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Case Report

Insulin-Dependent Diabetes Mellitus Type 1 Is Potentially Reversible, a Case Report

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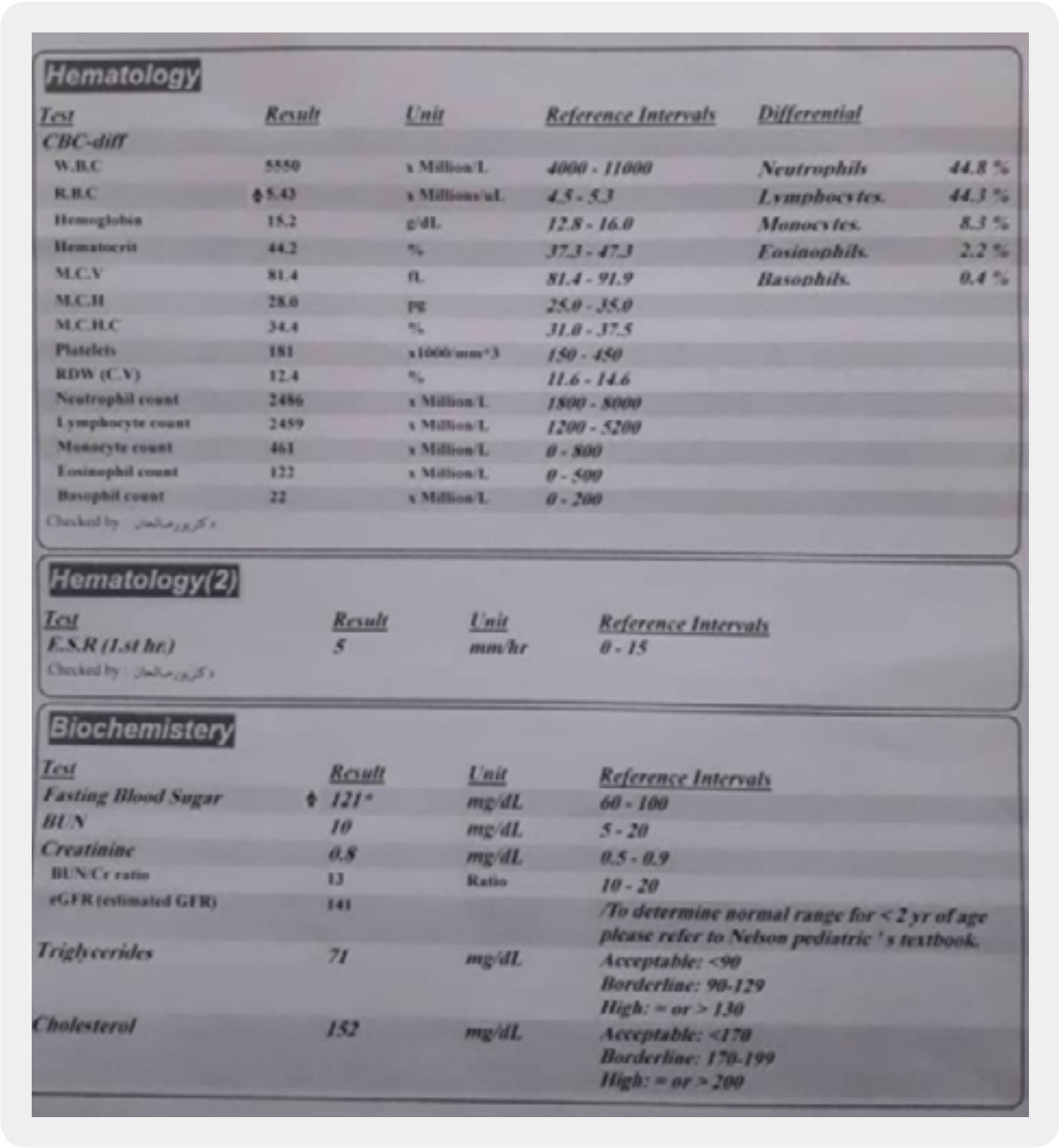
History

Here we report an exceptional case of a Persian young man 14 years old Mr. Ali.T who under sudden distress, Erythrocytosis (fig 1), increased HbA1C and glucose levels in the last three months (fig 1,2), possible H. Pylori infection and inflammation but normal fatty acids, Triglyceride, and Cholesterol levels (fig 3), got Diabetes Mellitus type 1 (DMT1) diagnosis (fig 1-3) and insulin-dependent treatments with 2 insulin injections subcutaneous daily (60 injections per month) with some symptomatic food allergies, visit our practice in May 2020, in Tehran, Iran.



