

thyroid cancer, but it is also approved for unresectable hepatocellular carcinoma (HCC). Side effects such as fatigue, hypertension, palmar-plantar erythrodysesthesia, diarrhea, decreased appetite, and hypothyroidism are frequently reported adverse effects. However, the incidence of hyperthyroidism is a less known phenomenon. This case describes a patient with HCC on lenvatinib therapy who develops destructive thyroiditis. A 69-year-old man with hepatitis c cirrhosis complicated by progressive hepatocellular carcinoma (Child-Pugh class A6) presented with generalized weakness, unintentional weight loss, heat intolerance, palpitations, and tremors. He had been started on chemotherapy with lenvatinib 12 mg/day four weeks prior to presentation. Endocrinology was consulted due to abnormal thyroid function tests (TFTs). He had no previous history of thyroid function abnormalities or family history of thyroid disease. Laboratory tests revealed a hyperthyroid state [total thyroxine (T4), 14.3 µg/dL (normal range 4.5-11.7); free thyroxine (FT4), 1.9 ng/dL (normal range 0.9-1.7); thyroid-stimulating hormone (TSH), 0.07 µIU/mL (normal range 0.27-4.20), and negativity for antibodies [anti-thyroid peroxidase antibody (TPO Ab), 0.8 IU/mL (normal range 0.0-9.0); thyroglobulin antibody (Tg Ab), < 0.9 IU/mL (normal range 0.0-4.0); thyroid stimulating immunoglobulin (TSI), 90% (normal range < 122)]. Ultrasonography revealed a mildly prominent thyroid gland, a homogenous echo texture, and no suspicious thyroid nodules. In addition, a 99m-technetium (99mTc) scintigraphy demonstrated reduced radioactive uptake that was consistent with thyroiditis. Therefore, this patient was diagnosed with lenvatinib-induced destructive thyroiditis. Palpitations improved with a beta-blocker and the patient was resumed on a lower dose of lenvatinib, 8 mg/day, one week later. About six weeks after the initial dose of lenvatinib, his TFT results normalized. Lenvatinib disrupts cell proliferation which can affect many organs, including the thyroid gland. Although several theories have been proposed, the exact underlying mechanism by which this occurs remains unknown. It is important to note that in addition to hypothyroidism, lenvatinib may also cause hyperthyroidism in the form of a transient destructive thyroiditis. This emphasizes the need to routinely check TFTs prior to the initiation of lenvatinib and throughout the course of therapy.

Thyroid

THYROID DISORDERS CASE REPORT

Low-Dose Prednisone Therapy is Efficacious in Treating Painful Subacute Thyroiditis

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Subacute thyroiditis (SAT) usually presents with neck pain, radiating to ears and is often associated with hyperthyroidism. Currently the available treatment involves administration of NSAID or in more symptomatic patients

prednisone 40mg daily tapered over 6 weeks or longer. We report successful treatment of 3 patients (Pts) with SAT with low-dose prednisone therapy (20mg/day) (LDP20) initially and tapered over 4 weeks. **Patient 1:** A 32-year-old female presented with severe neck pain radiating to both ears and low-grade fever of 2-weeks duration. Two weeks prior, patient had cold-like symptoms lasting for 3 days. Physical examination: HR 110bpm, tremors of fingers noted, tenderness of the anterior neck present, thyroid 30-gms in size. Labs: ESR 92 mm/hr, CRP 3.2 mg/dL, TSH <0.005 uIU/mL, free T4 2.71 ng/dL, total T3 168 ng/mL. Thyroid scan and uptake showed a 24-hrs uptake <1%, thyroid gland not visualized, consistent with SAT. Patient was treated with atenolol and LDP20 tapered over 4 weeks. Pain significantly improved after 2 days of treatment. Six weeks later TSH was 0.9 uIU/mL with a free T4 1.4 ng/dL and ESR 8 mm/hr. **Patient 2:** A 19-year-old female presented with left-ear pain, anterior neck pain, fever, and extreme fatigue. PE: HR 111bpm, heat shield present, tender-to-palpation thyroid, brisk DTR. Lab: CBC normal, ESR 98 mm/hr, CRP 9.9 mg/dL, TSH <0.01 uIU/mL, free T4 3.8 ng/dL, total T3 210 ng/mL. Thyroid scan and uptake: uptake <1%, no thyroid gland visualized and SAT was diagnosed. Patient was started on LDP20 and atenolol. Four days following prednisone therapy her symptoms completely resolved and prednisone was tapered off over 4 weeks. Thyroid functions were normal by the seventh week. **Patient 3:** A 38-year-old male presented with fever, fatigue, severe neck pain, palpitation and a weight loss of 8 pounds. PE: HR 120 bpm, thyroid severely tender on palpation, brisk DTR. Lab: normal CBC, ESR 128 mm/hr, CRP 11.9 mg/dL, TSH <0.001 uIU/mL, free T4 4.2 ng/dL, total T3 201 ng/mL. Thyroid scan: thyroid gland not visualized and uptake was < 1%. SAT was diagnosed and patient was treated with propranolol and LDP20. After 5 days the dose of prednisone was reduced to 15mg/day and the prednisone was tapered over five weeks. Patient had resolution of symptoms in 70 hours and remained asymptomatic for the next 12 months of follow-up. Thyroid function normalized by the eighth week. **Conclusion:** SAT is a painful disabling thyroid disorder apparently caused by a viral infection; and NSAID or high-dose steroid treatment remains the standard of care. We have treated 3 Pts with relatively lower doses of prednisone than previously recommended and attained remission successfully. Thus side effects can be avoided with lower prednisone dose.

Thyroid

THYROID DISORDERS CASE REPORT

Lyme Disease: An Autoimmunity-Based “Destructive Thyroiditis” or Just Another “Non-Thyroidal Illness”?

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