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Case Report
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Exacerbation of Hashimoto's Thyroiditis and Onset of Fibromyalgia as Possible Components of Post-COVID-19 Syndrome: A Case Report

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Abstract

Background: Post-COVID-19 syndrome, an ill-defined sequelae of Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) infection, and its diagnostic symptoms are routinely being investigated and updated, but currently comprise a wide array of manifestations. Per currently available literature, most frequently reported presentations include fatigue, chronic dyspnea, cognitive impairments, and gastrointestinal upset. To date, no symptoms indicative of thyroid disease or fibromyalgia have been reports as constituents of post-COVID-19 syndrome.

Case Report: We present a case of persistently abnormal thyroid function tests despite appropriate treatment and the development of symptoms consistent with fibromyalgia following a COVID-19 infection in a 44-year-old female with a past medical history of Hashimoto's thyroiditis, alcohol use disorder, depression, and obesity. She reported continuous sinus pressure and congestion, fatigue, cognitive impairments, gastrointestinal upset and discomfort, shortness of breath, headache, hematuria, arthralgias and myalgias, and worsening depression that progressively intensified over three years following her initial COVID-19 infection. Despite multiple emergency department and primary care visits, a definitive cause of her symptoms was not determined; however, following consultation with internal medicine, a post-COVID-19 syndrome was suspected.

Conclusion: Thyroid disease and fibromyalgia have not been identified as chronic ramifications of SARS-CoV-2 infection, though long-term consequences of a COVID-19 infection are presently being investigated and apprised. While a conclusive diagnosis of post-COVID-19 syndrome cannot be made for our subject, we suspect her exacerbation of Hashimoto's thyroiditis and her other tenacious, ill-defined fibromyalgia-like symptoms to be sequelae of her initial 2020 COVID-19 infection.

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Introduction

Numerous short-term and long-term complications of Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2), the disease process responsible for the Coronavirus 19 (COVID-19) pandemic, are being investigated as components that may comprise what is being termed "post-COVID-19 syndrome." While this illness remains poorly understood, the most reported consequences following infection with COVID-19 include fatigue and dyspnea that lasts for months, potentially years, after the initial infection [1]. Various other persistent symptoms have also been noted, including cognitive impairments, typical and atypical angina, arthralgias and myalgias, palpitations and other cardiac disorders, olfactory and gustatory deficits, headache, cough, and gastrointestinal upset [1]. Additional long-term disease outcomes, such as arthritis, dysautonomia, thrombotic microangiopathy, trigeminal neuritis, and dermatomyositis, have also been reported [2-6]. Other reports have identified specific syndromes, including Guillain-Barré Syndrome, Sweet's syndrome, Multisystem Inflammatory syndrome, and Opsoclonus-Myoclonus syndrome, as possible reverberations [7-9].

Post-COVID-19 syndrome has been shown to affect COVID-19 survivors of all disease severities, including individuals who never required hospitalization or supplemental oxygen, and age groups, including infants, children, and young, middle-aged, and elderly adults [1,10]. In spite of the variegated demographic that post-COVID-19 syndrome appears to affect, female sex, greater than five early symptoms, early dyspnea, prior psychiatric disorders, and specific biomarkers (*e.g.* D-dimer, CRP, and lymphocyte count), have been identified as potential risk factors for the

development of this post-viral syndrome [1,11,12].

Many of these associated risk factors for post-COVID-19 syndrome are consistent with the profile of this case report's subject: A 44-year-old female, with a history of major depressive disorder and alcohol use disorder, developed COVID-19 with early dyspnea, fever, chills, cough, headache, gastrointestinal upset, and sinus congestion in April of 2020. Our subject meets four of the five noted risk factors–female sex, prior psychiatric disorders, early dyspnea, and greater than five early symptoms–recognized by the Yong study, with uncertainty regarding the fifth factor (presence of specific biomarkers) [1]. While our subject may possess some prototypical characteristics for the development of post-COVID-19 syndrome, the symptoms she experienced in the three years following her 2020 COVID-19 infection have yet to be extensively explored or detailed as potential long-term outcomes of an acute SARS-CoV-2 infection: Exacerbations of Hashimoto's Thyroiditis and possible evolution of fibromyalgia.

Case Presentation

We describe a case of a persistent, marked exacerbation of suboptimal thyroid function tests and the development of symptoms consistent with fibromyalgia after a SARS-CoV-2 infection in a 44-year-old female with a history of Hashimoto's thyroiditis, alcohol use disorder, depression, and obesity. Our subject was diagnosed with an active COVID-19 infection in April 2020 that involved dyspnea and constitutional upper respiratory infection symptoms including cough, fever, chills, headache, and sinus congestion, but was otherwise uncomplicated. She did not require supplemental oxygen or hospitalization during this acute illness and reports a spontaneous, self-limited recovery.