Hematology

Test	Result	Unit	Reference Intervals	Differential	
CBC-diff					
W.B.C	5550	x Million/L	4000 - 11000	Neutrophils	44.8 %
R.B.C	↑5.43	x Millions/uL	4.5 - 5.3	Lymphocytes.	44.3 %
Hemoglobin	15.2	g/dL	12.8 - 16.0	Monocytes.	8.3 %
Hematocrit	44.2	%	37.3 - 47.3	Eosinophils.	2.2 %
M.C.V	81.4	fL	81.4 - 91.9	Basophils.	0.4 %
M.C.H	28.0	pg	25.0 - 35.0		
M.C.H.C	34.4	%	31.0 - 37.5		
Platelets	181	x1000/mm^3	150 - 450		
RDW (C.V)	12.4	%	11.6 - 14.6		
Neutrophil count	2486	x Million/L	1800 - 8000		
Lymphocyte count	2459	x Million/L	1200 - 5200		
Monocyte count	461	x Million/L	0 - 800		
Eosinophil count	122	x Million/L	0 - 500		
Basophil count	22	x Million/L	0 - 200		

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Hematology(2)

Test	Result	Unit	Reference Intervals
E.S.R (1st hr.)	5	mm/hr	0 - 15

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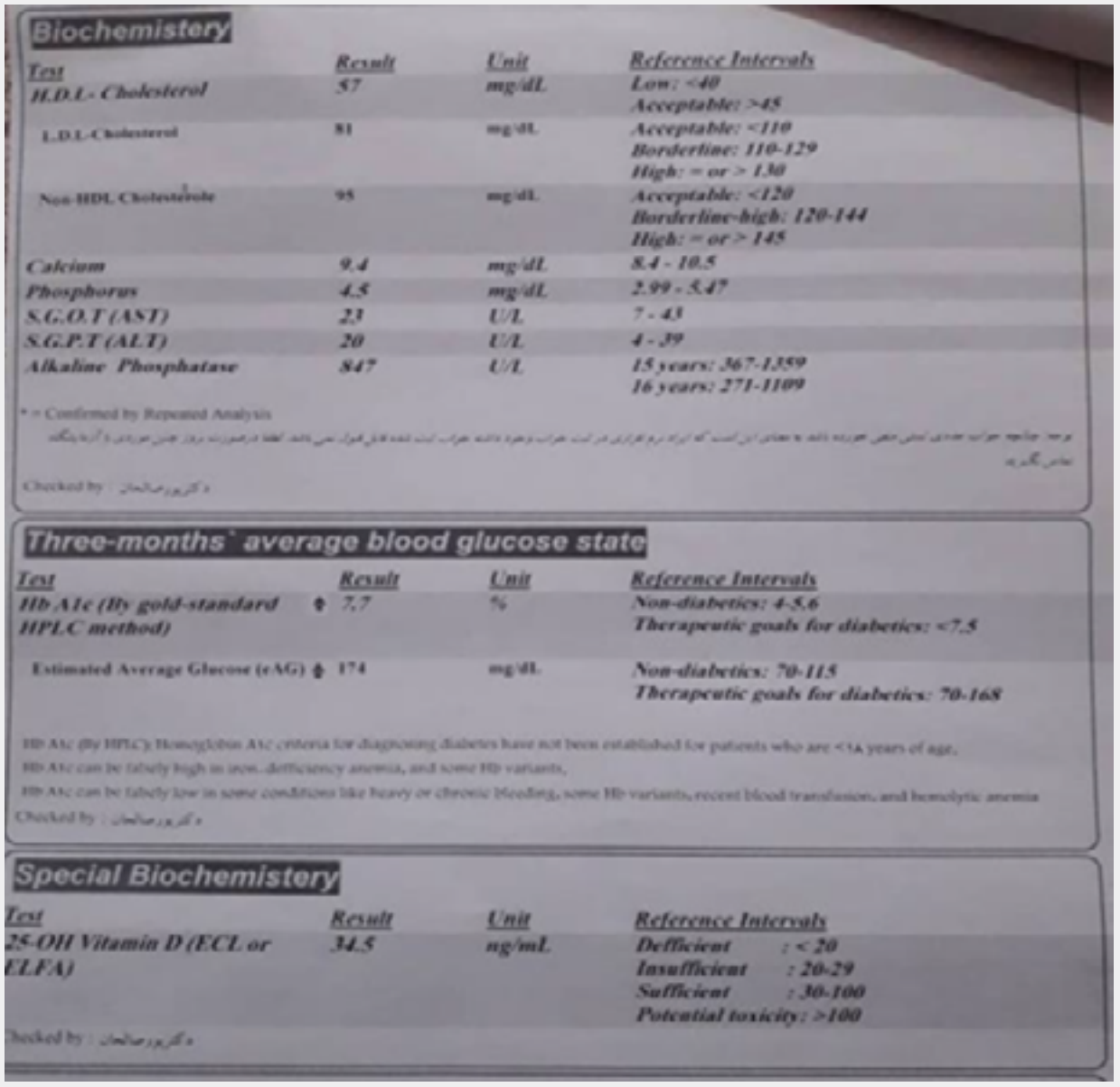
Biochemistery

