

Cachexia

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Why in News?

New research links cancer cachexia to disrupted brain-liver signalling, redefining it as a neuro-metabolic disorder.

- **Cachexia** - It is a complex; **chronic metabolic syndrome** associated with cancer.
- **Prevalence** - It affects up to **80% patients** with advanced cancers and accounts for a sizeable fraction of cancer deaths.
- **Nature** - Unlike starvation, cachexia involves increased energy expenditure and metabolic breakdown of muscle and fat driven by tumour-host biology.
- **Symptoms**
 - Persistent loss of body weight.
 - Loss of appetite (anorexia).
 - Progressive muscle wasting and weakness.
 - Reduced response to chemotherapy and radiotherapy.
- **Causes - Tumour-driven inflammation** - Disrupts normal brain-liver nerve signalling through the vagus nerve.
- **Liver metabolic reprogramming** - Altered neural signals **suppress HNF4α (a key regulator of liver metabolism)**, leading to appetite loss, systemic inflammation and muscle wasting.
- **Progression Stages** - Early metabolic imbalance with appetite loss.
- Progresses into liver metabolic dysfunction.
- Systemic inflammation and muscle breakdown.
- Advanced wasting with severe loss of weight and strength.
- **Diagnosis** - Regular monitoring of body weight along with assessment of muscle mass and strength.
- Evaluation of appetite and nutritional status.
- Metabolic and biochemical assessment.

- **Treatment - Early Recognition** - Cachexia should be identified as early as possible, as slowing progression is easier than reversing advanced wasting.
- **Integrated Care** - Treatment should focus on the whole patient, not just tumour control, combining multiple supportive strategies.
- **Anti-Inflammatory Support** - Inflammation needs to be addressed, though targeting single cytokines alone has shown limited success.
- **Targeted Nutrition** - Nutritional support is necessary to maintain function, even though feeding alone cannot reverse cachexia.
- **Physical Activity** - Exercise, where feasible, helps preserve muscle strength and functional capacity.
- **New treatment possibilities - Neuro-modulation (experimental)** - Interrupting abnormal vagus nerve signalling, including electrical stimulation, has reduced weight and muscle loss in animal models.
- **Liver-directed therapy (experimental)** - Preventing loss of HNF4 α (a key regulator of liver metabolism) or restoring hepatic metabolic programmes has limited wasting in preclinical studies.

References

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