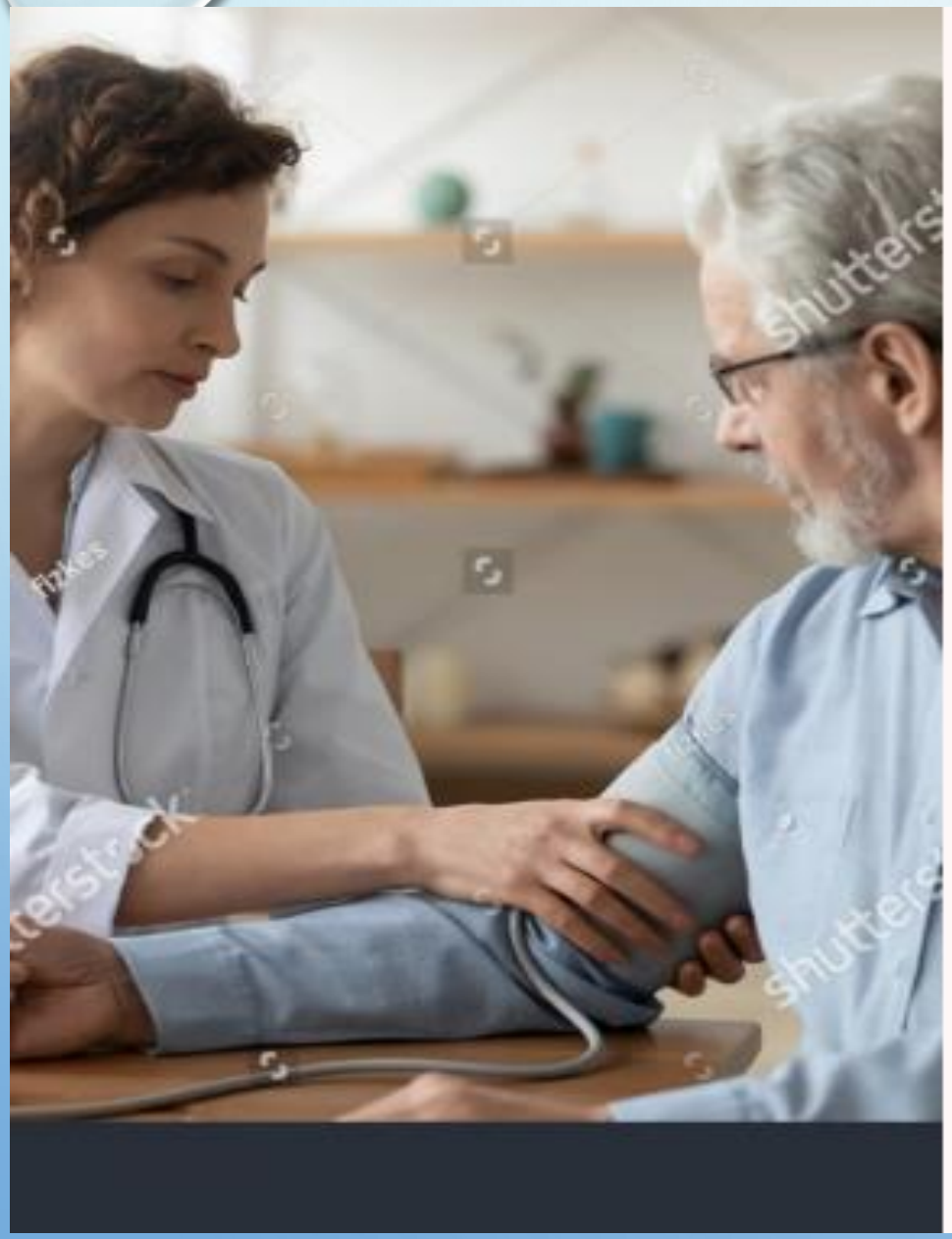

بسم الله الرحمن الرحيم



Sarcopenia in Patients with Type 2 Diabetes Mellitus

Diagnosis, Pathophysiology, Causes, Drugs and Treatment

SARCOPENIA IN PATIENTS WITH TYPE 2 DIABETES MELLITUS

DIAGNOSIS, PATHOPHYSIOLOGY, CAUSES, DRUGS, AND TREATMENT

OUTLINE

- Definition and Overview of Sarcopenia
- Epidemiology in Type 2 Diabetes Mellitus (DM2)
- Pathophysiological Mechanisms of Sarcopenia
- Diagnostic Criteria and Tools
- Drug-Related Causes of Sarcopenia
- Management and Treatment Strategies

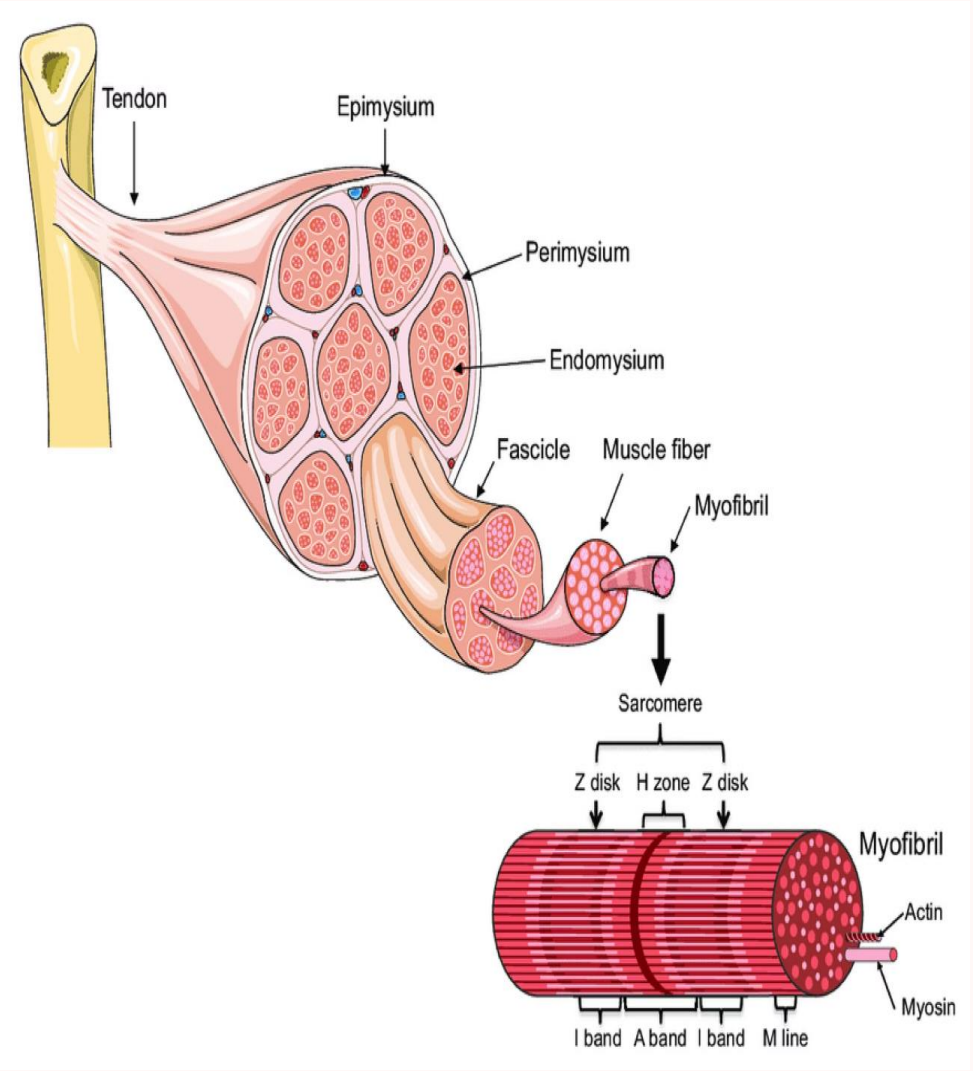
Section 1 – Definition and Overview

What is Sarcopenia?

