DIFFICULTIES OF DIAGNOSTICS IN DEBUT OF DIABETES MELLITUS

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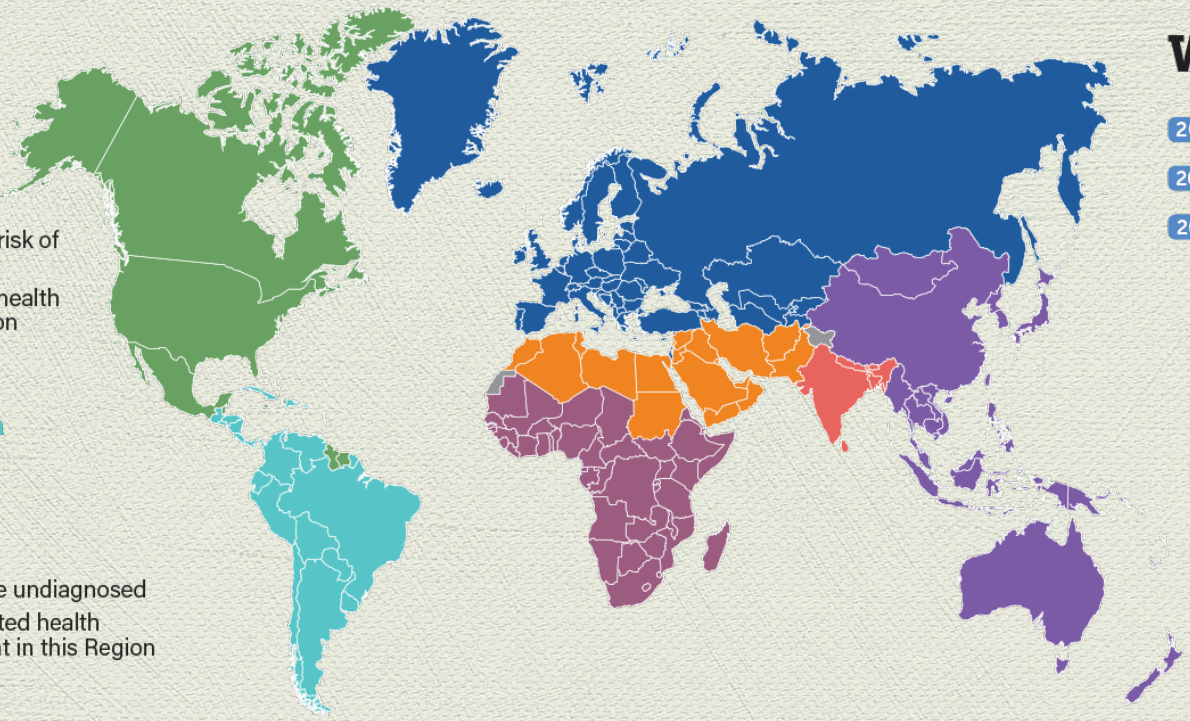
Why Diabetes?



North America & Caribbean

2045 63 million
2030 56 million
2019 48 million

↑ 33% increase

- 1 in 6 adults in this Region is at risk of type 2 diabetes
- 43% of global diabetes-related health expenditure occurs in this Region

South & Central America

2045 49 million
2030 40 million
2019 32 million

↑ 55% increase

- 2 in 5 people with diabetes were undiagnosed
- Only 9% of global diabetes-related health expenditure for diabetes is spent in this Region

WORLD

2045 700 million
2030 578 million
2019 463 million

↑ 51% increase

Europe

2045 68 million
2030 66 million
2019 59 million

↑ 15% increase

- 1 in 6 live births are affected by hyperglycaemia in pregnancy
- The Region has the highest number of children and adolescents (0–19 years) with type 1 diabetes – 297,000 in total

Africa

2045 47 million
2030 29 million
2019 19 million

↑ 143% increase

- 3 in 5 people with diabetes are undiagnosed
- 3 in 4 deaths due to diabetes were in people under the age of 60

Middle East & North Africa

2045 108 million
2030 76 million
2019 55 million

↑ 96% increase

- 1 in 8 people have diabetes
- 1 in 2 deaths due to diabetes were in people under the age of 60

South-East Asia

2045 153 million
2030 115 million
2019 88 million

↑ 74% increase

- 1 in 5 adults with diabetes lives in this Region
- 1 in 4 live births are affected by hyperglycaemia in pregnancy

Western Pacific

2045 212 million
2030 197 million
2019 163 million

↑ 31% increase

- 1 in 3 adults with diabetes lives in this Region
- 1 in 3 deaths due to diabetes occur in this Region

From- International Diabetes Federation, world atlas

Introduction

"DIAGNOSIS IS NOT THE END, BUT THE BEGINNING OF PRACTICE."

