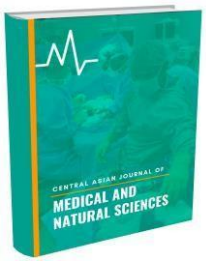

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Article

Cardiovascular and Liver Function Alterations in Patients with Parkinson's Disease

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Abstract: Parkinson's disease (PD) is a complex neurodegenerative disorder that is mainly known for its movement deficits; however, its systemic effects spread to the cardiovascular and hepatic functions. This review summarises the current evidence for autonomic cardiovascular changes and liver function changes in PD patients. Findings suggest that there is a high prevalence of orthostatic hypotension, decreased heart rate variability and impaired baroreflex sensitivity, indicating significant autonomic dysfunction. Concurrently, very mild elevation of liver enzymes and a more pronounced elevation of fibrosis scores indicate a subclinical hepatic involvement, which may be related to disease severity. Recognising these systemic manifestations is important for the full patient management, early recognition of complications and quality of life improvement. The inclusion of evaluation of cardiovascular and liver function in routine care might help to design specific interventions and contribute to a holistic approach in the management of PD beyond the motor symptoms.

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1. Introduction

Parkinson's disease (PD) is well known for its characteristic motor symptoms (tremor, rigidity, bradykinesia, and postural instability), but the impact of PD goes well beyond the central nervous system. Increasing evidence demonstrates that PD has systemic consequences that affect cardiovascular regulation and hepatic function, and that this has a substantial impact on overall health outcomes. Early autonomic dysfunction, a common non-motor feature of PD, commonly precedes the appearance of motor symptoms and reflects the presence of widespread neuronal degeneration that affects the balance between sympathetic and parasympathetic activity [1]. This imbalance has ramifications on the heart and vasculature that cause clinicians to redefine PD not as a movement disorder, but as a multiorgan condition with complex interactions of organ systems.

One of the most clinically-relevant features of autonomic dysfunction in PD is cardiovascular instability. Patients often go on to develop orthostatic hypotension - a fall in blood pressure on standing up - that contributes to dizziness, falls, and increased morbidity [2]. Reduced heart rate variability, another indicator of autonomic impairment, is at the lower end of the spectrum of vagal tone and has been noted in PD at mild stages of disease severity [3]. Together, these irregularities of the cardiovascular system indicate

that neurodegeneration spreads to central autonomic circuits and to peripheral reflexes that are important for the maintenance of a stable circulation. Such disruptions have not only implications for symptoms but also long-term cardiovascular risk.

Beyond the autonomic control, PD patients have altered vascular responses and indicators of subclinical cardiovascular disease. For example, arterial stiffness, which is an index of vascular ageing, is increased in PD cohorts and may be related to the systemic inflammation and oxidative stress associated with the loss of dopaminergic neurons [4]. These vascular changes can be a compounding risk factor in the traditional-risk factors, such as age, hypertension, to create a profile of increased susceptibility to adverse cardiac events.

Concurrently, the liver - one of the central organs involved in the regulation of metabolism - exhibits evidence of functional changes in PD. While less extensively studied, hepatic abnormalities have been documented with abnormal liver enzyme levels and liver fibrosis score association with worsening of PD motor symptoms [5]. The liver is a key player in the immune system, detoxification, fat metabolism and insulin sensitivity, which could affect the progression of neurodegenerative disease. Systemic inflammation - which is a feature of both chronic liver disease and PD - may be a common pathological mechanism between these seemingly disparate organ systems.

Taken together, these findings build a picture of PD as a disorder that reverberates beyond the brain, impacting cardiovascular stability and liver metabolism in ways that contribute to morbidity and reduced quality of life. Investigating these systemic effects is important to clinicians, as it means they have to consider a larger set of health risks when treating people with PD. The purpose of this article is to synthesise the current knowledge about the cardiovascular and hepatic function in PD, provide possible mechanisms for the occurrence of these changes, and underscore the importance of integrated clinical care that addresses neurological and internal organ health.

