Pathophysiology: Why does muscle go away?

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Sarcopenia affects 40-70% of cirrhosis patients through multifactorial mechanisms including hyperammonemia, myostatin activation, and chronic inflammation, requiring early multimodal treatment approaches.

KEY POINTS

- Sarcopenia in cirrhosis is defined by skeletal muscle index below 50 in males and below 39 in females measured at the third lumbar vertebra on CT scan, with higher prevalence in decompensated patients and males.
- The speaker described key pathophysiological mechanisms: ammonia stimulates myostatin via NF-kB, blocking mTOR and causing decreased protein synthesis with increased autophagy; endotoxemia from leaky gut, TNF-alpha, and hormonal imbalances in males contribute synergistically.
- Myosteatosis (muscle fat infiltration) is a distinct finding that can occur independently of sarcopenia and was stated to be independently associated with decompensation and mortality in a multicenter study.
- The speaker reported that a phase 2 trial of an androgen receptor agonist showed significant improvement in skeletal muscle index and decreased hepatic encephalopathy risk, with phase 3 ongoing.
- In the discussion, the speaker recommended ultrasound of rectus femoris as a practical alternative to CT with sensitivity and specificity above 90%, particularly useful in low-resource settings or acutely ill patients.
- The speaker emphasized that sarcopenic muscle releases inflammatory mediators creating a vicious cycle with liver disease, and recommended starting treatment as early as possible with a multimodal approach targeting portal hypertension, malnutrition, and hormonal imbalances.

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