In medicine it is important to know the correct diagnosis in order to give the best possible treatment to the patient. In case of diabetes if people don't get properly diagnosed they often don't get the proper treatment. When making a diagnosis we try to use blood test, patient's physical characteristics and way that the disease progresses. We correlate all that together with what we most commonly see.

Someone who is thin and young, with a more severe onset along with ketoacidosis is a classic Type 1.

An older adult who is overweight with a gradual increase in glucose is probably a Type 2.

We always use these traits to determine the type of diabetes and often we are so much into textbook that we forget that maybe there are other types of Diabetes, well don't worry I am here today to introduce you guys to a rare form of diabetes called is LADA (aka type 1.5).

What is Type 1.5 (LADA) ?

DM-1	LADA	DM-2
β Cell destruction, leading to absolute insulin deficiency.	Initial β Cell destruction and then insulin deficiency develops.	β Cell dysfunction leading to reduced insulin production & obesity.
Autoimmune disorder	Autoimmune antibody present but insulin is not needed.	Not autoimmune disorder
Diagnosed in childhood or young age (<30 years).	Diagnosed in 35-45 years old.	Diagnosed in older age (>45 years) or in adults (obese).
Insulin dependent	Starts as insulin independent but becomes insulin dependent in 6 years.	Insulin independent (Usually)

Etiology and Epidemiology

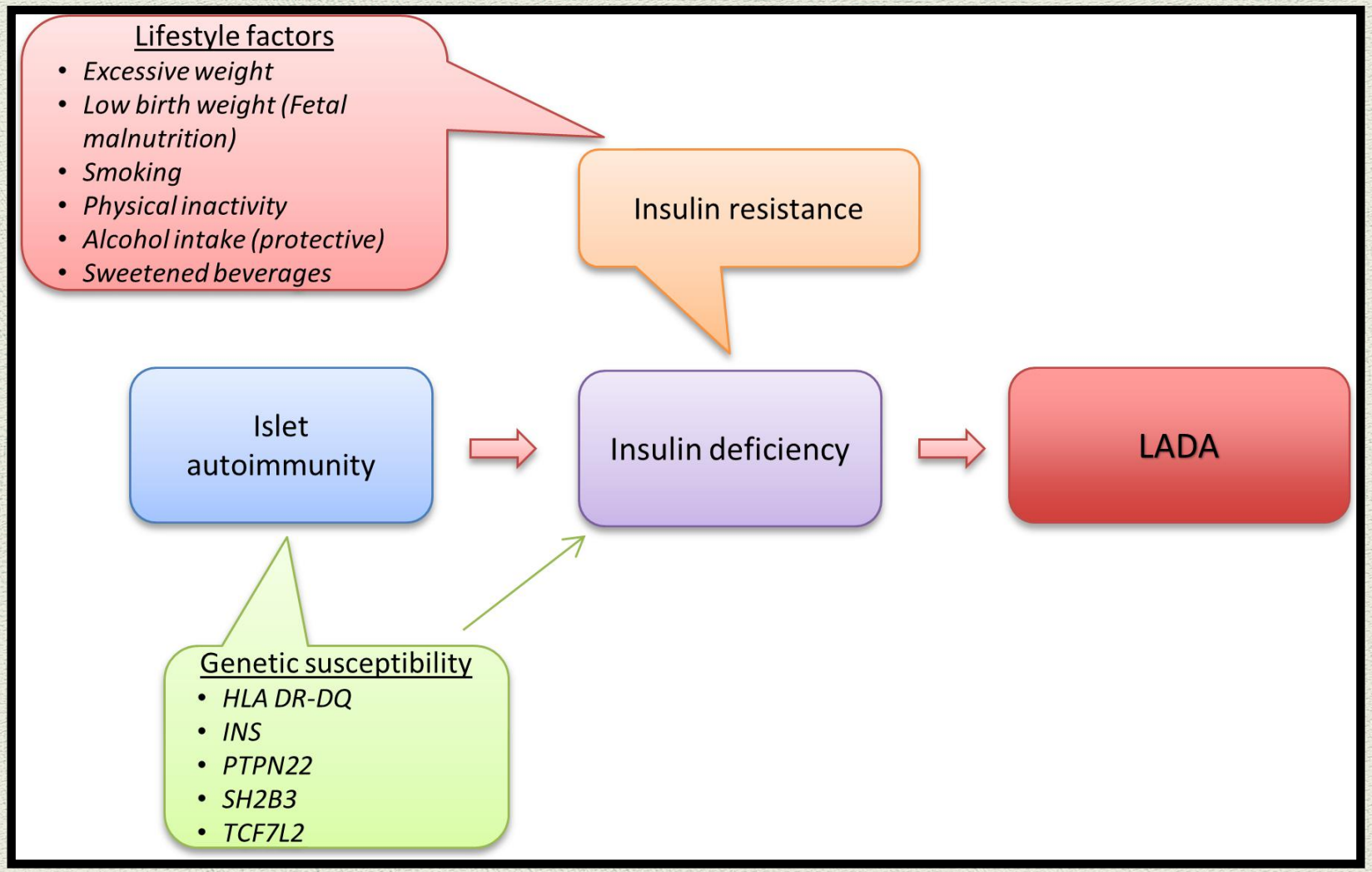
It is evident that LADA shares several lifestyle risk factors with DM-T2, namely, excess body weight, greater waist-hip ratio, low birth weight, intake of two or more sweetened beverages daily, and heavy smoking.