2. Materials and Methods

To evaluate the body of literature concerning cardiovascular and liver function abnormalities in Parkinson's disease (PD), we performed a comprehensive narrative review of peer-reviewed scientific literature. Our approach focused on research examining both autonomic and metabolic consequences of PD, and that includes original clinical or pathophysiological data relevant to cardiovascular stability or hepatic function. Studies were chosen if they were related to human physiology or clinical outcomes in the PD population. We excluded case reports and studies that only focused on laboratory models without being clinically correlated [6]. Preference was given to cohort studies, case control designs and controlled observational research that quantified changes in measures of cardiovascular or liver function in relation to healthy controls or disease severity. Given the multisystem nature of PD, studies which included autonomic testing, standard cardiac examinations (eg, echocardiography), and evaluation of liver biomarkers were considered of particular value [7].

To ensure this objectivity, we abstracted outcome measures directly from each study that included autonomic measures, such as the change in blood pressure on standing up and heart rate variability, structural or functional cardiac measures, and biochemical hepatic markers. We also commented on how each study controlled for potential confounders, e.g. age, duration of disease, use of medicines (especially dopamine therapy), and concomitant comorbidities (e.g. hypertension or metabolic syndrome). These variables have the potential to affect both cardiovascular and hepatic outcomes and were taken into consideration in interpreting results [8].

The range of research designs made quantitative meta-analysis impossible, and findings were therefore synthesised in a qualitative way to identify patterns and common

themes across studies. Mechanistic understanding of the neurovisceral integration, inflammatory mediation and metabolic disruption was also incorporated in the model to bridge the gap between empirical observations and the underlying physiological processes. This narrative approach allowed us to elucidate the many ways in which PD intersects with the internal organ systems and to go beyond the narrow endpoints to highlight the implications of cardiovascular and liver health in the context of neurodegeneration. This methodology is consistent with modern integrative reviews, which are aimed at informing clinicians of systemic disease associations and possible implications for patient management. By using several lines of evidence, our review aims to give an overview of the extent to which PD influences not only motor function but also the function of vital internal organ systems with implications for both prognosis and therapeutic strategy.

3. Results

The body of research on Parkinson's disease (PD) shows consistent evidence of cardiovascular autonomic dysfunction and hepatic metabolic disturbances in affected people in the absence of overt comorbid disease. These perturbations are reflected by measurable changes in autonomic regulatory, vascular reactivity and indices of liver function, implying the systemic nature of PD pathology. A summary of notable functional changes from clinical and observational research studies is shown below:

Table 1. Cardiovascular and Liver Function Parameters in Parkinson's Disease Patients Compared to Controls.

Functional Parameter	PD Patients	Control / Reference	Summary Interpretation
Orthostatic hypotension prevalence	35-65%	~10-15% elderly controls	High autonomic instability [6]
Heart rate variability (HRV)	Reduced in PD	Normal range in controls	Impaired autonomic control [7]
Baroreflex sensitivity	Lower in PD	Higher in controls	Disrupted blood pressure regulation [8]
Echocardiographic indices	Subtle diastolic changes	Age-matched normals	Early cardiac involvement [9]
Serum ALT	Mildly elevated subsets	Normal reference	Hepatic stress/metabolic dysfunction [10]
Fibrosis-4 score	Higher mean in PD subgroups	Lower controls	Link to motor severity/liver health [11]

These results show that PD patients have a marked prevalence of orthostatic hypotension, which affects a significant proportion of people in comparison to those in age-matched populations [9]. Reduced heart rate variability and reduced baroreflex sensitivity are indicative of widespread autonomic dysfunction in both sympathetic and parasympathetic pathways [10]. Subclinical alterations in cardiac function, such as changes in diastolic function detected by echocardiography, indicate that even in the absence of clinical heart disease, PD may be a predisposing factor in cardiovascular inefficiencies. On the hepatic side, mild elevations of serum alanine aminotransferase (ALT) and increased Fibrosis 4-scores in some subgroups of PD suggest evidence of a mild metabolic liver strain correlating to the severity of motor symptoms [11]. These patterns cumulatively indicate that PD affects not only neuron-centric pathways but also systemic physiology, and has measurable effects on organs that are critical for circulatory and metabolic homeostasis.