Test	Result	Unit	Reference Intervals
Fasting Blood Sugar	↑ 121*	mg/dL	60 - 100
BUN	10	mg/dL	5 - 20
Creatinine	0.8	mg/dL	0.5 - 0.9
BUN/Cr ratio	13	Ratio	10 - 20
eGFR (estimated GFR)	141		/To determine normal range for < 2 yr of age please refer to Nelson pediatric ' s textbook.
Triglycerides	71	mg/dL	Acceptable: <90 Borderline: 90-129 High: = or > 130
Cholesterol	152	mg/dL	Acceptable: <170 Borderline: 170-199 High: = or > 200

Figure 1: *Serology, immunology, and hormone analysis tests showed potentially diabetes indications with an increase in fasting Glucose level, RBCs count but WBC, Hb, HCT, MCV, MCH, MCHC, platelets count, RDW, neutrophils count, lymphocytes, monocytes, eosinophils, basophils count were normal. Simultaneously the ESR, BUN, creatinine, Cholesterol, triglyceride levels were normal.*



Biochemistery

Test	Result	Unit	Reference Intervals
H.D.L- Cholesterol	57	mg/dL	Low: <40 Acceptable: >45
L.D.L-Cholesterol	81	mg/dL	Acceptable: <110 Borderline: 110-129 High: = or > 130
Non-HDL Cholesterole	95	mg/dL	Acceptable: <120 Borderline-high: 120-144 High: = or > 145
Calcium	9.4	mg/dL	8.4 - 10.5
Phosphorus	4.5	mg/dL	2.99 - 5.47
S.G.O.T (AST)	23	U/L	7 - 43
S.G.P.T (ALT)	20	U/L	4 - 39
Alkaline Phosphatase	847	U/L	15 years: 367-1359 16 years: 271-1109

* = Confirmed by Repeated Analysis

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Three-months' average blood glucose state

Test	Result	Unit	Reference Intervals
Hb A1c (By gold-standard HPLC method)	7.7	%	Non-diabetics: 4-5.6 Therapeutic goals for diabetics: <7.5
Estimated Average Glucose (eAG)	174	mg/dL	Non-diabetics: 70-115 Therapeutic goals for diabetics: 70-168

Hb A1c (By HPLC): Hemoglobin A1c criteria for diagnosing diabetes have not been established for patients who are <18 years of age.
Hb A1c can be falsely high in iron deficiency anemia, and some Hb variants.
Hb A1c can be falsely low in some conditions like heavy or chronic bleeding, some Hb variants, recent blood transfusion, and hemolytic anemia

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Special Biochemistery

Test	Result	Unit	Reference Intervals
25-OH Vitamin D (ECL or ELFA)	34.5	ng/mL	Deficient : < 20 Insufficient : 20-29 Sufficient : 30-100 Potential toxicity: >100