- Loss of skeletal muscle mass and strength, particularly in the elderly
- Affects both muscle function and physical performance
- Contributes to frailty, falls, and disability in DM2 patients

What is Sarcopenia?

Sarcopenia represents a progressive loss of skeletal muscle mass and strength that leads to functional decline, frailty, and disability. It is a key determinant of quality of life in patients with type 2 diabetes mellitus.



This condition affects not only muscle quantity but also muscle quality, impacting the ability to perform daily activities and maintain independence. In DM2 patients, sarcopenia creates additional metabolic challenges that worsen overall health outcomes.

Section 1 – Definition and Overview

Types of Sarcopenia?

- Primary (age-related) vs. Secondary (disease-related)
- Acute vs. Chronic Sarcopenia
- Stages: Presarcopenia, Sarcopenia, Severe Sarcopenia

Classification of Sarcopenia:

Primary (Age-Related)

Natural decline associated with aging process, no other specific cause identified

Secondary (Disease-Related)

Caused by underlying conditions like diabetes, cancer, or organ failure

1

Pre-Sarcopenia

Low muscle mass only

2

Established Sarcopenia

Low mass + low strength or performance

3

Severe Sarcopenia

All three criteria present

Section 1 – Definition and Overview

Sarcopenia and Type 2 Diabetes Mellitus (DM2)?

- DM2 accelerates muscle atrophy through metabolic imbalances
- Sarcopenia exacerbates insulin resistance and worsens glycemic control
- A vicious cycle leading to further metabolic disturbances

Section 2 – Epidemiology and Clinical Significance

Epidemiology of Sarcopenia

- Affects 10–30% of elderly individuals
- Prevalence increases in patients with DM2 (up to 40%)
- Higher rates in individuals with poor glycemic control and comorbidities

Section 2 – Epidemiology and Clinical Significance

Clinical Burden of Sarcopenia

- Reduced muscle strength leads to increased fall risk and fractures
- Worsens quality of life, leading to disability and dependence
- Associated with higher hospitalization rates and mortality in DM2.

Section 2 – Epidemiology and Clinical Significance

Impact of Sarcopenia on Diabetes Outcomes

- Sarcopenia impairs insulin sensitivity and glucose uptake
- Worsens overall glycemic control in DM2 patients
- Increases cardiovascular risk due to muscle-related metabolic changes

Section 3 – Pathophysiology

Overview of Pathophysiology

- Complex interaction between metabolic, inflammatory, and hormonal factors
- Chronic hyperglycemia and insulin resistance are central drivers
- Mitochondrial dysfunction and oxidative stress play key roles

Section 3 – Pathophysiology

Insulin Resistance and Muscle Protein Metabolism

- Insulin normally stimulates muscle protein synthesis via the mTOR pathway
- Insulin resistance leads to reduced mTOR activation and muscle wasting
- Increased muscle catabolism via the ubiquitin–proteasome pathway

Section 3 – Pathophysiology

Inflammatory Mechanisms

- Chronic inflammation in DM2: increased TNF-α, IL-6, and CRP
- Inflammatory cytokines inhibit muscle regeneration via IGF-1 suppression
- Elevated cytokine levels promote muscle protein degradation

Section 3 – Pathophysiology

Mitochondrial Dysfunction

- DM2 causes reduced mitochondrial biogenesis in muscle cells
- Impaired oxidative phosphorylation leads to muscle weakness
- Increased ROS production causes muscle damage and fatigue

Section 3 – Pathophysiology

Hormonal Imbalance and Sarcopenia

- Low testosterone and estrogen levels contribute to muscle wasting
- Reduced growth hormone (GH) and IGF-1 signaling exacerbate sarcopenia
- Altered cortisol and adipokine secretion also promote muscle catabolism

Section 3 – Pathophysiology

Microvascular Impairment

- Diabetic microangiopathy reduces blood flow to muscle tissue
- Impairs nutrient and oxygen delivery, causing muscle atrophy
- Capillary rarefaction further decreases muscle regeneration potential

Microvascular Abnormalities

Endothelial Dysfunction

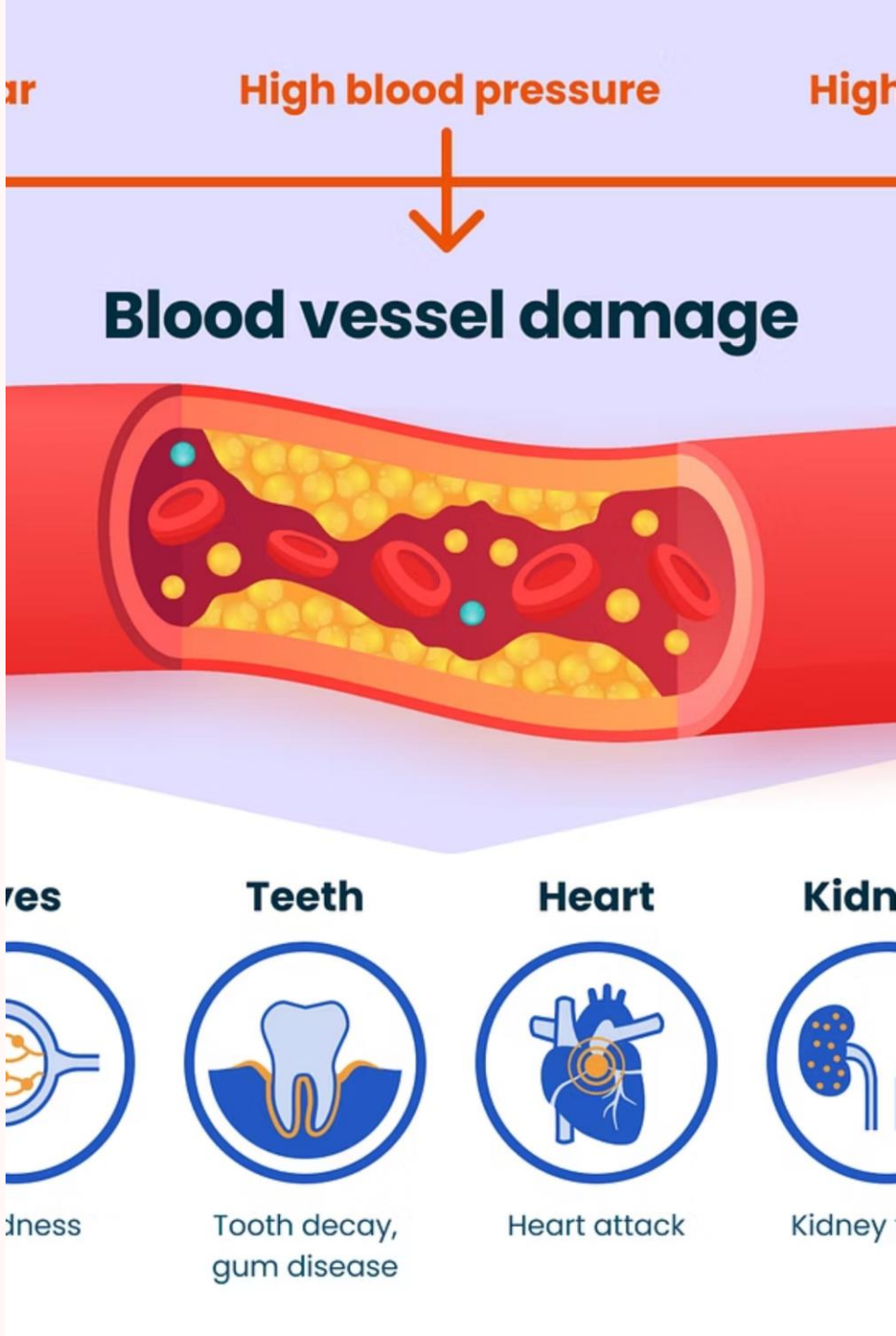
Damaged blood vessel lining reduces nutrient and oxygen flow to muscle tissue