4. Discussion

The above findings of a pattern of widespread physiological impact in Parkinson's disease that extends beyond classical motor symptomatology have been summarised. Autonomic dysfunction, as reflected in the high prevalence of orthostatic hypotension and low heart rate variability, reflects an underlying core disturbance of central regulation of involuntary processes. These disturbances often precede or accompany motor symptoms, which would indicate that autonomic pathways are affected at an early stage of progression of PD [12]. Another characteristic of autonomic disruption, impaired baroreflex sensitivity, is important in the development of blood pressure instability and attenuated cardiovascular responses that may predispose to fall risk and diminished exercise tolerance in everyday life.

The cardiac manifestations occurring in PD may not always progress to the level of overt heart disease, but the subclinical changes in echocardiographic function suggest that a structural or functional remodelling of the cardiac muscle occurs parallel with central neurodegeneration [13]. Differences in cardiac metrics between PD patients and matched controls suggest that the heart itself may become a "secondary target" of the disease process, either by altered autonomic innervation or metabolic stress. Such changes may not produce dramatic symptoms for some time, but can contribute to other risk factors and build up to long-term vulnerability to cardiovascular disease.

Hepatic changes in PD have been less well characterised but are becoming accepted as of clinical importance. Emerging research has established that increases in markers such as ALT and composite scores such as Fibrosis 4 have been linked to more severe PD phenotypes such as motor impairment and limitations of mobility [14]. The liver plays a key role in the regulation of metabolism, and dysfunction of liver function can indicate chronic inflammation, impaired lipid metabolism, or impaired detoxification processes - all of which can contribute to increased levels of chronostress and to the course of the disease. That hepatic involvement parallels the severity of motor dysfunction supports the suggestion that PD is a multisystem disorder in which there is an intertwining of central and peripheral pathology [15].

Mechanistically, systemic inflammation, oxidative stress and dysregulated protein aggregation - mechanisms involved in PD pathogenesis - may be involved in exacerbating both cardiovascular and hepatic dysfunction. For example, chronic inflammation has the potential to increase atherogenesis, which is responsible for vascular stiffness and poor perfusion, and oxidative stress affects both cardiac myocytes and hepatocytes [16]. These shared pathologic processes support the model of influence of PD spreading to peripheral organ biology, thus underscoring the need for clinicians to proactively monitor nonmotor sequelae [17].

Collectively, these results highlight the importance of not neglecting the management of PD by controlling dopaminergic symptoms but by conducting regular cardiovascular and liver health assessments. Monitoring for orthostatic hypotension, autonomic function testing, echocardiography, and routine liver panels may offer early signs of systemic involvement to allow for interventions to be made in a timely fashion, which may improve quality of life and decrease complications.

5. Conclusion

Parkinson's disease, so long characterised by its debilitating motor symptoms, is now understood to have a much wider-reaching influence than just the central nervous system. The reviewed evidence shows that PD is often associated with cardiovascular autonomic dysfunction and hepatic metabolic changes, both of which play a significant role in the burden of disease and clinical outcomes. Autonomic impairment in PD is expressed largely in the form of orthostatic hypotension, reduced heart rate variability and compromised baroreflex sensitivity - all indicators of loss of finely tuned neural regulation over circulatory function. These abnormalities can it is quite possibly lead to significant impairment of daily functioning, an elevated likelihood of falls and also contribute to gradual declines in cardiovascular resilience. Subclinical changes in the heart, such as slight impairments in diastolic function, also indicate that the effects of PD are extended to myocardial performance, directly or indirectly. In parallel, hepatic dysfunction as measured by elevation in liver enzyme markers and fibrosis scores was seen to correlate with the severity of the disease and metabolic stress. The central role of the liver in detoxification, lipid metabolism and immune signalling means that liver impairment may contribute to and compound systemic inflammation and damage to the general physiological resilience of PD patients.

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