalities—both prolonged latencies and decreased amplitudes—are common in advanced PD and serve as reliable predictors of autonomic dysfunction [20]. This aligns with the foundational findings by Hirashima and Yokota, who demonstrated that abnormal SSRs are not significantly influenced by levodopa treatment, suggesting that SSR reflects intrinsic degeneration rather than treatment effects [21].

In addition, Papadopoulou et al. provided further confirmation by demonstrating that prolonged SSR latency and reduced amplitude are consistent biomarkers not only in PD but also in other neurodegenerative syndromes with autonomic involvement, indicating their broader diagnostic utility [22].

Collectively, these findings corroborate the strong diagnostic relevance of SSR testing in PD. The consistent pattern of prolonged latency and diminished amplitude points to sympathetic dysfunction, which may occur independently of motor symptom severity. This supports incorporating SSR evaluations in comprehensive autonomic assessments for PD patients.

5. Conclusion

The current study highlights a significant impairment in sympathetic autonomic function among Iraqi patients with Parkinson's disease, as detected through prolonged latencies and diminished amplitudes in SSR measurements. These neurophysiological alterations suggest that autonomic dysfunction is a prevalent yet often overlooked component of PD in this population. Early identification of such abnormalities using SSR may serve as a valuable tool in the clinical assessment and management of PD-related non-motor symptoms. Further large-scale and longitudinal studies are warranted to validate these findings and to explore their prognostic implications in disease progression and treatment responsiveness.

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Conflict of interest

No conflict of interest.

Ethical approval

This study was conducted with approval from the Ghazi Al-Hariri Surgical Teaching Hospital Ethics Committee. All participants provided informed consent. We confirm this manuscript is original, not under consideration elsewhere, and that all authors have approved its submission.

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