Capillary Rarefaction

Loss of small blood vessels in diabetic muscle reduces perfusion capacity

Chronic Ischemia

Inadequate blood supply impairs muscle regeneration and repair mechanisms



Section 3 – Pathophysiology

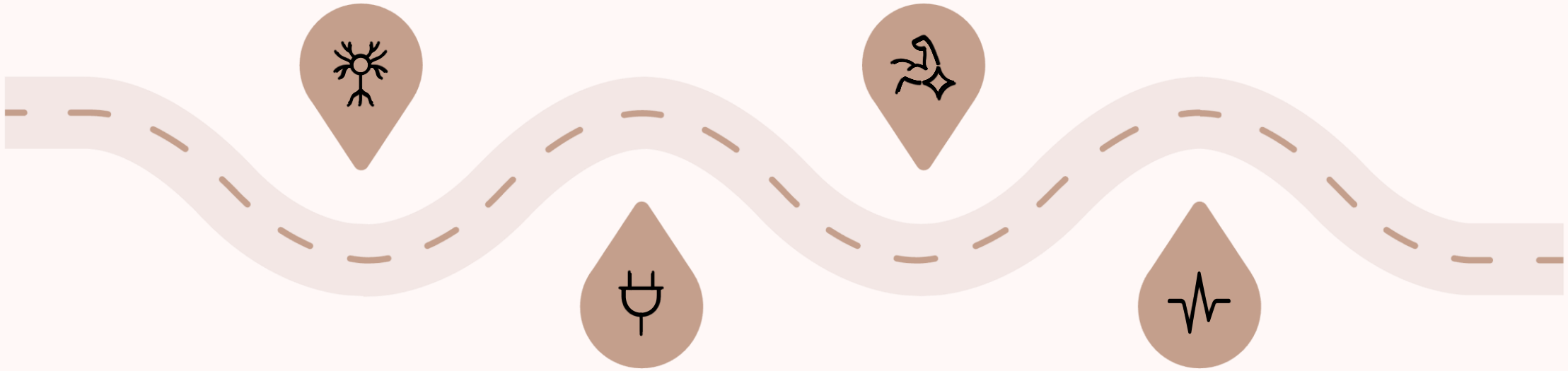
Neuromuscular Factors

- Diabetic neuropathy results in muscle denervation and atrophy
- Altered motor unit recruitment and muscle fibers' reduced contractility
- Loss of motor coordination increases fall risk and accelerates sarcopenia

Neuromuscular Changes:

Nerve Damage

Hyperglycemia injures peripheral nerves



Denervation

Loss of motor axons and connectivity connectivity

Muscle Atrophy

Fiber shrinkage and reduced mass

Impaired Recruitment

Altered motor units and weaker contractions

Diabetic neuropathy creates a cascade of neuromuscular complications that directly contribute to muscle loss and functional decline.

Section 3 – Pathophysiology

Lipotoxicity and Sarcopenia

- Intramyocellular lipid accumulation reduces insulin sensitivity
- Interference with muscle cell signaling leads to insulin resistance
- Increased fat storage in muscle fibers leads to oxidative stress

Diagnosis in Clinical Practice

Integrated Diagnostic Algorithm

- **Step 1:** Screening with SARC-F or calf circumference
- **Step 2:** Confirm diagnosis with strength, mass, and performance tests
- **Step 3:** Identify secondary causes (lab tests, medication review)

Section 4 – Diagnosis

Screening Tools

- SARC-F questionnaire (Strength, Assistance, Rise, Climb, Falls)
- Calf circumference as an indirect indicator of muscle mass
- Basic functional assessments like chair stand test

SARC-F:

STRENGTH

AMBULATION

RISING FROM A CHAIR

STAIR CLIMBING

HISTORY OF FALLING

Component	Question	Scoring	Score
Strength	How much difficulty do you have in lifting and carrying 4.5 kgs?	None = 0 Some = 1 A lot or unable = 2	
Assistance in walking	How much difficulty do you have walking across a room?	None = 0 Some = 1 A lot, use aids, or unable = 2	
Rise from a chair	How much difficulty do you have transferring from a chair to bed?	None = 0 Some = 1 A lot or unable without help = 2	
Climb stairs	How much difficulty do you have climbing a flight of 10 stairs?	None = 0 Some = 1 A lot or unable = 2	
Falls	How many times have you fallen in the past year?	None = 0 1-3 falls = 1 4 or more falls = 2	

Screening Tools:

1

SARC-F Questionnaire

Five-item self-report tool assessing Strength, Assistance walking,

Rising from chair, Climbing stairs, Falls

2

Calf Circumference

Simple measurement: < 34 cm (men) or < 33 cm (women) suggests low muscle mass

3

Functional Tests

Quick performance assessments suitable for outpatient clinical settings

Section 4 – Diagnosis

Diagnostic Overview

- Diagnosis based on muscle mass, strength, and physical performance
- Guidelines from EWGSOP2, AWGS, and FNIH
- Two-step diagnostic approach: screening followed by confirmation

Diagnostic Principles:

Muscle Mass	Muscle Strength	Physical Performance
Quantify lean tissue	Assess functional capacity	Evaluate real-world function

EWGSOP2 and AWGS guidelines recommend a systematic approach: screen first with simple tools, then confirm with objective testing. All three components must be evaluated for accurate diagnosis.

Muscle Mass Measurement Methods:

DXA (Dual-Energy X-ray Absorptiometry)

Gold standard for measuring appendicular lean mass. Provides precise, reproducible measurements with low radiation exposure. Widely used in research and clinical practice.

BIA (Bioelectrical Impedance Analysis)

Portable, affordable option for screening. Estimates body composition through electrical conductivity. Less precise than DXA but suitable for monitoring trends.

CT/MRI Scanning

Most precise quantification of muscle and fat distribution. Can assess muscle quality and fat infiltration. Expensive and typically reserved for research settings.

ioi 353 BODY COMPOSITION ANALYSIS

Name / ID			
Date	4/26/2021 12:50:00		
Height	156.0 cm	Age	53 yrs
Weight	92.6 kg	Gender	Female

Body Composition

[kg]

Weight /Over 92.6 [48.1~58.8]	Std.wt. 53.5			
L.B.M. /Over 50.5 [37.5~42.8]			Body Fat 42.1	
S.L.M. /Over 45.4 [34.5~39.5]		Mineral 5.1	Body Fat 42.1	
T.B.W. /Over 36.4 [27.0~30.8]	Protein /Over 9.0 [7.4~8.5]	Mineral /Over 5.1 [2.9~3.2]	Body Fat /Over 42.1 [10.7~16.1]	

Std.wt. : Standard weight L.B.M. : Lean Body Mass S.L.M. : Soft Lean Mass T.B.W. : Total Body Water
Mineral is conservative estimate.
The assessment of Under, Optimal and Over is decided by standard weight on Body Composition table.

