INVITED REVIEW ARTICLE

BEYOND THE BONE: RETHINKING SPINE SURGERY THROUGH THE LENS OF SARCOPENIA

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Abstract

Historically, spinal surgery has prioritized bone integrity, often overlooking the biomechanical significance of the paraspinal muscles. Sarcopenia, an age-related decline in muscle mass, strength, and function, has emerged as a critical modifier of spinal pathology and surgical outcomes. Sarcopenia is characterized by progressive myosteatosis, particularly in the multifidus and erector spinae, which contributes to spinal imbalance, chronic pain, and functional decline. Fatty infiltration, a key marker of muscle degeneration, is strongly associated with frailty, reduced mobility, and impaired quality of life.

Spinal musculature plays an essential role in maintaining posture, segmental control, and load-bearing capacity. Muscle deterioration exacerbates spinal deformities and degrades surgical outcomes, particularly during procedures involving extensive dissection or fusion. Even minimally invasive techniques may result in scarring and atrophy, increasing the risk of complications, such as nonunion, adjacent segment disease, or implant failure.

As spinal sarcopenia becomes increasingly prevalent in aging populations, surgical strategies must evolve accordingly. Minimally invasive motion-preserving approaches, along with targeted rehabilitation, are essential for preserving muscle function. Radiological assessments should routinely include muscle density and bone metrics, and sarcopenia should be integrated into surgical planning as well as risk stratification.

Recognizing the spinal musculature as a dynamic, modifiable structure—rather than a passive one—is imperative. Addressing sarcopenia through a comprehensive preoperative evaluation, intraoperative techniques, and postoperative rehabilitation may significantly enhance patient outcomes and long-term spinal health.

Keywords: Spine; Aging; Sarcopenia.

INTRODUCTION

In the last part of the past century, spine surgeons' primary focus was traditionally centered on bone integrity. Their training, which was rooted in anatomical dissections and static radiological imaging, did not emphasize the dynamic nature of human movement or the biomechanical role of soft tissues, particularly the spinal musculature. Consequently, the paraspinal muscles that stabilize the spine have often been compromised during spinal surgical procedures, leading to unintended long-term consequences.

Despite showing optimal radiological outcomes postoperatively, many patients continued to experience diminished quality of life. They were often discharged with a simple set of illustrated exercises and then referred for physical therapy and rehabilitation. Through the subsequent efforts of physical therapy researchers, the crucial role of muscle function, especially as it relates to aging and spinal pathology, has been brought to the forefront.

This shift in understanding led to the conceptualization of sarcopenia, defined as the age-related loss of muscle mass, strength, and function. The etiology of sarcopenia remains multifactorial and has not yet been completely elucidated. A confluence of determinants, including suboptimal nutritional status, sedentary behavior, and the presence of age-associated chronic illnesses,contributes to the progressive decline in skeletal muscle mass and function observed with advancing age (1).

Several interrelated pathophysiological mechanisms have been implicated in the onset and progression of this degenerative condition, including chronic low-grade inflammation, mitochondrial dysfunction, oxidative stress, resistance to insulin and anabolic stimuli, and alterations in endocrine function (2). A diagram of the pathogenesis of sarcopenia during aging is shown in Figure 1. Different physiological and pathological factors contributing to muscle protein breakdown and ultimately resulting in sarcopenia should also be considered (Figure 2) (3).