Approximately 5-20% people who are diagnosed with DM-T2 are actually LADA.

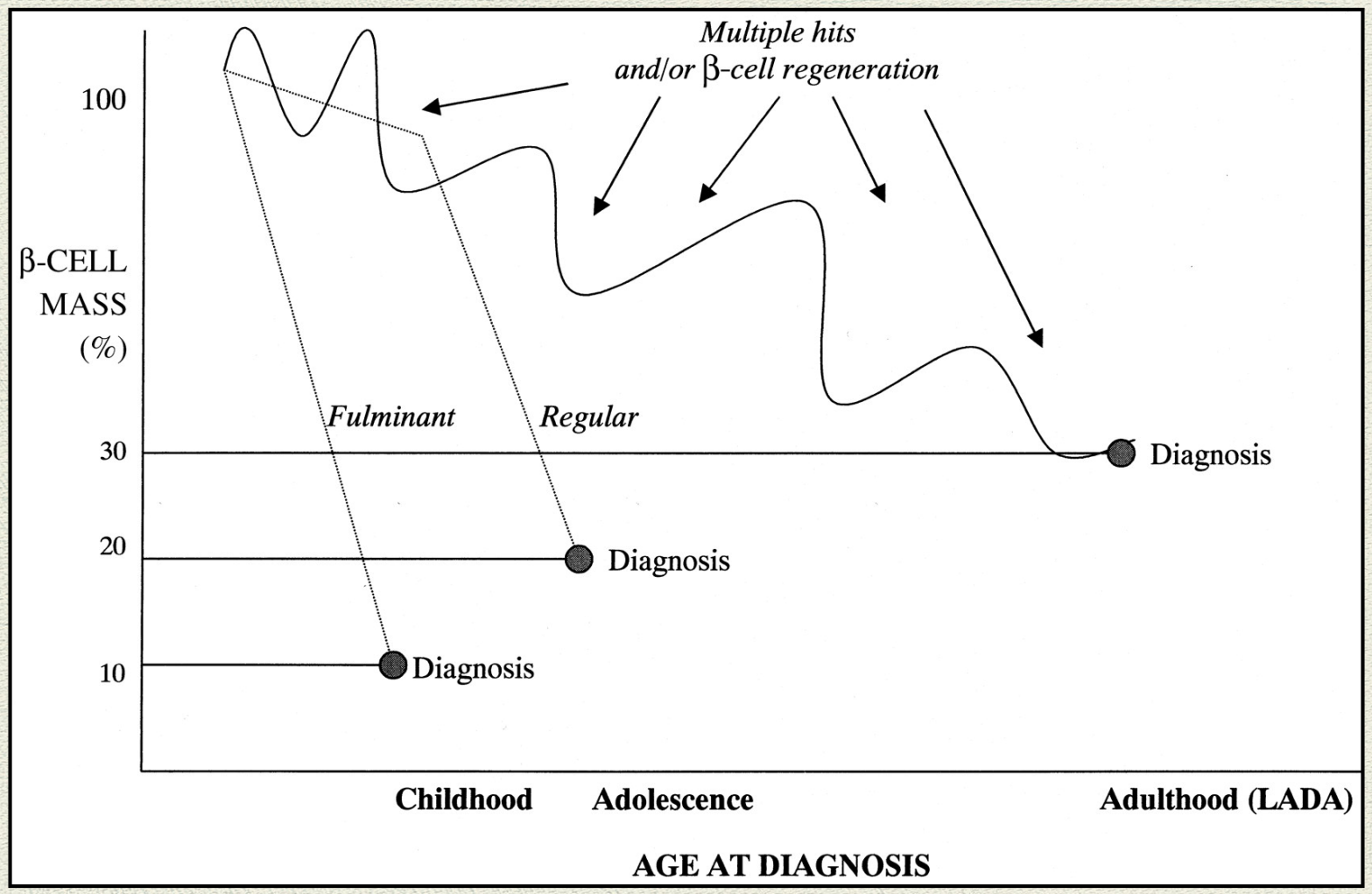
LADA is the most frequent form of adult-onset autoimmune DM. LADA study from Europe discloses that almost 10 % of 6000 adults with adult-onset DM had islet Beta cell autoantibodies.