Assessment of Weight Control

Item	Under	Optimal	Over
Weight kg	70 80 90 100 110 120 130 140 150 (%)		92.6
B.M.I. kg/m²	14.50 16.50 18.50 21.75 25.00 27.50 30.00 32.50 35.00 (kg/m²)		38.1
P.B.F. %	10.0 15.0 20.0 25.0 30.0 35.0 40.0 45.0 50.0 (%)		45.5
S.L.M. kg	70 80 90 100 110 120 130 140 150 (%)		45.4

B.M.I. : Body Mass Index P.B.F. : Percent Body Fat

Abdominal Analysis

Type	Subcutaneous	Balanced	Borderline	Visceral I	Visceral II
Level	1 5 9 11 16				19
V.F.A. cm²	40 80				260
A.C. cm	105.5cm (Less than 88cm)	W.H.R.	1.01 [0.70 - 0.85]		

A.C. : Abdominal Circumference
A.C. is an estimated value in case of measure the navel circumference.
Accuracy of abdominal analysis may be decreased in case of idiosyncrasy.

Control Guide

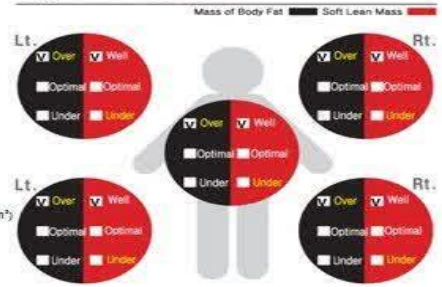
[kg]

	Measured data	Control		Goal to control
Weight	92.6	+ 39.1	Target to control	+ 28.7
M.B.F.	42.1	+ 28.7	Control/week	0.5
S.L.M.	45.4	+ 8.4	Duration of control	57 week
B.M.R.	1197 kcal	T.E.E.		1580 kcal
A.M.B.	61 yrs	Impedance	446 Ω	

M.B.F. : Mass of Body Fat B.M.R. : Basal Metabolic Rate T.E.E. : Total Energy Expenditure A.M.B. : Age Matched of Body
Age Matched of Body is reference value.
Control guide and calorie prescription are proposed value for your body type.
The T.E.E. is estimated value.

Body Type

Segmental Assessment



Item	Lt.Arm	Rt.Arm	Trunk	Lt.Leg	Rt.Leg
M.B.F. kg	2.79	2.87	21.44	7.50	7.48
S.L.M. kg	2.85	2.78	23.02	8.37	8.38

Segmental Assessment is reference value.

Blood Pressure

Systolic mmHg		Diastolic mmHg	
Pulse bpm			

You need to control 550 kcal from T.E.E. 1580 kcal.

By diet Reduce 220 kcal	Diet prescription calorie 1360kcal
By exercise Consume 330 kcal	Exercise prescription calorie 330 kcal

Body Composition Change

	Date	Weight	M.B.F.	S.L.M.
Previous				
Present	4/26/2021	92.6	42.1	45.4

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Section 4 – Diagnosis

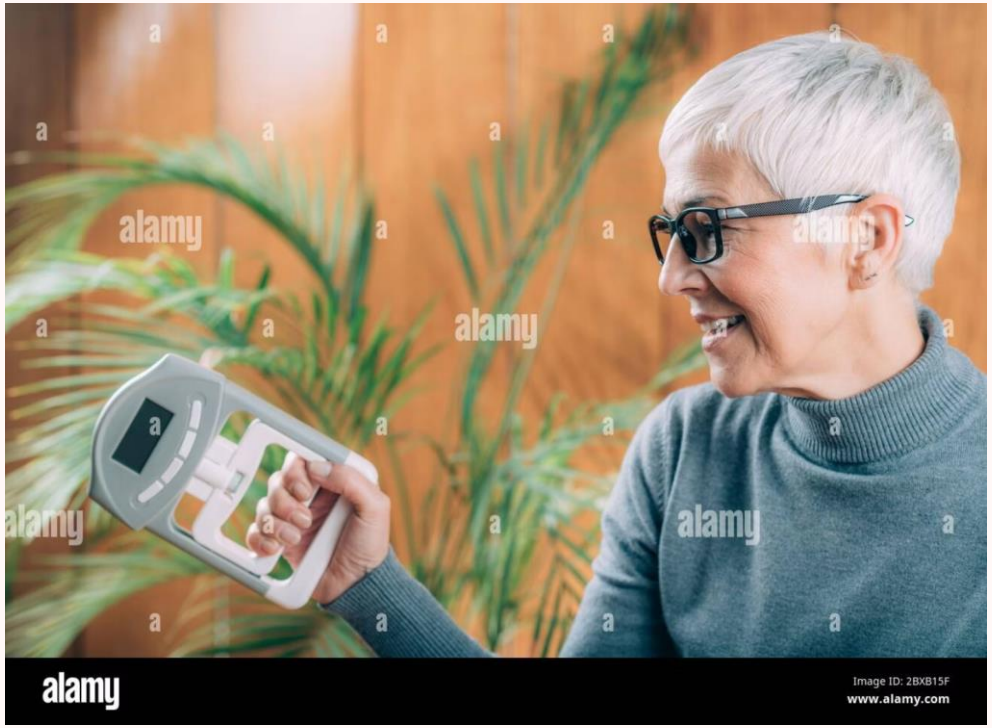
Measuring Muscle Strength

- Handgrip dynamometry as the most reliable test
- Chair stand test (5 times sit-to-stand in 30 seconds)
- Correlates strongly with overall physical performance

Measuring Muscle Strength:

- **Handgrip dynamometry as the most reliable test**
- **Chair stand test (5 times sit-to-stand in 30 seconds)**
- **Correlates strongly with overall physical performance**

Muscle Strength Assessment



Hand-Grip Dynamometry

Gold standard for measuring muscle strength. Simple, quick, and highly predictive of overall functional capacity.

Chair Stand Test

Time required to rise from a chair five times without using arms. Reflects lower body strength and functional independence.

Clinical Significance

Strength measurements correlate strongly with mortality risk and quality of life outcomes.

Section 4 – Diagnosis

Measuring Muscle Mass

- Dual-energy X-ray absorptiometry (DXA) as the gold standard
- Bioelectrical impedance analysis (BIA) for quick screening
- MRI or CT for precise muscle volume quantification

Section 4 – Diagnosis

Measuring Physical Performance

- Gait speed test (a speed of ≤0.8 m/s indicates functional impairment)
- Short Physical Performance Battery (SPPB) for comprehensive assessment
- Timed Up and Go (TUG) test to evaluate mobility

Physical Performance Tests:

Gait Speed Assessment

Walking speed < 0.8 m/s indicates poor performance and predicts adverse outcomes. Simple 4-meter walk test provides valuable prognostic information.

Short Physical Performance Battery (SPPB)

Comprehensive assessment combining balance, gait speed, and chair stand tests. Scores range from 0-12, with lower scores indicating greater disability risk.

