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Review Article

Pathophysiological Link Between Muscle Atrophy and Hypertension in Spinal Cord Injury Patients

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ABSTRACT

Spinal cord injury (SCI) is a devastating neurological condition associated not only with motor and sensory impairment but also with profound long-term systemic complications. Among these, rapid skeletal muscle atrophy and blood pressure dysregulation represent two interlinked and clinically significant sequelae that substantially increase cardiometabolic risk and mortality in individuals with SCI. Following injury, denervation, mechanical unloading, and altered metabolic signaling lead to accelerated loss of muscle mass, fiber-type transformation, fat infiltration, and endocrine dysfunction, often resulting in a sarcopenic-obesity phenotype. In parallel, disruption of sympathetic autonomic pathways produces complex blood pressure abnormalities, ranging from chronic hypotension and orthostatic intolerance to episodic autonomic dysreflexia and sustained hypertension.

Growing evidence supports a bidirectional relationship between muscle atrophy and hypertension in SCI. Loss of metabolically active muscle reduces insulin sensitivity, myokine secretion, and peripheral vascular capacity, thereby promoting inflammation, arterial stiffness, and elevated blood pressure. Conversely, chronic and episodic hypertension impairs skeletal muscle microcirculation and endothelial function, potentially exacerbating muscle wasting and limiting rehabilitation outcomes. These processes are further amplified by insulin resistance, oxidative stress, autonomic imbalance, and renal mechanisms, creating a vicious cycle of neuromuscular and cardiovascular deterioration.

This review synthesizes current evidence on the mechanistic links between skeletal muscle atrophy and hypertension following SCI, highlighting the central role of muscle as both a structural and endocrine organ in cardiovascular regulation. Understanding this integrated pathophysiology has important clinical implications, underscoring the need for muscle-centered, multidisciplinary rehabilitation strategies—including resistance training, neuromuscular electrical stimulation, nutritional optimization, and individualized blood pressure management—to mitigate long-term cardiovascular risk, improve functional independence, and enhance quality of life in people living with SCI.

Keywords: Hypertension, Muscle atrophy, Spinal cord injury

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INTRODUCTION

Spinal cord injury (SCI) is a catastrophic neurological condition that results in varying degrees of motor, sensory, and autonomic impairment below the level of injury. Beyond the immediate loss of motor function, one of the most significant long-term consequences of SCI is rapid and progressive skeletal muscle atrophy, driven by denervation, reduced mechanical loading, and altered metabolic signaling. Muscle mass can decline by nearly, 30–50% within the first few months after injury, leading to profound reductions in strength, physical function, and overall metabolic health. In parallel, individuals with SCI commonly develop abnormalities in cardiovascular regulation due to disruption of sympathetic pathways, resulting in autonomic dysreflexia, orthostatic hypotension, and chronic hypertension. Among these, neurogenic hypertension has emerged as a major contributor to secondary cardiovascular complications and remains a leading cause of morbidity and mortality in the SCI population. The combination of vascular dysregulation, impaired baroreflex sensitivity, inflammation, and oxidative stress further accelerates cardiac and vascular remodeling.

Growing evidence indicates a bidirectional relationship between skeletal muscle atrophy and hypertension in SCI. Loss of active muscle tissue significantly reduces metabolic rate, insulin sensitivity, and peripheral vascular capacity, promoting vascular stiffness and

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elevating blood pressure. Conversely, chronic hypertension worsens microvascular perfusion and endothelial function within skeletal muscle, potentially exacerbating muscle wasting. This creates a vicious clinical cycle, where muscle deterioration and blood pressure instability perpetuate each other, compounding disability and impairing rehabilitation outcomes.

Understanding the interplay between muscle biology, autonomic cardiovascular regulation, and rehabilitation strategies in SCI is essential for optimizing long-term care. Interventions such as functional electrical stimulation, resistance training, neuromodulation, and targeted pharmacotherapy have shown promising potential in mitigating muscle atrophy, while improving cardiovascular stability. However, comprehensive clinical guidelines and mechanistic clarity are still evolving. Therefore, exploring the integrated pathway linking skeletal muscle atrophy and hypertension in spinal cord injury is crucial to developing more effective, multidisciplinary rehabilitation protocols aimed at improving survival, functional independence, and quality of life for individuals living with SCI.[1]