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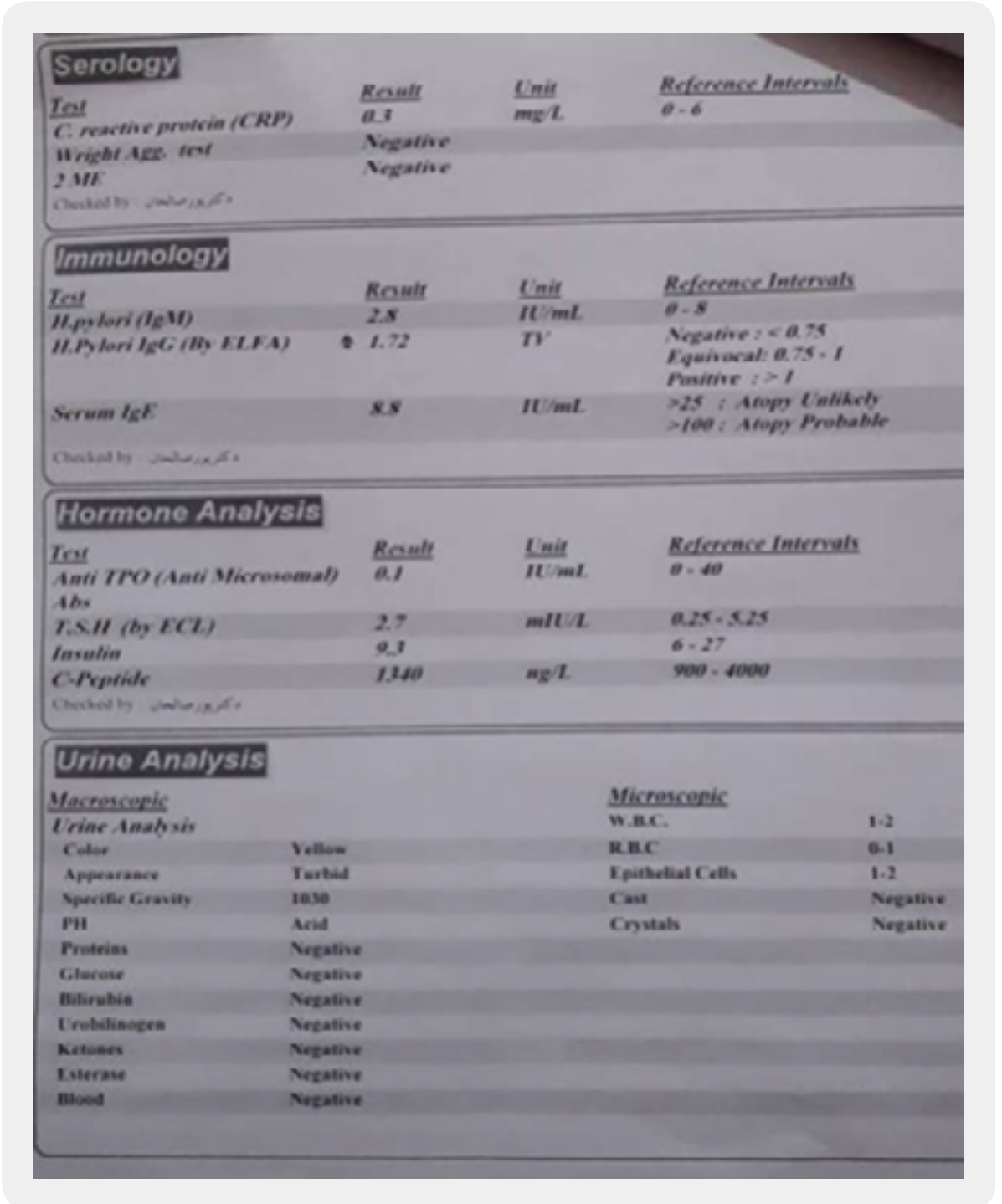
Figure 2: *Biochemistry measurements of (non-)HDL and LDL -Cholesterol, calcium, phosphorus, AST/ALT/ Alkaline phosphatase hepatic enzymes activity, vitamin D and fatty acid metabolism were normal. Though, the HbA1C and glucose tests showed increased levels in the last three months.*