Timed Up-and-Go Test

Measures time to stand from chair, walk 3 meters, turn, and return to seated position. Evaluates mobility, balance, and fall risk.

Section 4 – Diagnosis

Laboratory and Biomarkers

- Serum creatinine and cystatin C as markers of muscle health
- Myostatin and follistatin for muscle growth regulation
- Inflammatory markers (TNF-α, IL-6, CRP) and metabolic parameters (HbA1c)

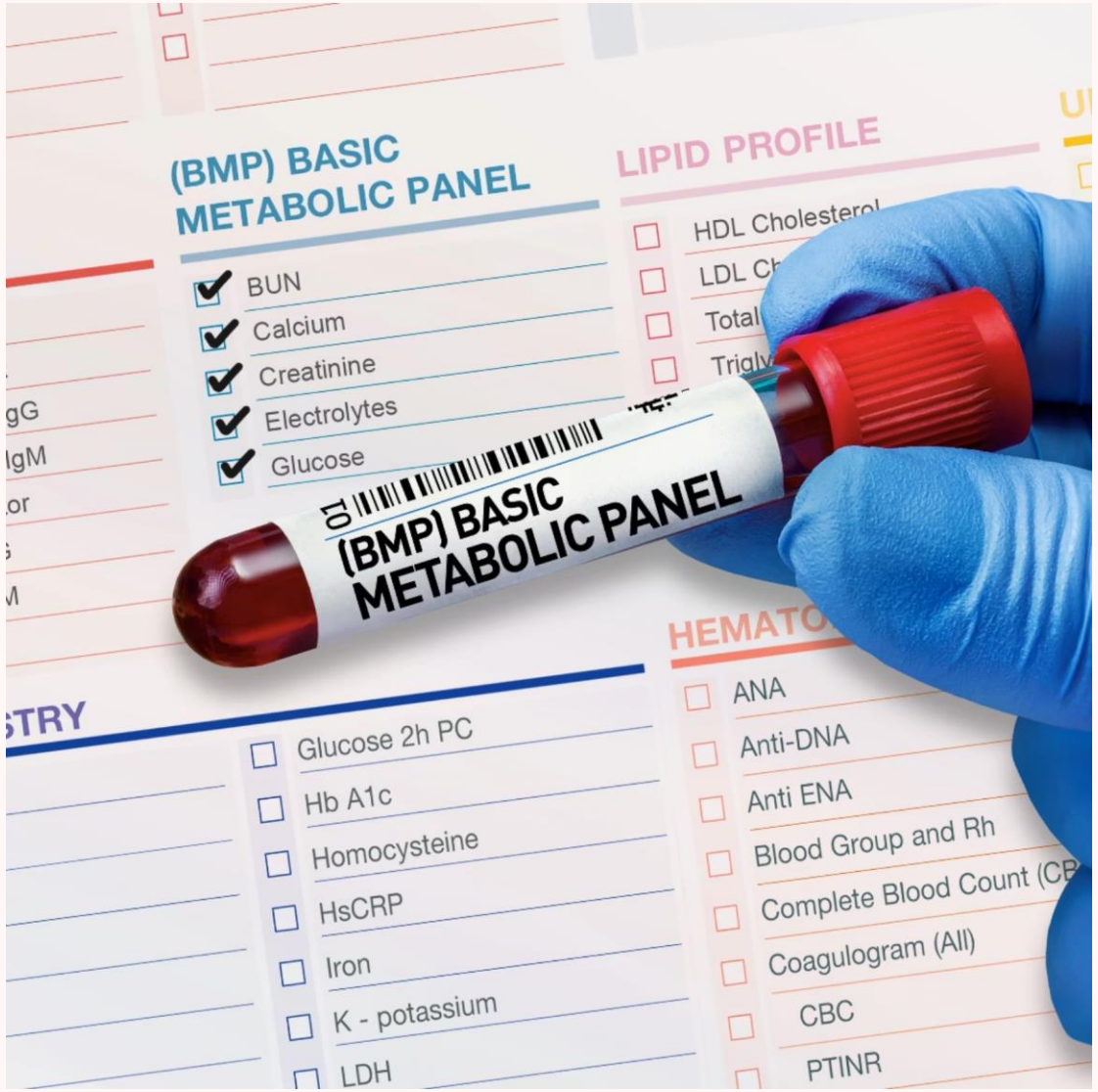
Laboratory and Body Composition Assessment:

Serum Markers

- Creatinine and cystatin C reflect muscle mass
- Myostatin/follistatin indicate anabolic state
- TNF-α, IL-6, CRP measure inflammation
- HbA1c for glycemic control

Body Composition Examples

- DXA: Appendicular Lean Mass = 14.5 kg (≤ 15 = low)
- BIA: Skeletal Muscle Index = 6.4 kg/m² (men < 7)
- CT: Visible thigh muscle fat infiltration



Section 5 – Causes and Contributing Factors

Aging and Sarcopenia in DM2

- Sarcopenia naturally worsens with age, especially in those with DM2
- Insulin resistance and metabolic changes accelerate muscle loss
- Age-related anabolic resistance worsens in DM2 patients

Section 5 – Causes and Contributing Factors

Physical Inactivity

- Sedentary lifestyle is common in DM2 patients
- Lack of muscle use exacerbates atrophy and insulin resistance
- Regular exercise is critical to managing sarcopenia in DM2

Section 5 – Causes and Contributing Factors

Nutritional Deficiencies

- Inadequate protein intake accelerates muscle wasting
- Vitamin D deficiency contributes to muscle weakness and falls
- Micronutrient imbalances (Zn, Mg) hinder muscle function

Section 5 – Causes and Contributing Factors

Chronic Hyperglycemia

- High blood sugar leads to glycation of muscle proteins (AGEs)
- AGEs cause muscle stiffness and reduced regeneration capacity
- Chronic hyperglycemia also increases oxidative stress in muscle fibers

Section 5 – Causes and Contributing Factors

Sarcopenic Obesity

- Simultaneous presence of obesity and sarcopenia in DM2
- Adipose tissue secretes inflammatory cytokines that increase muscle degradation
- This phenotype increases the risk of diabetes complications

Section 5 – Causes and Contributing Factors

Diabetic Neuropathy and Muscle Wasting

- Sensory loss and muscle disuse due to peripheral neuropathy
- Motor denervation further accelerates muscle loss and weakness
- Early intervention can help prevent functional decline

Section 5 – Causes and Contributing Factors

Vascular Complications

- Microvascular damage reduces oxygen and nutrient supply to muscles
- Impaired perfusion accelerates muscle degeneration
- Diabetic vascular complications contribute to functional decline

Section 6 – Drug-Induced Sarcopenia

Drug Effects Overview

- Certain drugs used in DM2 treatment can induce sarcopenia
- Mechanisms: mitochondrial toxicity, reduced protein synthesis, and increased catabolism
- Includes both antidiabetic and non-diabetic medications

Section 6 – Drug-Induced Sarcopenia

Metformin and Sarcopenia

- Metformin's mitochondrial effects may reduce muscle cell energy
- Long-term use can lead to vitamin B12 deficiency
- Possible protective anti-inflammatory effects in some patients

Section 6 – Drug-Induced Sarcopenia

Thiazolidinediones (TZDs)

- Improve insulin sensitivity but increase adipose tissue mass
- No significant benefit on muscle mass; can exacerbate sarcopenia
- Fluid retention and weight gain may limit long-term use