Overview of SCI, Muscle Atrophy and Blood Pressure

Traumatic or non-traumatic spinal cord injury (SCI) results in varying degrees of motor paralysis and interruption of descending neural pathways. This loss of voluntary activation and neural input to skeletal muscle initiates rapid and profound muscle wasting below the level of injury. In the early post-injury period, muscle atrophy progresses quickly due to denervation, reduced mechanical loading, and altered metabolic signaling, leading to significant declines in muscle cross-sectional area and strength. Acute and subacute changes include a marked reduction in lean muscle mass along with progressive infiltration of intramuscular and visceral adipose tissue. These alterations evolve within weeks to months following injury and contribute to impaired glucose tolerance, dyslipidemia, insulin resistance, and overall increased cardiometabolic risk. Consequently, individuals with SCI are disproportionately predisposed to obesity, metabolic syndrome, and cardiovascular disease, despite reduced total body weight.[2]

Regulation of blood pressure in SCI is uniquely complex due to disruption of sympathetic autonomic pathways. Depending on the level, completeness, and chronicity of injury, individuals may present with a wide spectrum of cardiovascular instability. Those with high thoracic or cervical lesions often experience chronic hypotension and orthostatic intolerance, resulting from impaired sympathetic vasoconstriction and reduced cardiac output. Conversely, episodes of autonomic dysreflexia—a potentially life-threatening hypertensive crisis triggered by noxious stimuli below the level of injury—are common in chronic SCI above T6. These episodic surges in blood pressure may exceed 250 mmHg systolic and expose patients to increased risk of stroke, arrhythmias, and myocardial ischemia. In other cases, patients may develop conventional essential hypertension, particularly as they age or accumulate cardiometabolic risk factors associated with muscle-fat redistribution and physical inactivity.[3]

The combination of dysregulated skeletal muscle metabolism and blood pressure instability reflects a complex interaction between neuromuscular and autonomic dysfunction following SCI. Understanding these mechanisms is critical for developing targeted interventions to preserve muscle health, stabilize cardiovascular responses, and reduce long-term morbidity in this vulnerable population.

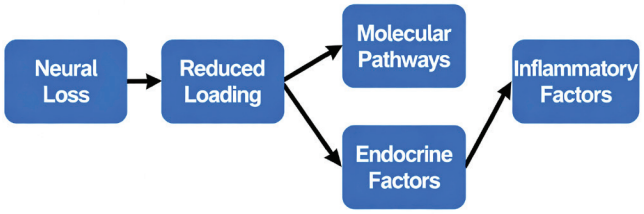

Figure 1: Mechanism of muscle atrophy after spinal cord injury.

Muscle Atrophy After SCI: Mechanisms

Following spinal cord injury, the immediate loss of descending motor drive and drastically reduced mechanical loading initiate a cascade of structural and metabolic alterations within skeletal muscle below the level of injury. In the absence of normal neural activation, paralyzed muscles rapidly undergo reduction in muscle fiber cross-sectional area and significant loss of contractile proteins. A well-characterized shift in muscle phenotype occurs, with transformation from fatigue-resistant oxidative type I fibers to fast-twitch glycolytic type IIx fibers, resulting in decreased endurance, increased fatigability, and impaired metabolic efficiency. Over time, these muscles experience substantial fat infiltration, including intramuscular and intermuscular adipose accumulation, contributing to metabolic dysfunction and systemic inflammation.[4]

At the molecular level, muscle atrophy after SCI is driven by activation of catabolic signaling pathways, most notably the ubiquitin–proteasome system (UPS) and the autophagy–lysosome pathway, which collectively mediate accelerated proteolysis and breakdown of myofibrillar proteins. Additionally, there is upregulation of negative regulators of muscle growth such as myostatin, which suppresses protein synthesis and satellite cell activation. The local inflammatory milieu, characterized by elevated cytokines including tumor necrosis factor-α (TNF-α) and interleukin-6 (IL-6), further promotes protein degradation, apoptosis, and mitochondrial dysfunction.[5,6] Figure 1 showing the basic mechanism of muscle atrophy after spinal cord injury.

Endocrine dysregulation contributes significantly to progressive muscle wasting in chronic SCI. Reduced levels of anabolic hormones such as testosterone, growth hormone, and insulin-like growth factor-1 (IGF-1), along with insulin resistance, impair muscle hypertrophy signaling and limit regeneration capacity. Concurrently, increased oxidative stress resulting from impaired mitochondrial metabolism leads to cellular damage and exacerbates inflammatory pathways, creating a feed-forward cycle that accelerates muscle degeneration. Collectively, these mechanisms result not only in rapid early atrophy but also in long-term failure of muscle recovery and regeneration in individuals living with chronic SCI.[7]

Hypertension Phenotypes in SCI

In high cervical or upper thoracic SCI, disruption of descending sympathetic pathways predisposes to autonomic dysreflexia, characterized by paroxysmal hypertensive episodes triggered by noxious stimuli below the lesion. In lower thoracic or lumbar lesions and long-standing SCI, traditional cardiovascular risk factors become prominent, and sustained hypertension is associated with aging, increased adiposity, physical inactivity, and metabolic syndrome.