Serology

Test	Result	Unit	Reference Intervals
C reactive protein (CRP)	0.3	mg/L	0 - 6
Wright Agg. test	Negative		
2 ME	Negative		

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Immunology

Test	Result	Unit	Reference Intervals
H.pylori (IgM)	2.8	IU/mL	0 - 8
H.Pylori IgG (By ELFA)	1.72	TV	Negative : < 0.75 Equivocal: 0.75 - 1 Positive : > 1
Serum IgE	8.8	IU/mL	>25 : Atopy Unlikely >100 : Atopy Probable

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Hormone Analysis

Test	Result	Unit	Reference Intervals
Anti TPO (Anti Microsomal) Abs	0.1	IU/mL	0 - 40
T.S.H (by ECL)	2.7	mIU/L	0.25 - 5.25
Insulin	9.3		6 - 27
C-Peptide	1340	ng/L	900 - 4000

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Urine Analysis

Macroscopic Urine Analysis		Microscopic	
Color	Yellow	W.B.C.	1-2
Appearance	Turbid	R.B.C	0-1
Specific Gravity	1030	Epithelial Cells	1-2
PH	Acid	Cast	Negative
Proteins	Negative	Crystals	Negative
Glucose	Negative		
Bilirubin	Negative		
Urobilinogen	Negative		
Ketones	Negative		
Esterase	Negative		
Blood	Negative		

Figure 3: *Immunologic analysis showed an increased H. Pylori-IgG but IgE was normal. Besides, the Insulin, C-peptide, and other hormones' levels, serology CRP, and urine analysis were normal.*

After our Medical team visited diagnostics and treatments (D&T), we did succeed to change our case's metabolism as such, which our D&Ts caused his whole insulin metabolism was changed, considerably. Our D&Ts previously changed another patient's metabolism and restored her complex cyanotic tetralogy of Fallot [1]. Besides, our case who DMT1 was confirmed, in less than 24 hours become independent of insulins' injections, however. Moreover, we did succeed to decrease his Insulin injections frequency, considerably.

Physical Examinations

A 14-years old Persian young thin man presented with DM1 disorders increased HbA1c and glucose in the last three months' checkups, potential gastrointestinal tracks and hematologic inflammation and H.Pilory chronic infection were generally chronic-related distressed with anxiety for treatments, and with glucose-and, not fatty acids 'metabolic syndrome showed increased systemic HbA1c levels in the last two months. Our SONAPS diagnostic and associated treatments and diet-related approaches showed remarkable signal transduction disorders in the Vagal nervous system associated with central nervous systems (CNS) and (co) related brain-gut-heart axis leakages.

Lesson Learned

Known is that DMT1 is an irreversible metabolic disease, which could potentially progress toward severe insulin-dependent metabolic disorders and an increase in morbidity and mortality rates. The Saliva amylase overproduction (originating from Saliva-Kidney-GIT axis) and associated side effects might chronically result in the DMT1 (ir-)reversibly [1-5]. Where does serum amylase come from? and what is the mechanism of amylase production/recirculation/clearance disorders? are not completely elucidated yet. Moreover, recent data are suggesting that the serum amylase concentration redirects the equilibrium between the rates of amylase entry into-, and removal from the systemic blood circulation [3-5]. The pancreas and salivary glands have amylase concentrations that are several orders of magnitude greater than that of any other normal tissue, and these two organs probably account for almost all of the serum amylase activity in normal persons [4-8].

Recall, the Pancreatic hyperamylasemia might be resulted from an offense to the pancreas, ranging from trivial cannulation of the pancreatic duct to severe pancreatitis [4-8]. Hyperamylasemia due to salivary-type isoamylase is observed in conditions involving the salivary glands, as we also reporting in our case here (fig 4). In addition, this type of hyperamylasemia occurs in conditions, in which there is no clinical evidence of salivary gland diseases, such as chronic alcoholism, postoperative states (particularly postcoronary bypass), lactic acidosis, anorexia nervosa or bulimia, and malignant neoplasms that secrete amylase [4-8]. Hyperamylasemia can also result from decreased metabolic clearance of amylase due to renal failure or macroamylasemia [4-8]. Moreover, in our case Mr. Ali T. now 15 years old showed healthy kidney and glomerular filtrations during last years' checkups.