However, in October 2022, over two years after her initial SARS-CoV-2 infection, she presented to the Emergency Department (ED) with a "week and a half" long headache, frontal sinus pain and pressure, rhinorrhea, and depressive symptoms. She was diagnosed with acute sinusitis and was prescribed Augmentin and instructed to return to the ED if symptoms worsened. Two months following this ED visit, in December 2022, she returned to the ED with complaints of generalized malaise and fatigue in addition to nausea, body aches, polydipsia, and "foul-smelling" urine. Urinalysis was unremarkable but blood tests revealed significant abnormalities in thyroid function, most outstanding of which was a Thyroid Stimulating Hormone (TSH) level >100.00 uIU/mL, despite reported compliance with daily Levothyroxine Sodium 75 mcg. At this time, she was advised to follow-up with her primary care physician to discuss changes in medication dosage to control her hypothyroidism more optimally.

Three days after discharge, again in December 2022, our subject presented again to the ED with persevering fatigue, nausea, and "foul" smelling urine as well as new-onset polyuria, epigastric abdominal pain, and two episodes of non-bloody, nonbilious emesis. During this visit, she admitted to a relapse of alcohol use one week prior, and laboratory tests revealed elevated total bilirubin (1.5), aspartate aminotransferase (41), and TSH level, with TSH remaining >100.00 uIU/mL despite a normal T4 value. Urinalysis was again unremarkable during this presentation. Chest X-ray had revealed a potential pneumomediastinum, but a follow-up CT scan demonstrated no evidence for this and instead disclosed a small 6 mm calcified nodule in the upper lobe of the right lung. She was given 1 liter of Intravenous (IV) Lactated Ringer's solution, 1 gram of IV Tylenol, and 4 mg IV Zofran and was advised for a 6-month imaging follow-up evaluation of the lung nodule. Two days later, she presented to her primary care physician for follow-up and had noted an improvement of her symptoms, but her dose of Levothyroxine was not changed at this appointment.

Approximately one month later in January 2023, she presented to urgent care clinic with a five-day history of headache and sinus pain and pressure as well as persistent epigastric pain. COVID-19 home testing was negative at this time. During this visit, physical exam had revealed mild left flank tenderness but was otherwise noncontributory, and laboratory studies disclosed an elevated alkaline phosphatase (181 IU/L) and TSH (13.7 uIU/mL). Urinalysis demonstrated mild hematuria but was otherwise noncontributory. However, 6 days later, she once again presented to urgent care complaining of fatigue, nausea, periumbilical stomach pain, and mid to lower back pain. Urinalysis again revealed hematuria, so she was scheduled for a bilateral renal ultrasound in two weeks from the visit.

Five days prior to the ultrasound appointment, she presented again to ED with complaints of headache, dizziness, and maxillary sinus pressure. Laboratory results at this visit disclosed hyponatremia (135.0) and metabolic alkalosis (CO_2 20.0), and urinalysis again revealed trace hematuria. Her headache and dizziness improved after administration of meclizine, Tylenol, and ketorolac, and she was discharged. She returned to the ED three days later with previously reported symptoms and new-onset cough, fevers, vague, diffuse chest pain, and generalized malaise. Labs revealed correction of her hyponatremia but worsened alkalosis (CO_2 17.0), elevated alkaline phosphatase (158 IU/L), and TSH (10.40 uIU/mL) with normal free-T4. She again reported compliance with her 75-mcg Levothyroxine dose and noted no improvement in symptoms following administration of IV fluids, Tylenol, Toradol, and metoclopramide. Allergic sinusitis was suspected, and she was discharged on montelukast and fluticasone.

She again returned to the ED two days later, with much of the same symptoms and abnormalities in laboratory values, but this time with tachypnea and tachycardia. A CT of the abdomen and pelvis with contrast was ordered during this visit and revealed a moderate sized hiatal hernia but no evidence of other pathology. She was ultimately admitted to the hospital, where she received an Internal Medicine (IM) consult that revealed an extensive past workup in late 2021 and 2022 that had included an MRI of the brain, a cardiology consult, stress echocardiogram, and Zio patch cardiac monitoring, and a diagnostic home sleep study. The Zio path cardiac monitoring had disclosed accelerated ectopic ventricular rhythm and non-sustained ventricular tachycardia, and the sleep study results were consistent with severe obstructive sleep apnea. Despite this newly revealed past medical history, the consulting IM physician did not believe that "any of her confounding diagnoses explain[ed] her prolonged symptoms," and reported high suspicion of prolonged COVID syndrome. This physician had advised our subject to get evaluated at a post-COVID clinic and keep a diary of her symptoms as an attempt to identify her patterns of waxing and waning.

