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Short Commentary

Rising Significance of Dynapenic Abdominal Obesity in Chronic Kidney Disease: Insights from a Chinese Cohort

Zhipeng Chen*, Feng Zhao, Siqi Li and Ling Zhu

School of Medical Technology, Jiangsu College of Nursing, Huai'an City, Jiangsu Province, China

Abstract

The aim of this commentary is to highlight the divergent impact of Dynapenic Abdominal Obesity (D/AO) on Chronic Kidney Disease (CKD) risk in middle-aged versus older adults. Based on a nationwide prospective cohort study (CHARLS, N = 5,932), D/AO conferred a 2.4-fold higher CKD risk in middle-aged adults (OR 2.40; 95% CI 1.14–4.59; p=0.01), while no significant association was observed in older populations (OR 1.56; 95% CI 0.96–2.46; p=0.06). These findings underscore the urgent need for age-stratified prevention strategies targeting muscle strength and visceral adiposity.

Keywords: Abdominal obesity; Age disparity; China Health and Retirement longitudinal study; Chronic kidney disease; Dynamic

Introduction

Chronic Kidney Disease (CKD) affects 8.2% of Chinese adults, with aging populations bearing the greatest burden [1,2]. Dynapenic Abdominal Obesity (D/AO)—a confluence of low muscle strength (dynapenia) and abdominal obesity—has emerged as a novel risk factor for CKD, yet its age-specific implications remain unexplored [3]. This commentary discusses key findings from our prospective cohort study investigating D/AO and CKD risk in Chinese adults aged ≥45 years.

Key Findings

Overall cohort

D/AO was associated with 57% higher CKD incidence after multivariable adjustment (OR 1.57; 95% CI 1.03-2.35).

***Corresponding author:** Zhipeng Chen, School of Medical Technology, Jiangsu College of Nursing, No.9 Keji Road, Qingjiangpu District, Huai'an City, Jiangsu Province, China, Tel: +86 19852561923; E-mail: chen.zhip@foxmail.com

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Age disparity

Middle-aged adults (45-59 years): D/AO elevated CKD risk by 140% (OR 2.40; 95% CI 1.14-4.59; p = 0.01). Older adults (≥60 years): No significant association (OR 1.56; 95% CI 0.96-2.46; p = 0.06).

Screening utility

Handgrip strength (<28 kg men, <18 kg women) and waist circumference (≥90 cm men, ≥85 cm women) offered a low-cost tool for identifying high-risk individuals.

Interpretation and Implications

Biological mechanisms

The synergy between dynapenia and visceral adiposity may accelerate renal decline through:

- Inflammatory crosstalk: Visceral fat elevates pro-fibrotic cytokines (e.g., IL-6), while muscle atrophy reduces nephroprotective myokines (e.g., irisin) [4,5]
- Metabolic dysregulation: Ectopic lipid deposition promotes insulin resistance and podocyte injury [6]

Clinical practice

- Middle-aged adults: D/AO screening via grip strength and waist circumference should be integrated into routine health checks. Early interventions (e.g., resistance training, dietary management) may mitigate CKD risk [7]
- Older adults: Competing risks (e.g., cardiovascular mortality) may mask D/AO's impact; holistic comorbidity management is prioritized [8].

Public health perspective

- Prevention focus: Midlife interventions targeting D/AO could reduce CKD burden in aging societies
- Policy action: National health programs should incorporate musculoskeletal-metabolic health metrics into CKD surveillance

Unresolved questions

- Do mitochondrial dysfunction or gut-kidney axis disruptions mediate the age-specific effects of D/AO?
- Can resistance training combined with nutritional interventions delay CKD onset in high-risk middle-aged adults?
- Why does D/AO's renal impact attenuate in older populations? Is this due to survivorship bias or physiological adaptations?

Conclusion

D/AO is a modifiable CKD risk factor with age-stratified clinical relevance. Public health policies must prioritize midlife interventions

while adapting geriatric care to address multifactorial risks. Our findings advocate for integrating musculoskeletal and metabolic health into CKD prevention frameworks—a step toward precision public health.

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Conflicts of Interest

The authors declare no conflicts of interest.

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