## Of Self-Management of Chronic Care: A Trap for the Unwary!

Gholam Pishdad<sup>1</sup>, Reza Pishdad<sup>2</sup>, and Parisa Pishdad<sup>1</sup>

<sup>1</sup>Shiraz University of Medical Sciences Medical School <sup>2</sup>Rutgers School of Medicine

October 23, 2020

## Abstract

Self-management is an essential component of chronic care. In this regard, the patient, even if well-informed about their disease or condition, needs to appreciate the necessity of physician's supervision and shared decision-making.

Ms. M, a 30-year-old biology teacher presented with complaints of anorexia and weakness and was diagnosed with primary hypothyroidism. On levothyroxine treatment, her anorexia and weakness subsided and she felt well. And after her TSH and free T4 returned to a stable normal range, she was counselled that later on she should come for a visit anytime she had any symptoms or if she planned to get pregnant, otherwise, onceyearly endocrine visit would be good enough. Ms. M had read much about hypothyroidism, and thought she knew enough to manage her own disease. She continued to take her medicine regularly every morning almost 30-60 minutes before breakfast. So, 4 years passed and she had not gone for any follow-up visits. Every year Ms. M would refer to a private medical laboratory near her home to have her TSH checked, and it remained in a stable normal range. Now in a couple of months, it would be the 5<sup>th</sup> year that she was managing her hypothyroidism when she sensed that her anorexia and weakness were making a return. So as usual, on her own, and almost 1 month earlier than her every year, she referred to the same medical laboratory and wanted her TSH measured. She expected an elevated TSH. And, in fact, the laboratory report of a higher than normal TSH further enhanced her self-confidence. Based on her own knowledge, she concluded that she needed to increase the daily dose of her thyroid hormone to recheck a TSH in 2 months for further dose adjustment. But, just to beat that annoying weakness and anorexia sooner, she decided to temporarily-just for a few days-double the dose of her thyroid hormone and then cut back and continue with only 25% increase. To the best of her knowledge, in her case, there were not any contraindications for such a brief enhancement of thyroid hormone therapy: She was young enough and did not have any cardiovascular disease.

Just a few days after the doubling of the daily dose of levothyroxine, she needed an emergency hospitalization for a suspected acute abdomen. She had experienced a severe abdominal pain with intractable nausea and vomiting. She had a fever and her blood pressure was low with a significant orthostatic change. Her routine laboratory data among other abnormalities revealed a low sodium and elevated potassium. And a very low serum cortisol with highly elevated ACTH documented an Addisonian crisis. She responded to intravenous dextrose-saline and hydrocortisone and was discharged on prednisone and fludrocortisone to be followed by her endocrinologist. The endocrinologist noted black facial freckles, hyperpigmented oral mucosa and gingivae, and dark palmar creases. He adjusted the doses of her adrenal medicines, and, much to the patient's surprise, cut back on her thyroid hormone. Two months later Ms. M was doing well, had no complaints, and her lab data, including ACTH and TSH, were all normal. She had remained curious as to why her elevated TSH should return to normal with smaller doses of levothyroxine. The endocrinologist explained in simple terms that an emerging adrenal insufficiency with loss of cortisol feedback–and not a worsening hypothyroidism–was the cause of her recent mildly-elevated TSH. He continued that ironically this time, her anorexia and weakness were not related to her thyroid, but they were indeed the symptoms of that adrenal insufficiency. He added that the later-appearing fever, hypotension, and severe gastrointestinal symptoms were in fact the manifestations of a life-threatening adrenal crisis that had been triggered by that enhancement in her thyroid hormone dosage. He insinuated to her that she had failed to appreciate the importance of physician's supervision and shared decision-making .... He concluded, "In that situation, the adrenal insufficiency initially did not reveal itself fully but faked a mild hypothyroidism only to cheat you to open the door to a wolf in sheep's clothing!"

## References:

- Bornstein SR, Allolio B, Arlt W, Barthel A, Don-Wauchope A, Hammer GD, Husebye ES, Merke DP, Murad MH, Stratakis CA, Torpy DJ. Diagnosis and Treatment of Primary Adrenal Insufficiency: An Endocrine Society Clinical Practice Guideline. J Clin Endocrinol Metab. 2016; 101: 364-389.
- Barnett AH, Donald RA, Espiner EA. High concentrations of thyroid-stimulating hormone in untreated glucocorticoid deficiency: indication of primary hypothyroidism? Br Med J (Clin Res Ed). 1982; 285:172-173.
- Rosario PW, Mourão GF, Calsolari MR. Is confirmed elevation of the serum TSH with normal concentrations of circulating thyroid hormones sufficient for the diagnosis of subclinical hypothyroidism? *Eur Thyroid J* 2015; 4: 273-274.
- 4. Pishdad GR, Pishdad R, Pishdad P. From an out-of-date experience. Endocrine. 2020; 69:464-465.
- Rabi DM, Kunneman M, Montori VM. When Guidelines Recommend Shared Decision-making. JAMA. 2020; 323: 1345-1346.