From RASMUSSEN university

Pathophysiology



From- Frontier in Physiology



From- American Diabetes care association

History and Physical signs

- Patients with LADA may present with symptoms of polyuria, polydipsia, nocturia, fatigue, visual changes, tingling in the feet, and weight loss or may be asymptomatic.
- A personal or family history of autoimmune disease should raise suspicion that a patient with hyperglycemia has LADA or DM-T1.
- A history of low-birth-weight is a strong risk factor for both LADA and DM-T2.
- A history of smoking, alcohol consumption, the number of sweetened beverages are risk factors.
- Age less than 50 years.
- Symptomatic hyperglycemia, A fasting blood glucose 270 mg/dl or higher, HbA1C 10%
- BMI of less than 25 kg/m2.
- A personal or family history of autoimmune disease

Two or more criteria when positive yielded a sensitivity of 90 % and specificity of 71 % while less than 2 criteria virtually excluded LADA.

Patient Presentation

- Patient M
- 40 Years old
- Lives in Mariupol, Donetsk
- Married and a child
- Not working

Complaints

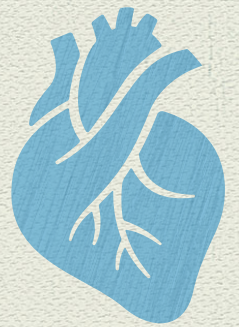
- Thirst

- Weakness

- Insomnia

- Edema of lower extremities

- Palpitation



- Decreased visual clarity

Anamnesis

- In January, the patient had an attack of Acute Pancreatitis.
- The 1st glycemic increase upto 12 mmol/L was observed in early 2018. Afterwards he was diagnosed with DM-T2 and oral sugar lowering therapy was started.
- During this time from January to May, 16 Kg weight loss was observed in the patient.
- In 2019 the patient went into ketoacidosis coma.
- Afterwards the patient began taking Insulin therapy and on admission he was injected with Pharmasulin H30/70.
- **Patient discontinued therapy in full way and was hospitalized due to failing of health.**

Objective

- Consciousness - clear
- State – moderate severe
- Body position – active
- Patient can orient himself in place, time, his personality is normal
- Weight -80 kg, height- 189 cm,BMI- 22
- Pale skin and mucosa
- Musculoskeletal system – No change
- BR (breathing rate) – 18 /min
- Lung percussion: No clinically significant changes
- Lung auscultation: Vesicular breathing