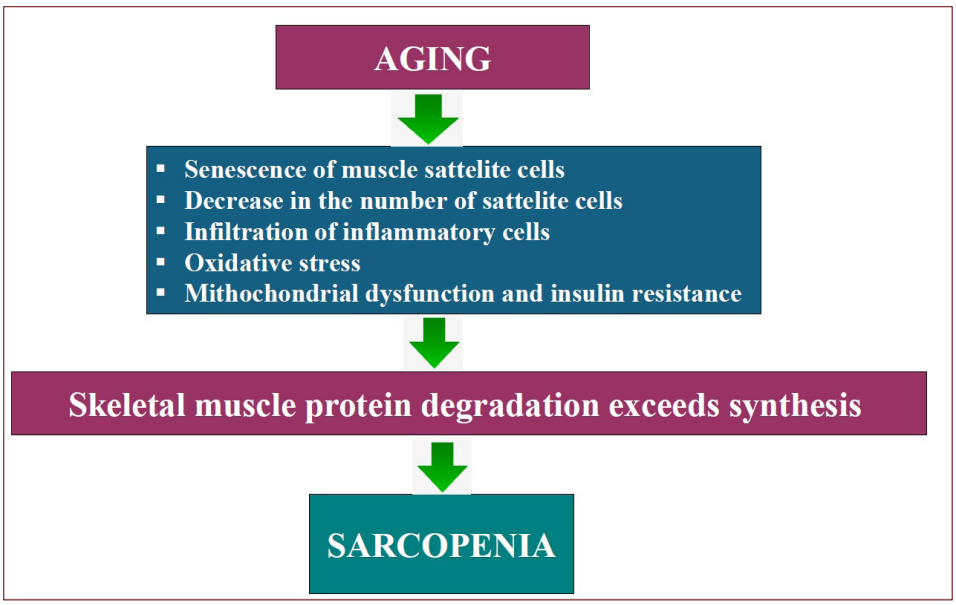

Figure 1. Schematic diagram of the pathogenesis of sarcopenia during the aging process.

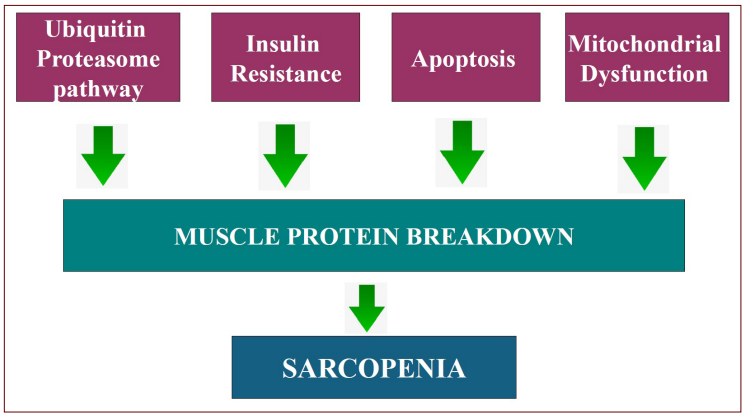

Figure 2. Various physiological and pathological factors contribute to muscle protein breakdown, ultimately resulting in sarcopenia.

Spinal Myosteatosis

Muscle mass peaks in early adulthood and gradually declines after 50 years of age. Age-related atrophy is associated with structural alterations, including the replacement of muscle tissue with adipose tissue, increased intramuscular fat and connective tissue, muscle fiber-type transformation, and localized inflammation (4). In recent decades, research on sarcopenia has emphasized that the paraspinal muscle volume and strength are significantly reduced in affected individuals, directly affecting spinal function (1).

Myosteatosis, a fatty degeneration characteristic of spinal sarcopenia, has been increasingly observed in older, obese (5), and postmenopausal women (6), with a progressive increase in fat infiltration from the cranial to the caudal spine (7). The multifidus muscle showed the most significant decline (8). Definitions of sarcopenia have become closely associated with frailty syndrome, which is typically characterized by unintentional weight loss, self-reported exhaustion, muscle weakness (e.g., reduced grip strength), slow walking speed, and reduced physical activity.

To mitigate back pain, patients often reduce the movement of painful areas, which leads to disuse and subsequent muscle atrophy. This promotes anterior trunk tilt, increased intramuscular pressure, and ischemic progression. The resulting ischemic pain leads to further disuse, creating a vicious cycle in which pain leads to increased fat infiltration, postural imbalance, and exacerbated pain (9).

Fat infiltration is a widely used marker of muscle degeneration (10). Objective quantification systems such as those developed by Kjaer, Goutallier, and Kalichman provide grading scales for fatty infiltration, primarily using CT and MRI, the current gold standards for evaluating the multifidus, erector spinae, and psoas muscles (10,11,12).

Spinal Dynamics

The erector spinae muscles are arranged vertically like the ropes of a mast, maintain both sagittal and coronal balance by resisting external trunk loads, and preserve upright posture. They function as global tension bands that regulate spinal movement and bear axial pressure on the lumbar spine (13).

