**Case 1**

**Results:**

FT3:> 30.8 pmol / l, FT4: 98.9 pmol / l, TSH: <0.005 mIU / l, Antithyroglobulin: 153.3 kIU / l, Antithyreoperoxidase: 387 kIU / l, TRAK: 23.49 IU / l

**Thyroid sonography:**   
Right lobe: 19 x 16 x 61 mm. Left lobe: 19 x 18 x 63 mm. A marginally large thyroid gland with reduced echogenicity and a markedly small-deposited inhomogeneous structure has a significantly increased perfusion.

**Case 2**

**Results:**   
FT4: 3.7 pmol / l, TSH: 155.476 mIU / l, Antithyreoglobulin: 2298.5 kIU / l, Antithyreoperoxidase: 3797 kIU / l

**Thyroid sonography:**   
Thyroid small, parenchyma is inhomogeneous, echogenicity is reduced, perfusion is normal, mobility is maintained. The right lobe has a size of 8x8x32 mm, volume 1 ml. The left lobe has a size of 10x7x39 mm, ie 1.5 ml. Enlarged lymph nodes are not seen.  
Conclusion: Atrophic thyroiditis

**Case 3**

A 45-year-old patient comes for chest pain, coronarography with a finding of significant RIA stenosis and insignificant stenotic changes in other coronary arteries. The patient has a positive family history of premature manifestations of coronary heart disease. Physical findings include arcus lipoides cornae and bilateral xantelasmata of the eyelids.

**Results:**   
Total cholesterol 10.73 mmol / l, LDL-cholesterol 8.76 mmol / l, HDL-cholesterol 1.44 mmol / l, triglycerides 1.17 mmol / l.

**Case 4**

Minerals + Osmolality: Na: **127** [137..146], K: 3.9 [3.8..5.0], Cl: 100 [97..108], Ca: 2.16 [2.00. .2.75], P: **0.43** [0.65..1.61], Mg: 0.81 [0.70..1.00]

Nitrogen metabolites: Urea: 5.8 [2.8..8.0], Creat .: 90 [44..110]

Liver tests: Bilirubin: **22.1** [2.0..17.0], Bili ex: 4.5 [0.0..5.1], ALT: 0.49 [0.10..0, 78], AST: 0.37 [0.10..0.72], GGT: 0.39 [0.14..0.84], ALP: 1.66 [0.66..2.20]

Protein: Albumin: **26.1** [35.0..53.0], CB: 69.8 [65.0..85.0], CRP: **222.1** [0.0..5.0]

Lipids: Chol: **8.90** [2.90..5.00], TAG: **13.51** [0.45..1.70], HDL-chol: **0.21** [1.00..2.10 ], Cholesterol LDL: 1.81 [0.00..3.00], Atherogenicity index: **41.4** [0.0..4.2], Non-HDL calculation: **8.7** [0.0. .3.8]

Thyroid: TSH: 1,649 [0,500..4,900]

Diabetic profile: Glycemia: **13.2** [3.9..5.6], Glyc.HbA1c: **96.0** [20.0..42.0]

Antibodies: Anti IA2: <7.5 [0.0..7.5], Anti GAD: <5.00 [0.00..5.00], C peptide: 0.94000 [0.37000 .. 1.47000]

ABR: no sights

Urine chemically: pH: 6.0 [5.0..7.0], Density: **1.028** [1.015..1.025], Protein orient **.: 1.00** [0.00..0.30], Glycosuria**:> 56.0** [0.0..0.0], Ketone bodies: negative. [0,0..0,0], Bilirubin: neg, Urobilinogen: neg, Urinary erythrocytes: **100** [0..10], Leuko: neg, Nitrites: neg

Urine - sediment: Mucus: pos, ERY: **14,0** [0,0..5,0], LEUKO: 4,0 [0,0..10,0], Epit.pl: 5,0 [0, 0..15.0]

**Case 5**

Minerals + Osmolality: Na: 140; **135** [137..146], K: 3.8; 4.1 [3.8..5.0], Cl: 97; 103 [97..108], Ca: 2.23 [2.00..2.75], P: 0.69 [0.65..1.61], Fe: **37.4** [6.6. .28.0]

Nitrogen metabolites: Urea: 6.2 [2.0..6.7], Creat .: 99 [44..104]

Liver tests: Bilirubin**: 37.6** [2.0..17.0], Bili ex: **9.2** [0.0..5.1], ALT: 0.31 [0.10..0, 78], AST: 0.33 [0.10..0.72], GGT: 0.26 [0.14..0.68], ALP: 0.87 [0.66..2.20]

Protein: CB: 76.7 [65.0..85.0], CRP: <1.0 [0.0..5.0]