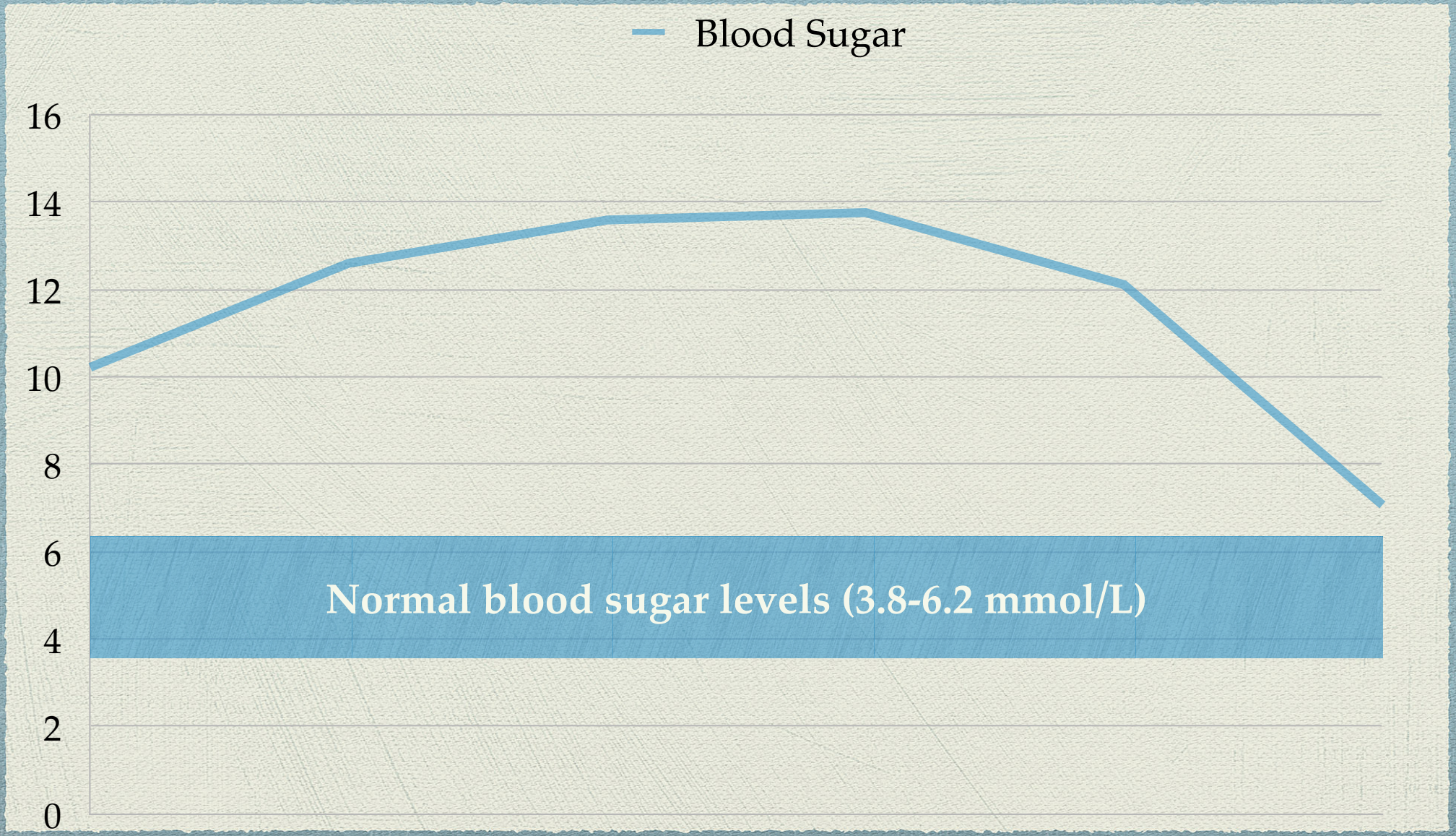
Lab results

Test	Result	Index
Hemoglobin	145	140-160 g/L
RBC count	4.3 x 10^12	4.2-5.9 x 10^12 cells/ L
Hb A1c	6.7%	4.6-6 %
Leukocytes	6 x 10^9	4-9 x 10^9 cells/L
MCV	0.85	0.85-1.05
ESR	32	1-10 mm/sec
PTT	28	24-34 sec
Fibrinogen	3.1	2-4 g
Fibrin	14	9-18 mg

Test	Result	Index
Cholesterol	4.84	3.62-6.21 mmol/L
Beta- Lipoprotein	58	35-55 units
Triglycerides	0.7	0.45-1.86 mmol/L
Cortisol	400	138-635
AST	21.4	0-35 U/L
ALT	17	0-35 U/L
Total blood protein	70	60-78 g/L
Prothrombin	97%	90-100 %
Fibrinolytic activity	240	180-300 min

- The patient refused the test for At- GAD.

