As I said, most of the patients were diagnosed with hypothyroidism. Ever subclinical hypothyroidism due to autoimmune thyroid disease. The diagnosis can be a result of a screening, or can be driven by comorbidities. Say a patient who has type 1 diabetes or other autoimmune diseases, may be screened with a TSH. And the TSH accounts above normal range.

All the patient presents directly because of symptoms that he/she relates to hypothyroidism or the provider recognizes that those symptoms can be ascribed to hypothyroidism. So in the primary care setting the first screening test is TSH, which is a cheap, reliable, and easy to administer.

A TSH above normal range should prompt the question whether this is associated with a real thyroid pathology, or may be related to assays false, or interference. And next, once the annual-star is confirmed of an elevated TSH, at least in the thyroidology world we tend to confirm the histology of autoimmune thyroid disease, with the measurement of TPO, thyroid peroxidase antibodies.

There is a lots of chatter about following the title of TPO antibodies. Personally this is a very important diagnostic tool, it has nothing to do with prognosis in terms of association with comorbidities. And personally I do not follow title over antibodies to see any response to therapy or intervention. I do it once and then I move on.

Next question is what we do. Do we need to treat patients or not? Subclinical hypothyroidism defined as a TSH between upper range of normal all the way up to 10 micro units milliliter, is associated in large studies with an increased risk for comorbidities, particularly cardiovascular comorbidities. What has not been proven is that the treatment of hypothyroidism really causes an improvement in this risk profile.

So that brings the question, do we really want to treat the patients or not? And if we decide to treat the patients, what are we going to measure? Its saliva is not COVID. The decision to treat patients is usually driven by the combination of elevated TSH, and some symptoms of that patients may ascribe to hyperthyroidism, and they are very vague.

So, I would be very hard pressed not to find a patient that complains of fatigue, and maybe cold intolerance, and maybe some depression, maybe some weight gain, since the patient moved from being 20% years old to be 60 years old. And that's the problem in the sense that which one of these symptoms and signs is related to hypothyroidism, versus which one of the symptoms and signs are just the effects of growing older, and being less active?

In general, the treatment for hyperthyroidism are safe, so the threshold for treating is very low. And it makes sense as longer as we discuss with the patient the expectations, and we maintain the treatment in the safe range. So thyroid formulations are relative in narrow index, narrow therapeutic index. The means that we need to be careful in titrating the therapy, and in maintaining the thyroid hormone levels within normal range.

TSH is exceedingly sensitive to changes in thyroid hormone and in particular in T4 levels. So TSH is a perfect marker for dosing thyroid hormone and particular levothyroxine. At the same token, I would strongly advocate not to allow perceived therapeutic improvements as a reason to over treat patients, leading to a suppression of TSH.

We know from larger clinical series, all the way from the framing and study that suppress TSH is associated particularly in the elderly with a severe cardiovascular risk, and risk for fractures. And we really don't want to increase the burden of morbidity in our patients. So, once the decision of treating patients is started, it's very important to monitor the therapy, to discuss with the patient the modality of assumption of the drug, and the reminding patients that thyroid hormone formulations are exceedingly sensitive to interference in absorption.

So, everything that can affect the absorption of thyroid hormone needs to be discussed and monitored. And whatever the patient ends up doing, I tell the patient just keep doing what you're doing. Ideally thyroid hormone formulation should be taken first thing in the morning, with water only. And then every the patient waiting at least 30 minutes before breakfast.

Occasionally that's not possible. And if that's not possible, I ask the patient to keep doing what you're doing. We can always titrate the dose according to the interference with food or medications, but the patient needs to be aware that once we reach the target, the modality of assumption needs to be maintained the same.