Thyroid: FT4: 20.3 [11.5..22.7], TSH: **0.181** [0.500..4.900]

Diabetic profile: Glycemia: **19.0** [3.9..5.6], Glyc.HbA1c: **96.0** [20.0..42.0]

Antibodies: Anti IA2: **7.1** [0.0..1.1], Anti GAD: **2.30** [0.00..1.45], INS: **2.41** [2.60..24.90 ], C peptide: **0.15000** [0.37000..1.47000], Anti AAI (free): 4.7 [0.0..5.5]

ABR: Lactate: 1.30 [0.50..2.00], pH: **7.246 [**7.350..7.440], pCO2: **4.33** [4.70..6.00], Act. Bicarbonate: 13, 6 [22,0..26,0], Standard bicarbonate: **14,2** [22,0..26,0], Current excess current: **-12,5** [-2,0..2,0], pO2: **4.6** [9.9..13.5], sat.O2: 53.9 [94.0..99.0], tot.CO2: **12.3** [23.0..27.0 ],

Blood count: no sights

Urine chemically: pH: 6.0 [5.0..7.0], Density: **1.042** [1.015..1.025], Protein orient .: neg, Glycosuria:> **56.0** [0.0..0.0 ], Ketone bodies: **4,0** [0,0..0,0], Bilirubin: neg, Urobilinogen: neg, Urinary erythrocytes: **75** [0..10], Leuko: neg, Nitrites: neg

Urine - sediment: Mucus: pos, ERY: **13,0** [0,0..10,0], LEUKO: **22,0** [0,0..20,0], Epit.pl: **11,0** [0, 0..10,0], Epit.k: <1,0 [0,0..3,0]

**Explanation No. 1:**

FT3:> 30.8 pmol / l [3.4..6.3], FT4: 98.9 pmol / l [10.0..18.7], TSH: <0.005 mIU / l [0.500 .. 4,900], Antithyreoglobulin: 153.3 kIU / l [0.0..60.0], Antithyreoperoxidase: 387 kIU / l [0..60], TRAK: 23.49 IU / l [0.00..1 , 75]

**The findings of hormonal examination show hyperthyroidism, positivity of TRAK (antibodies against TSH receptor) indicates Graves-Basedow disease.**

Ultrasound findings of a patient with Graves-Basedow is attached.

Thyroid sonography: Right lobe: 19 x 16 x 61 mm. Left lobe: 19 x 18 x 63 mm. A marginally large thyroid gland with reduced echogenicity and a markedly small-deposited inhomogeneous structure has a significantly increased perfusion.

**Explanation No.2:**

FT4: 3.7 pmol / l [11.5..22.7], TSH: 155.476 mIU / l [0.500..4.900], Antithyroglobulin: 2298.5 kIU / l [0.0..60.0] , Antithyreoperoxidase: 3797 kIU / l [0..60]

**The findings of hormonal examination show peripheral hypothyroidism, the positivity of antibodies against thyroglobulin and thyroid peroxidase indicate chronic lymphocytic thyroiditis.**

Ultrasound findings of a patient with chronic lymphocytic thyroiditis.

Thyroid sonography: Thyroid small, parenchyma is inhomogeneous, echogenicity is reduced, perfusion is normal, motility is maintained. The right lobe has a size of 8x8x32 mm, ie 1 ml. The left lobe has a size of 10x7x39 mm, volume 1.5 ml. I can't see enlarged lymph nodes.  
Conclusion: Atrophic thyroiditis

**Explanation No. 3:**

The patient is heterozygous for familial hypercholesterolemia caused by a mutation in the LDL-receptor gene. As a result, the catabolism of LDL particles is reduced, total and LDL cholesterolemia increase, and the risk of premature manifestation of atherothrombotic vascular complications increases dramatically. It is an autosomal dominantly transmitted disease, so it is necessary to examine family members!

**Explanation No.4:**

First manifestation of Type 2 diabetes in septic state (CRP), a male patientbwas born 1991, obesity

- Mineral imbalance

- Significant mixed dyslipidemia associated with insulin resistance and decontrolled diabetes

- High fasting glucose and very high HbA1c, glycosuria at the same time

- Both antiIA2 and antiGAD negative antibodies do not support diagnosis of Type 1 diabetes, similar as other manifestations (obesity, dyslipidemia)

**Explanation No. 5:**

First manifestation of Type 1 diabetes, a female patient was born 1980, normal habitus

- High fasting glucose and very high HbA1c, glycosuria at the same time

- The positivity of antiIA2, antiGAD, low insulin and C-peptide antibodies support diagnosis of Type 1 diabetes

- This is also supported by the presence of metabolic (keto) acidosis with ketone bodies in urine