Section 6 – Drug-Induced Sarcopenia

Sulfonylureas and Sarcopenia

- Risk of hypoglycemia may reduce physical activity
- Potentially worsens sarcopenia by reducing exercise and movement
- No direct effects on muscle mass but an indirect contributor

Section 6 – Drug-Induced Sarcopenia

Insulin Therapy

- Anabolic effects on muscle mass in the short term
- Overuse can cause weight gain with minimal muscle gain
- Needs to be combined with exercise and nutritional support

Section 6 – Drug-Induced Sarcopenia

Statins and Sarcopenia

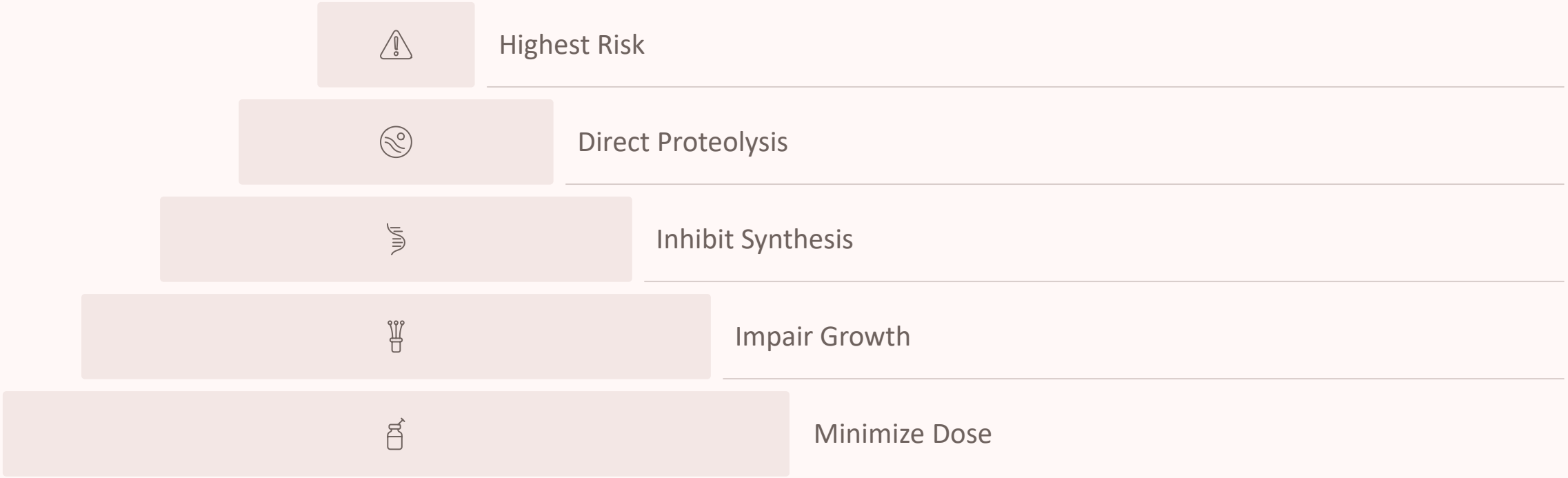
- Statins can cause myopathy by affecting mitochondrial function
- Reduced coenzyme Q10 levels may contribute to muscle damage
- Monitor creatine kinase (CK) levels for early detection of muscle injury

Section 6 – Drug-Induced Sarcopenia

Glucocorticoids

- Known to induce muscle catabolism via cortisol elevation
- Prolonged use accelerates muscle wasting and weakness
- Requires dose adjustment and close monitoring in DM2

Glucocorticoids:



Glucocorticoids directly stimulate muscle proteolysis while inhibiting protein synthesis and fiber growth. Dose minimization and careful tapering are essential to preserve muscle mass.

Table 1 Effects of anti-diabetic drugs on patients with geriatric type 2 diabetes mellitus and sarcopenia.

Anti-diabetic drugs	Effect on sarcopenia	Good option or poor option
Biguanides	Positive[31,33-36,38-40]/negative[41-44]	Unclear
Insulin secretagogues	Negative[46-48]	Poor
-Glucosidase inhibitors	No data	No data
Thiazolidinediones	Positive[35,53-56]	Careful use
Dipeptidyl peptidase IV inhibitors	Positive[57-60]/neutral[62]	Good
Glucagon-like peptide-1 receptor agonists	Positive[63,70,71]/negative[62]	Unclear
Sodium-glucose cotransporter-2 inhibitor	Positive[72,73]/unclear[6,74]	Unclear
Insulin	Positive/unclear[75]	Unclear

Effects of anti-diabetic drugs on patients with geriatric type 2 diabetes mellitus and sarcopenia.

Biguanides : Positive?

Insulin secretagogues: Negative

Glucosidase inhibitors :No data

Thiazolidinediones: Positive

Dipeptidyl peptidase IV inhibitors :Positive

Glucagon-like peptide-1 receptor agonists: Positive

Sodium-glucose cotransporter-2 inhibitor :unclear

Insulin :Positive

Section 7 – Diagnosis in Clinical Practice

Integrated Diagnostic Algorithm

- **Step 1:** Screening with SARC-F or calf circumference
- **Step 2:** Confirm diagnosis with strength, mass, and performance tests
- **Step 3:** Identify secondary causes (lab tests, medication review)

Section 7 – Diagnosis in Clinical Practice

Considerations in DM2 Diagnosis

- Adjust diagnostic cut-offs for BMI and body fat distribution in DM2
- Regular monitoring of muscle function in elderly DM2 patients
- Account for comorbidities and polypharmacy

Section 8 – Management and Treatment

Treatment Goals

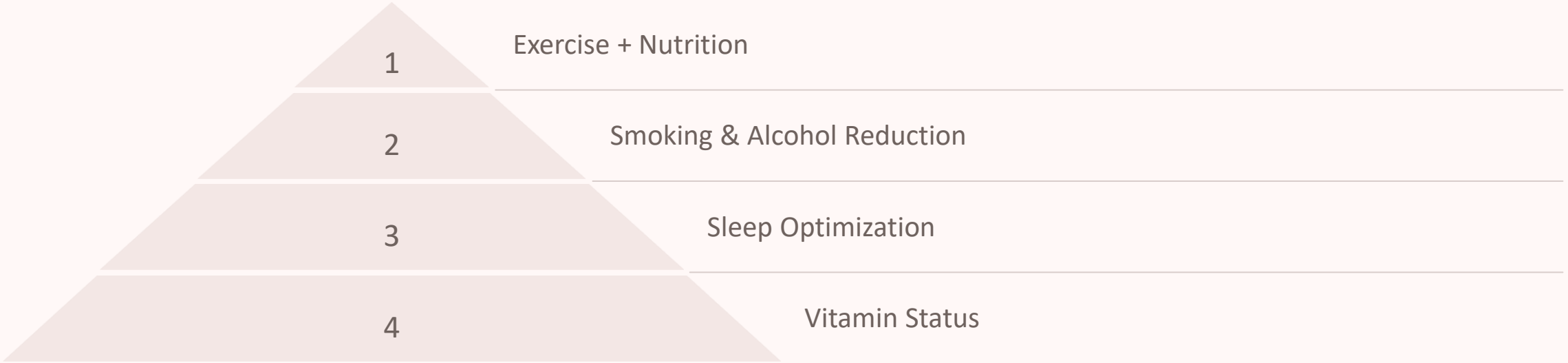
- Reverse or slow the progression of sarcopenia
- Improve muscle strength and insulin sensitivity
- Reduce the risk of falls, fractures, and disability

Section 8 – Management and Treatment

Lifestyle Modifications

- Exercise: resistance training 2–3 times a week, aerobic activity ≥150 min/week
- Protein intake: 1.2–1.5 g/kg body weight/day
- Weight management and optimizing glycemic control

Lifestyle Foundations



Exercise and nutrition form the cornerstone of sarcopenia therapy.
All other interventions build upon this foundation for optimal results.