The multifidus is the most medially located deep paraspinal muscle with the broadest muscle attachment across the spinal segments. Vertical and transverse fiber orientations play a key role in proprioception and the fine-tuning of segmental mobility. The multifidus spans two to five motion segments, with the shortest fascicles attaching deeply to the spinous processes and helping distribute stress during trunk stabilization and rotation (14). A moderate correlation has been reported between the multifidus cross-sectional area and the global spinal or spinopelvic alignment.

The psoas muscle, particularly its intermediate and superficial fascicles, is the only paraspinal muscle that connects the spine directly to the lower limbs. It contributes significantly to postural stability by increasing lumbar stiffness via axial compression and supports ambulation through its relatively short moment arms and equal-length fascicles (15).

Muscle density is as critical as bone density in determining spinal endurance. Paraspinal muscle quality is closely linked to lumbar vertebral bone mineral density (BMD), aligning with the "musculoskeletal unit" theory. This theory suggests that reducing paraspinal muscle fatty infiltration—for example through targeted exercise—could lead to improvements in lumbar BMD, possibly revealing a reciprocal relationship between sarcopenia and osteopenia (6).

Well-conditioned spinal musculature can effectively compensate for minor sagittal or coronal imbalances, resulting in fewer symptoms. Conversely, poor muscle quality often results in significant symptoms, even with moderate misalignment (16). Extensive muscle degeneration is implicated in flat back syndrome, and there is a linear correlation between fat infiltration and the severity of spinal curvature deformities (17). Spinal sagittal imbalance cannot be fully explained by the vertebral or disc height alone, and muscle quality is a significant determinant. Accordingly, lumbar muscle fat infiltration is recognized as an independent factor associated with reduced quality of life (18).

The detrimental effects of sarcopenia progress slowly and insidiously, unlike the acute muscle loss observed in malnutrition, sepsis, and cancer. Therefore, its clinical impact may require considerable time.

Surgical Implications

Painful spinal syndromes in elderly patients are often treated surgically when conservative treatment options are exhausted or ineffective. However, surgical spinal fusion techniques have long been known to damage paravertebral muscles. This is almost unavoidable in procedures involving osteotomies to correct sagittal or coronal imbalance (19). Even with minimally invasive techniques, muscle specimens often exhibit extensive scarring postoperatively (20).

Studies have demonstrated that paraspinal muscle volume diminishes following spinal surgery, including anterior stand-alone fusion (21). Furthermore, dysfunctional motion segments and mechanical instability were more pronounced after fusion than decompression-only procedures (22).

Surgical dissection and the resulting scarring contribute to long-term muscle dysfunction and complications such as nonunion (23), adjacent segment degeneration (24), and proximal junctional failure (25). These complications compromise patient satisfaction and quality of life, and may culminate in failed back surgery syndrome (26). Recent studies have associated increased fat infiltration in the lumbar paraspinal region with poor outcomes after surgery (27). Just as spinal implants in osteoporotic bone are more prone to failure, those placed in atrophic musculature are also at a higher risk of dislodgement, particularly in long-level fusions (28).

This necessitates a shift toward minimally invasive and motion-preserving hybrid dynamic techniques. As the prevalence of spinal sarcopenia increases in tandem with the aging population, preserving paraspinal muscle integrity during surgery is paramount. Similarly, mitigating intramuscular fat accumulation in older adults should be prioritized.

Postoperative rehabilitation should include structured physical activity, particularly resistance and multicomponent exercise training targeting the paravertebral muscles both selectively and unilaterally, based on the patient's functional deficits (29). Additionally, it is essential to reestablish the muscle balance between the trunk flexors and extensors (e.g., rectus abdominis vs. erector spinae, external/internal obliques vs. multifidus, and

erector spinae vs. psoas) through core stabilization programs (30).

CONCLUSION

Given the significance of paraspinal muscle function, muscle atrophy assessments should become standard components of radiology reports along with bone density measurements. Spine surgeons must be cognizant of these parameters during preoperative planning.

A critical gap is the omission of muscle density as a modifier in surgical decision-making algorithms. Further studies investigating the prognostic value of paraspinal muscle atrophy on postoperative outcomes are urgently needed. Such insights can support surgical decision-making and improve treatment strategies.

Only through such integrative approaches can the spinal musculature be preserved—not as a passive structure, but as a dynamic, functional system essential to spinal biomechanics and long-term patient outcomes.

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