Ultrasound for the Assessment of Muscle Architecture in Parkinson's disease: A Scoping Review

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Summary

Parkinson's disease (PD) affects muscle function. A scoping review identified 17 articles on ultrasound (US) assessment of muscle architecture in PD. This revealed the research is mostly observational, methodologically diverse, and potentially underpowered. The findings indicated minimal differences in muscle architecture at rest but impaired responsiveness during muscle contractions in PD compared to healthy controls. Muscle architecture was associated with PD clinical symptoms and diminished functional performance, while exercise enhanced muscle thickness. Although US is promising, more robust and longitudinal studies are required to assess PD's impact on muscle architecture and functionality, as well as the therapeutic benefits of exercise.

Introduction

Parkinson's disease (PD) is an idiopathic disease of the nervous system characterised by tremor, bradykinesia and rigidity. As a movement disorder, PD affects muscle function which is influenced by muscle architecture [1]. While ultrasound (US) is a well-established tool for assessing the architectural characteristics of muscle [2], its application in PD remains unexplored. This scoping review maps existing literature on US assessment of muscle architecture in PD.

Methods

A review was conducted in line with the PRISMA-ScR framework [3]. A structured literature search was conducted in multiple databases (Scopus, PubMed, Web of Science, etc.) for studies on US assessment of skeletal muscle architecture in PD. Eligibility required original research written in the English language, assessing peripheral skeletal muscle architecture using US in PD. Seventeen studies were included, and data were extracted on study and participant characteristics, US methods (i.e., measurement methodology and approach to measurement and reporting), muscle architectural variables, clinimetric properties and study outcomes. Descriptive and numerical analyses followed.

Results and Discussion

Seventeen studies (2016–2024) with 797 participants (PD; n = 513, controls; n = 284) varied in design (i.e., study and participant characteristics), US measurement methodology (i.e., muscle of interest, reported muscle architectural variable) and approach to measurement and reporting (i.e., US system and acquisition procedure). Majority of studies were observational (n = 16) and many were limited by small sample sizes (PD: range 8 to 120) that may have lacked sufficient statistical power, all employed convenience sampling. PD participants varied in disease severity, stage, phenotype, and often presented with additional PD-specific conditions (i.e., sarcopenia, camptocormia, dysphagia). Muscle thickness, fascicle length, pennation angle, and cross-sectional area were most assessed, with vastus lateralis, gastrocnemius and rectus femoris being the most common targets. US methods differed significantly (i.e., US brand, probe type, frequency, measurement depth, frame rate). Muscle architectural assessments occurred mostly at rest. Most studies analysed muscle architecture using manual analysis. Only four muscles assessed clinimetric properties of the muscle measurement technique. US seems to be a reliable method; however, more reliability studies need to be conducted.

At rest, muscle architecture differences between PD patients and controls were minimal, though isolated variations (i.e., sex, PD subtype, conditions unique to PD) were noted. During contractions, PD patients exhibited impaired responsiveness, highlighting neuromuscular dysfunction. Bilateral differences within PD were minor but varied with progression and conditions like sarcopenia or dysphagia. Reduced muscle thickness correlated with greater PD clinical symptoms (i.e., rigidity and bradykinesia) and poor functional performance (e.g., balance, timed up and go). The only included exercise intervention study, demonstrated Pilates-based exercise may have therapeutic benefits through increased muscle thickness.

Conclusions

Research on muscle architecture in PD using US is limited and the studies reviewed had notable methodological limitations. While US shows promise, the methods varied, underscoring the need for standardised US protocols. Muscle architecture is evidently altered in PD, but underpowered studies limit definitive conclusions. More rigorous research, especially longitudinal and reliability studies, is needed to explore muscle architecture in PD and its influence on performance (e.g., walking gait and turning) and the effects of exercise on muscle architecture, functionality and strength.

References

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