Section 8 – Management and Treatment

Exercise Therapy

- Focus on both aerobic and resistance training
- Aerobic activity improves cardiovascular health and insulin sensitivity
- Resistance training increases muscle mass and strength

Exercise Therapy

Resistance Training

2–3 sessions per week targeting major muscle groups. Progressive overload essential for muscle growth and strength gains.

Aerobic Training

≥ 150 minutes weekly of moderate-intensity activity. Improves cardiovascular cardiovascular health and insulin sensitivity.

Combined Approach

Combination of resistance and aerobic exercise provides best metabolic benefit and functional outcomes.

Section 8 – Management and Treatment

Nutritional Interventions

- Protein-rich diet, especially leucine-based sources
- Adequate intake of vitamins D and E, omega-3 fatty acids
- Micronutrient supplementation as necessary: Zn , Mg,..

Nutritional Interventions and Protein Protocol

High-Quality Protein Sources

Leucine-rich sources including whey, fish, soy, and eggs provide optimal amino acid profiles for muscle protein synthesis.

1.2-1.6	0.4	1
g/kg/day	g/kg/meal	hour
Total daily protein intake for older DM2 patients	Protein distribution across 3 meals	Post-exercise protein timing window

Breakfast: 25g protein | Lunch: 35g protein | Dinner: 30g protein

Ensure vitamin D levels ≥ 30 ng/mL for optimal muscle response.

📊 میزان پروتئین پیشنهادی

گروه	میزان پروتئین (g/kg/day)	مثال عملی (kg 70)
سالمندان سالم	1.2-1.6	84-112 گرم/روز
سارکوپنی	1.6-2.0	112-140 گرم/روز
پس از جراحی	1.5-2.0	105-140 گرم/روز
بیماران سرطانی	1.2-2.0	84-140 گرم/روز

مثال‌های عملی منابع پروتئینی

غذا	مقدار	پروتئین (g)	لوسین (g)	کالری
تخم‌مرغ کامل	1 عدد (50g)	6.3	0.5	70
سفیده تخم‌مرغ	1 عدد	3.6	0.3	17
ماست یونانی	150g	15	1.5	90
پنیر کم‌چرب	30g	8	0.8	70
سینه مرغ کبابی	100g	31	2.5	165
ماهی قزل‌آلا	100g	20	1.8	208
ساردین کنسروی	100g	25	2.0	208
عدس پخته	100g	9	0.7	116
وی پروتئین	1 پیمانه (30g)	24	2.5	120

🍽️ برنامه نمونه توزیع پروتئین (110 گرم/روز)

وعده	زمان	غذا	پروتئین (g)	لوسین (g)
صبحانه	7:30	2 عدد تخم‌مرغ + 150g ماست یونانی + 30g پنیر	28	2.8
میان‌وعده 1	10:00	20g وی پروتئین + 10g بادام	24	3.0
ناهار	13:00	120g سینه مرغ + 100g عدس پخته + سبزیجات	32	2.5
میان‌وعده 2	16:00	150g ماست یونانی + 15g گردو	18	1.8
شام	19:00	100g ماهی قزل‌آلا + 80g لوبیا سبز	26	2.2
قبل خواب	22:00	20g کازئین	18	2.0
📊 مجموع			110g	14.3g

۱ـ فهرست جانشینی گروه نان و غلات	
نان سفید	یک برش ۳۰ گرمی
نان ساندویچی	یک برش ۳۰ گرمی
نان ماشینی	یک برش ۳۰ گرمی
نان جو	یک برش ۳۰ گرمی
نان سنگک	یک برش ۳۰ گرمی
جو پخته	نصف لیوان
برنج پخته	۵ قاشق غذاخوری
جوانه گندم	۳ قاشق غذاخوری
انواع آرد	۳ قاشق غذاخوری
ماکارونی پخته	۴ قاشق غذاخوری
انواع حبوبات (پخته)	۴ قاشق غذاخوری
باقلا پخته	نصف لیوان
بیسکوئیت سبوس‌دار	۲ عدد
ذرت بو داده	۳ لیوان
بلال	یک عدد کوچک یا نصف لیوان پخته
نخود سبز پخته	نصف لیوان
سیب زمینی	یک عدد کوچک
کدوتنبل پخته	نصف لیوان
حلوایی پخته	نصف لیوان
هر واحد این گروه ۱۵ گرم کربوهیدرات، ۳ گرم پروتئین، مقدار جزیی چربی و ۸۰ کیلوکالری انرژی دارد.	

۲ـ فهرست جانشینی گروه لبنیات	
شیر یا ماست	۱ لیوان ۲۴۰ سی سی
دوغ	۲ لیوان ۴۸۰ سی سی
کشک رقیق	۱۰ قاشق غذاخوری
کشک غلیظ	۴ قاشق غذاخوری
هر واحد این گروه حاوی ۱۲ گرم کربوهیدرات و ۸ گرم پروتئین و ۲/۵ درصد چربی، ۱۳۴ کیلوکالری انرژی است.	

۳ـ فهرست جانشینی گروه گوشت	
پنیر	۳۰ گرم
تخم مرغ	۱ عدد
جگر و دل و قلوه	۳۰ گرم
گوشت (گاو، گوساله، گوسفند)	۳۰ گرم
مرغ- ماهی- میگو	۳۰ گرم
هر واحد این گروه حاوی ۷ گرم پروتئین و بسته نوع چربی مقدار متفاوتی انرژی ۷۵،۵۵،۳۵ یا ۱۰۰ کیلوکالری است (هر ۳۰ گرم معادل یک قوطی کبریت)	

۵ـ فهرست جانشینی گروه میوه	
آلو برقانی– بخارا	۲ عدد
آلو زرد	۲ عدد
انجیر تازه	۲ عدد
انگور	۱۵ حبه (یک خوشه کوچک)
برگه زرد آلو	۸ تیمه
پرتقال	۱ عدد کوچک
تمشک خام	۱ لیوان
توت	نصف لیوان
توت خشک	۲ قاشق غذاخوری
توت فرنگی	یک و یک چهارم لیوان
خربزه	۱ لیوان خرد شده
خرما	۲/۵ عدد
خرمالو	۲ عدد
زالزالک	نصف لیوان
زردآلو	۴ عدد کوچک
سیب	۱ عدد کوچک
شلیل	۱ عدد متوسط
کشمش	۲ قاشق غذاخوری
کیوی	۱ عدد کوچک
گرمک	۱ لیوان خرد شده
گریپ فروت	نصف یک عدد
گلابی	نصف یک عدد بزرگ
گیلاس–آلبالو	۱۲ عدد (نصف لیوان)
لیمو شیرین	۱ عدد کوچک
موز	نصف یک عدد بزرگ
نارنگی	۱ عدد بزرگ
هلو	۱ عدد متوسط
هندوانه	یک و یک چهارم لیوان خرد شده
هر واحد میوه حاوی ۱۵ گرم کربوهیدرات ۶۰ کیلو کالری انرژی و ۲ گرم فیبر است.	