Population-based data suggest that hypertension prevalence in people with SCI is substantial (around one-quarter in some cohorts) and relates inversely to measures of muscular fitness and directly to body mass index.[8]

Body Composition, Sarcopenic Obesity and Blood Pressure

SCI-induced muscle atrophy often coexists with increased visceral and ectopic fat, a pattern similar to sarcopenic obesity described in the general population. Reduced muscle mass lowers resting energy expenditure and favors positive energy balance, leading to central adiposity, which is strongly linked to hypertension through activation of the renin–angiotensin–aldosterone system, sympathetic overactivity, and sodium retention. In SCI cohorts, higher fat mass and lower lean mass are associated with impaired glucose tolerance, dyslipidemia and elevated blood pressure, suggesting a shared body-composition-mediated pathway from muscle atrophy to hypertension.[9]

Myokines, Adipokines and Vascular Dysfunction

Skeletal muscle is an endocrine organ that releases myokines such as irisin, IL-6 (transiently in exercise), brain-derived neurotrophic factor, and others that promote vascular health, insulin sensitivity, and anti-inflammatory effects. In chronic muscle atrophy and inactivity, there is reduced secretion of protective myokines and relative predominance of pro-inflammatory cytokines and myostatin, which favor endothelial dysfunction, arterial stiffness, and hypertension. Excess adipose tissue in sarcopenic obesity produces adipokines (leptin, resistin) and inflammatory mediators that further impair nitric oxide bioavailability and enhance vasoconstrictor responses, reinforcing the blood pressure rise.[10,11]

Inflammation, Oxidative Stress and Arterial Stiffness

SCI is associated with chronic low-grade systemic inflammation, driven by recurrent infections, tissue hypoperfusion, adiposity, and muscle catabolism. Elevated circulating TNF-α, IL-1β, and IL-6 activate NF-κB and JAK/STAT pathways in vascular cells, increasing expression of adhesion molecules, promoting oxidative stress, and reducing endothelial nitric oxide synthase activity, all of which contribute to vascular remodeling and increased peripheral resistance. Over time, these processes lead to increased arterial stiffness and left ventricular afterload, establishing a structural substrate for sustained hypertension in individuals with chronic SCI and pronounced muscle atrophy.[12,13]

Autonomic Imbalance and Sympathetic Mechanisms

Loss of supraspinal control in SCI alters autonomic balance, with reduced sympathetic outflow below the lesion and compensatory changes above it. In cervical and high thoracic injuries, reflex sympathetic surges in response to afferent stimuli below the lesion cause intense vasoconstriction and abrupt hypertension (autonomic dysreflexia), while impaired baroreflex buffering limits heart rate and vasodilator responses. Muscle atrophy and physical deconditioning also reduce the skeletal muscle pump and vagal tone, shifting cardiovascular autonomic regulation toward a relatively sympathetic-dominant state during rest and activity, which can promote higher baseline blood pressures in less severe or incomplete lesions.[14]

Insulin Resistance, Metabolic Syndrome and Renal Effects

Reduced muscle mass, decreased physical activity, and increased adiposity after SCI contribute to insulin resistance and a higher prevalence of glucose intolerance and type 2 diabetes. Hyperinsulinemia enhances renal sodium reabsorption, activates the sympathetic nervous system, and stimulates smooth muscle proliferation in resistance vessels, thereby elevating blood pressure. Over time, microvascular damage in the kidney and other organs further impairs pressure natriuresis and perpetuates hypertension, forming a vicious cycle in which muscle atrophy, metabolic dysfunction, and vascular changes reinforce one another.[15]

Physical Fitness, Residual Muscle Function and Blood Pressure

Cross-sectional data in people with SCI show that higher muscular strength and endurance are associated with lower blood pressure and reduced prevalence of hypertension. Measures such as grip strength and arm-curl performance, which reflect residual muscle mass and conditioning, inversely correlate with systolic and diastolic pressures, independent of some confounders. These observations support a causal link whereby preservation or restoration of muscle mass and function may mitigate hypertension risk in SCI, likely through improvements in myokine profile, insulin sensitivity, and autonomic regulation.[16]