Sugar Profile



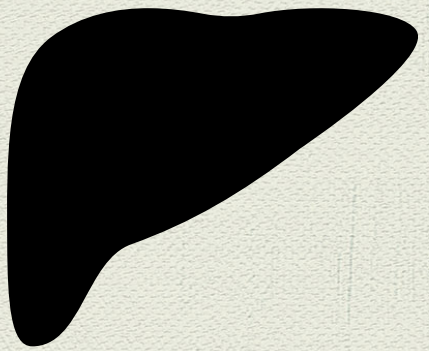
System Association

- EXCRETORY SYSTEM-

Test	Result	Index
Glucose in Urine	3	0-0.8 mmol/L
Filteration	155	60-150 ml/min

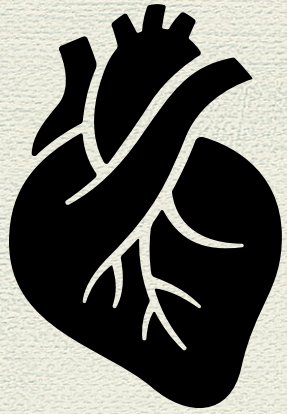
- On analysis of urine we observed Hyperfiltration & glycosuria.

- G.I.T.



- Ultrasound of GIT was conducted.
- Conclusion: No significant pathological changes were detected.

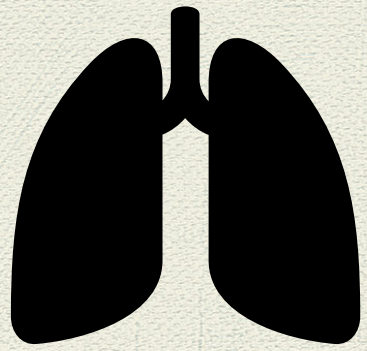
CARDIOVASCULAR SYSTEM



- BP on admission was 110/70.
- The heart was on sinus rhythm that is 62 beats/min.
- ECG reveals a normal heart and no pathology of the conduction system.
- Metabolic cardiomyopathy in combination with dysplasticity of ventricles was observed with preserved systolic function of the left ventricle, ejection fraction 65%.
- On Ultrasound we saw, mitral valve prolapse grade 1 with mitral regurgitation grade 1.

THYROID GLAND

- On palpation the area was not tender and no palpable nodules were felt. Hence no pathological changes were found.
- On ultrasound no significant pathology was seen.



CHEST X-RAY

- Chest x-ray shows no Focal, infiltrative shadows that are not defined.
- The roots are not expanded, structural integrity is maintained, the sinuses are free.
- No pathology was detected.

Consultations

- Ophthalmologist- No pathology
- Neurologist- Diabetic distal polyneuropathy
- Cardiologist- Metabolic and dysplastic cardiomyopathy

Treatment

1. **Insulin therapy**: Insulin Glulisine 6-8-8 units for the day & Insulin Glargine 20 units (For blood sugar)
2. Thioctic acid 600 mg 1 tab. (For diabetic neuropathy)
3. Torsemide 10 mg in the morning. (For diuresis)

From- MIMS drug info

Recommendation

1. Diet with restriction of fats and easily digestible carbohydrates, protein of animal origin.
2. Control of glycemia, glycosuria, blood pressure, weight.
3. Regular check up at endocrinologist, neuropathologist, cardiologist, ophthalmologist and nephrologist for control.

Conclusion

Anamnestic data in patients with the onset of diabetes mellitus are very important. In this clinical case, despite the lack of some laboratory tests due to the patient's refusal, we reserve the right to consider the LADA diagnosis as the most likely. The diagnosis of Type 1 Diabetes Mellitus in this case is not a mistake, since the therapeutic tactics will remain the same.

The understanding of LADA not only gives us better differential diagnosis but also insight to the world of Diabetes and other autoimmune disease & just makes us wonder how human body can be so,

inscrutable (That means impossible to understand or interpret)

"The question isn't how to get cured but how to **Live.**"

-Joseph Courad

-Thank You