۶ـ فهرست جانشینی گروه سبزی	
اسفناج پخته	نصف لیوان
بامیه پخته	نصف لیوان
پیاز پخته	نصف لیوان خرد شده
پیاز خام	یک عدد متوسط
جعفری خام	۵۰ ساقه
چغندر پخته	نصف لیوان
خیار	یک عدد متوسط
شلغم پخته	نصف لیوان
فلفل دلمه	۱ عدد متوسط
فلفل سبز	۹۰ گرم
قارچ پخته	نصف لیوان
قارچ خام	یک لیوان
کاهو	یک لیوان خرد شده
کرفس پخته	نصف لیوان
کرفس خام	یک لیوان
کلم، گل کلم خام	یک لیوان
کلم، گل کلم پخته	نصف لیوان
گوجه فرنگی	یک عدد متوسط
لوبیا سبز پخته	نصف لیوان
هویج	یک عدد متوسط
هر واحد سبزی حاوی ۵ گرم کربوهیدرات، ۲ گرم پروتئین، ۲۵ کیلو کالری انرژی و ۱ تا ۴ گرم فیبر است.	

توزیع مناسب پروتئین در وعده های غذایی: پروتئین نه در یک وعده سنگین بلکه باید به صورت تقسیم‌شده در روز مصرف شود تا تحریک سنتز پروتئین عضلانی به صورت متناوب انجام شود.

برای تحریک مطلوب سنتز پروتئین عضلانی بهتر است پروتئین در چند وعده پخش شود، نه تمام آن در یک وعده.

داده‌ها نشان می‌دهند هر وعده باید حدود 25–30 گرم پروتئین با کیفیت (یا معادل ~2.5–3 گرم لوسین) داشته باشد تا آستانه آنابولیک در سالمندان فعال شود.

3–5 وعده غذایی روزانه با تاکید بر انتخاب منابع پروتئینی با کیفیت بالا (لبنیات ، تخم‌مرغ، گوشت کم‌چرب، ماهی، پروتئین وی، سپس منابع گیاهی غنی از لوسین مانند مخلوط نخود و برنج، حبوبات ترکیبی و آجیل سویا)

توصیه عملی : اگر صبحانه معمول نان و پنیر است: اضافه کردن یک کاسه ماست/کشک یا یک یا دو عدد تخم‌مرغ به بهبود کیفیت پروتئین صبحانه کمک بزرگی می‌کند.

برای نهار و شام: جایگزینی بخشی از نان/سیب‌زمینی با حبوبات پخته (عدس/نخود) یا سبزیجاتِ پروتئینی (خورش لوبیا سبز) پروتئین را بالا می‌برد .کباب مرغ/ماهی یا خوراک ماهی (ساردین/قزل‌آلا) 2–3 بار در هفته توصیه می‌شود .آجیل (۳۰ گرم) به‌عنوان میان وعده تقریبا 5–7 g پروتئین اضافه می‌کند.

Section 8 – Management and Treatment

Pharmacologic Treatments

- Use of anabolic agents (SARMs, myostatin inhibitors) in clinical trials
- Hormone replacement (testosterone, growth hormone) when appropriate
- Metformin, combined with exercise, may have synergistic benefits

Section 8 – Management and Treatment

Advanced Pharmacotherapy

- Anti-inflammatory drugs targeting TNF-α and IL-6 pathways
- Mitochondrial enhancers to improve muscle cell function
- Research into novel agents like irisin and myokines

Pharmacologic and Adjunctive Therapies

Experimental Anabolics

SARMs and myostatin inhibitors under investigation

Hormone Replacement

When clinically indicated for deficiency

Integrated Approach

Exercise + protein + safe antidiabetic drugs

Advanced Targets and Future Drugs

Anti-Cytokine Agents

TNF-α and IL-6 blockers to reduce inflammatory muscle catabolism

Mitochondrial Enhancers

CoQ10 and L-carnitine to improve cellular energy production

Myokine-Based Therapies

Irisin and myonectin to promote muscle growth and metabolism

Clinical Integration

Screen All Patients

Assess older DM2 patients for muscle loss

Include Lifestyle

Nutrition and exercise in routine care

Optimize Therapy

Choose antidiabetic drugs for muscle health

Section 8 – Management and Treatment

Multidisciplinary Approach

- Coordination between endocrinologists, physiotherapists, dietitians
- Regular monitoring of body composition and muscle strength
- Empowering patients through education and engagement

Section 9 – Prognosis and Future Directions

Prognosis of Sarcopenia in DM2

- Early detection and intervention can significantly improve outcomes
- Untreated sarcopenia worsens frailty and increases risk of death
- Prognosis depends on timely diagnosis and comprehensive care

Section 9 – Prognosis and Future Directions

Research Gaps

- Standardization of sarcopenia diagnostic criteria in DM2
- Long-term studies on the effectiveness of anabolic agents
- Exploration of personalized treatment approaches based on genetics

Section 9 – Prognosis and Future Directions

Emerging Technologies

- Artificial intelligence for muscle mass measurement (CT, DXA)
- Wearable devices for monitoring daily physical activity
- Remote monitoring tools for exercise adherence

Section 10 – Summary and Conclusion

Key Takeaways

- Sarcopenia is prevalent in DM2 and worsens patient outcomes
- Early screening, proper diagnosis, and treatment are critical
- Lifestyle interventions, including exercise and nutrition, are foundational

Section 10 – Summary and Conclusion

Clinical Relevance

- Sarcopenia management should be integrated into DM2 care
- Multidisciplinary management improves outcomes
- Patient education is key to successful treatment

Section 10 – Summary and Conclusion

Conclusion

- Sarcopenia in DM2 is complex but manageable
- Early intervention with a comprehensive approach is essential for better prognosis
- Further research is needed to optimize treatments

Practical Implementation Checklist:

- ## Initial Assessment

  SARC-F questionnaire, calf circumference, hand-grip strength, gait speed

- ## Confirmatory Testing

  DXA or BIA for muscle mass, comprehensive metabolic panel, vitamin D level

- ## Intervention Plan

  Prescribe resistance + aerobic exercise, calculate protein needs, review medications

- ### Follow-Up Schedule

  Reassess strength monthly, body composition every 3-6 months, adjust plan as needed

- ## Patient Education

  Provide written materials, demonstrate exercises, connect with support resources

Clinical Case Example:

Patient Profile:

72-year-old male with 15-year history of type 2 diabetes, HbA1c 8.2%, presenting with falls and weakness.

Assessment Findings:

- SARC-F score: 6 (positive screen)
- Hand-grip: 22 kg (low for age)
- Gait speed: 0.7 m/s (impaired)
- DXA: Appendicular lean mass 18.2 kg (low)

Diagnosis:

Severe sarcopenia with diabetic complications

Intervention:

- Resistance training 3×/week
- Protein 1.4 g/kg/day
- Vitamin D supplementation, Zn, Mg, Q10
- Optimized diabetes medications

6-Month Outcome:

Improved strength (+15%),
better glycemic control (HbA1c 7.1%),
no falls

INTEGRATED APPROACH:

- EXERCISE
- PROTEIN
- CHOOSE ANTIDIABETIC DRUGS FOR MUSCLE HEALTH
- SUPPLEMENT:

Section 10 – Summary and Conclusion

Acknowledgements and References

- Acknowledge collaborators, clinical advisors, and institutions
- Key references: EWGSOP2, ADA, EASD guidelines
- Additional reading on sarcopenia and DM2


Thank You for Your Attention