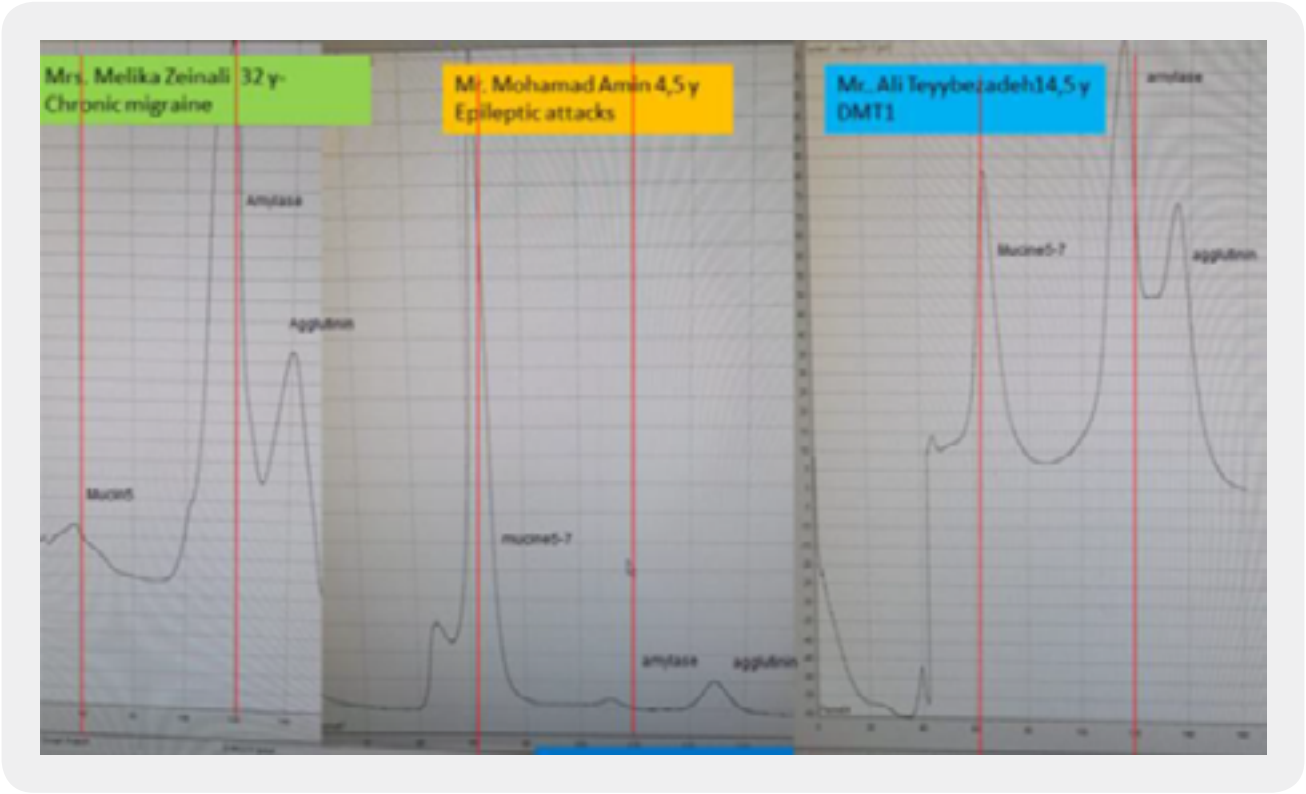

Figure 4: *Saliva proteomics of Mr. Ali T. (our case) compared to 2 different controls, who were used as negative controls for DMT1. Original data and their differences in saliva proteins i.e. Mucin 5, amylase, agglutinin expression are depicted and analyzed.*

Taken together, in our case either with combination of our in-house developed SONAPS as described [1], and/or with personalized diagnostics i.e allergic diet restrictions, adjustments of recommended appropriate diet supplements, personalized medical consult: not only significantly decreased the degree of Insulin injections frequency, but also did decrease his distressed condition, remarkably. Although, because of distance and limited access of patient to first degree Medicare and Medicaid he did fall back to insulin-dependent condition again, however. More in detail investigation in near future needed to unravel what we exactly done that made him insulin-independent.

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