Integrative Pathophysiological Model

Taken together, the evidence supports a multifactorial model in which SCI-induced muscle atrophy initiates a cascade of changes—sarcopenic obesity, chronic inflammation, altered myokine–adipokine balance, autonomic dysregulation, and insulin resistance—that converge on endothelial dysfunction, arterial stiffness, and increased peripheral resistance. Autonomic dysreflexia adds episodic extreme hypertension in high lesions, which may further damage the vasculature and kidneys, amplifying the effects of chronic metabolic and inflammatory drivers. This integrated framework highlights muscle as both a structural and endocrine nexus linking neurological injury to cardiovascular outcomes, suggesting that muscle-targeted strategies are central to hypertension prevention in SCI.[17]

Clinical and Research Implications

From a clinical standpoint, early and sustained interventions to preserve or rebuild muscle mass—such as neuromuscular electrical stimulation, resistance training of innervated muscles, and optimized nutrition—may reduce long-term hypertension risk in SCI. Routine assessment of body composition, muscular fitness, and metabolic parameters should complement traditional blood pressure monitoring to identify high-risk individuals and guide personalized therapy. Future research should focus on longitudinal studies clarifying temporal relationships between muscle loss and hypertension, mechanistic trials targeting myostatin or specific myokines, and interventions that integrate autonomic management with muscle-focused rehabilitation in diverse SCI populations.[18]

CONCLUSION

The available evidence indicates that spinal cord injury creates a tightly interlinked neuromuscular–cardiovascular syndrome in which muscle

atrophy and blood pressure dysregulation are mutually reinforcing rather than isolated complications. SCI rapidly induces profound skeletal muscle wasting through denervation, unloading, and activation of catabolic molecular pathways, with parallel accumulation of intramuscular and visceral fat and the development of a sarcopenic–obesity phenotype. This altered body composition, combined with endocrine and inflammatory disturbances, drives insulin resistance, dyslipidaemia, and systemic low-grade inflammation, thereby amplifying cardiometabolic risk even in individuals with relatively low absolute body weight. At the same time, injury-level–dependent disruption of sympathetic pathways produces a broad spectrum of blood pressure abnormalities, ranging from chronic hypotension and orthostatic intolerance to life-threatening autonomic dysreflexia and conventional essential hypertension as patients age and accumulate metabolic risk factors. Together, these processes establish a chronic internal milieu that favours endothelial dysfunction, arterial stiffness, and sustained increases in peripheral vascular resistance.[19]

Within this framework, skeletal muscle emerges as a central pathophysiological nexus connecting SCI to hypertension. Loss of contractile tissue not only reduces mechanical function but also diminishes the endocrine role of muscle, lowering the production of protective myokines and shifting the balance toward pro-inflammatory, pro-atrophic mediators such as myostatin and cytokines. The resulting sarcopenic obesity augments sympathetic activation, renin–angiotensin–aldosterone system activity, and renal sodium retention, all of which contribute to the development and maintenance of elevated blood pressure. Recurrent episodes of autonomic dysreflexia superimpose acute, extreme hypertensive surges that likely accelerate vascular and end-organ damage, compounding the more insidious effects of chronic metabolic and inflammatory stress. Consequently, hypertension in SCI should be viewed as a downstream manifestation of integrated neuromuscular, autonomic, and metabolic dysfunction rather than as an isolated cardiovascular diagnosis.[20,21]

Clinically, this integrative perspective has important implications. It supports early, proactive strategies to preserve or restore muscle mass and quality—through neuromuscular or functional electrical stimulation, resistance training of innervated musculature, and tailored nutritional support—as key components of blood pressure and cardiovascular risk management in SCI. Routine monitoring should extend beyond brachial blood pressure to include body composition, muscle strength, fitness indices, and metabolic parameters, enabling identification of patients with high-risk sarcopenic–obesity profiles. Blood pressure control needs to be individualized, balancing prevention of hypotension and autonomic dysreflexia with aggressive treatment of sustained hypertension, and should be integrated with interventions that target inflammation, oxidative stress, and insulin resistance. From a research standpoint, longitudinal mechanistic studies are needed to clarify temporal relationships between muscle loss, autonomic changes, and blood pressure trajectories, and to define how modifying muscle mass, myokine signaling, or adiposity alters incident hypertension and cardiovascular outcomes. Ultimately, framing SCI as a condition in which muscle atrophy and hypertension are mechanistically coupled underscores the rationale for muscle-centered, multidisciplinary approaches to reduce long-term morbidity and improve survival in this vulnerable population.[22-26]

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