**Patient K. O., female, 37 y/o**. Patient was healthy up to 33 years. At this age she underwent a surgery with following oncological treatment due to malignant melanoma. She was treated with interferon α‑2a (Roferon). In this time the patient was given temporarily levothyroxine, reportedly for milder hypothyroidism. At the age of 36 years metastasis to lymph nodes and spine occurred and she was given further treatment (radiotherapy and chemotherapy). Markers of thyroid gland function (TSH and FT4) were normal. Five months prior to our examination infusion therapy was started with pembrolizumabe administered once in three weeks and values of TSH and FT4 were monitored regularly. Due to long term lasting findings of TSH values elevation, patient came was presented to endocrinological examination. She reported only fatigue, bur her condition still enabled to carry out function of a manager. There was edema of face and acral parts, voice was deeper, hair thin, no goitre. Documentation shown that 5 weeks after the first administration of pembrolizumabe elevation of FT4 and drop in TSH concentration occurred. In the next weeks hypothyroidisms developed with elevation in TSH and increase in FT4 values. Antibodies TPOAb and TgAb were permanently negative. Ultrasound examination of thyroid gland spoke for chronic inflammation (thyroid gland reduced, with decreased echogenicity and non-homogenous structure, right lobe transversally 8 mm × 7 mm, left lobe 10 mm × 12 mm with finding of bordered hypoechogenic node - 12 mm in the right lobe).

* TgAb – antibody against thyreoglobulin (< 60 kIU/l)
* TPOAb – antibody against thyroid peroxidase (< 60 kIU/l)

**Questions:** Have cytokines or monoclonal antibodies an effect on thyroid gland function? What will be the next therapeutic approach?