Our subject never presented to a post-COVID clinic, though she presented once again to her primary care provider in March 2023 for follow-up and with new complaints of arthralgia, myalgia, muscle weakness, a 25-pound weight loss, and left upper quadrant pain. She also complained of persistent trouble concentrating and dizziness. She had been seen by urology, rheumatology, and endocrinology

without major findings. Labs revealed mildly elevated CRP as well as continued elevated TSH level (8.30 uIU/mL) –with yet again reported compliance of levothyroxine 75 mcg – and elevated alkaline phosphatase (166 IU/L). Urinalysis again demonstrated hematuria. Her levothyroxine dose was increased to 88 mcg at this visit, and she was sent home without further workup.

She was then lost to follow up until presentation to our clinic in October 2023. At the time of her first visit, she had reported that she saw a different physician between March and October that–due to her persistent, disparate symptoms without an identifiable etiology–had diagnosed her with fibromyalgia. Medical records from this diagnosis were unable to be obtained. However, she had presented to our clinic with symptoms like those reported in March 2023, some of which are consistent with fibromyalgia–abdominal pain, myalgias, arthralgias, stiffness, fatigue, depression, memory and concentration issues, and headaches. Urinalysis at our visit had again revealed hematuria, another unspecific symptom occasionally found in fibromyalgia patients.16 Blood tests had also demonstrated a persistently elevated TSH value (93.1 uIU/mL) as well as low total T3 (45 ng/dL) and free T4 (0.4 ng/dL) and elevated alkaline phosphatase (133 U/L). She reported compliance with her Levothyroxine 88 mcg dose up until one week prior to the appointment, as she needed a new prescription.

Her physical exam at this visit demonstrated a positive Murphy's sign, and, due to her abdominal pain and interminably elevated alkaline phosphatase being suspicious for gallbladder disease, we referred her for an ultrasound of the right upper quadrant. Regardless of the outcome of the ultrasound, her other symptoms would likely not be explained by gallbladder disease alone, though we wanted to achieve better control of her thyroid function before establishing a formal diagnosis of fibromyalgia. Her levothyroxine dose was increased to 100 mcg, and status post compliance with this dose, we will reevaluate her blood tests and symptoms to assess for fibromyalgia.

Discussion

Because post-COVID-19 syndrome is still continuously being appraised and its definitive diagnosis is dynamic, we are unable to confidently determine that our subject's three-year span of various symptoms were components of a post-COVID-19 syndrome. However, it is important to note that our subject stated on multiple occasions that her symptoms all began after her infection with COVID-19 in April 2020, making her infection with COVID-19 suspicious for being the instigating factor. It is also of note that she experienced other symptoms that have been documented as components of post-COVID-19 syndrome: her chest CT scan revealed a pulmonary nodule, and post-COVID chest CT lung abnormalities have been identified as a frequent complication of a resolved COVID infection, as has worsening of depression, which she experienced [13,14].

Fatigue, generalized malaise, myalgias, arthralgias, headache, and abdominal pain have also been reported as common post-COVID-19 syndrome symptoms, and these were among the most common complaints our subject routinely reported at her ED and primary care visits [1,10,14,15]. Many of these nonspecific, generalized symptoms are also frequent complaints from patients with Hashimoto's thyroiditis as well as patients suffering from fibromyalgia, making it difficult to ascertain the exact pathogenesis of our subject's symptoms [16-18]. Furthermore, Hashimoto's thyroiditis has been documented to induce an autoimmune and inflammatory syndrome that may resemble fibromyalgia, thereby further complicating the determination of her symptoms' origin [19]. Nevertheless, as was previously brought to attention, her symptoms began after her 2020 infection of COVID-19 –so whether her presentation was simply an isolated exacerbation of Hashimoto's thyroiditis or an isolated development of fibromyalgia or a manifestation of the after-effects of a COVID-19 infection, we can surmise that her original COVID-19 infection contributed to her health decline.

All the above is stated under the presumption that our subject was honest in her reported compliance of her levothyroxine prescription–we cannot firmly verify her compliance, though she was regularly refilling her dose as prescribed. We are also presenting this case under the presumption that she was genuinely experiencing the symptoms that she noted and was not factious in her complaints as a means of some ulterior motive. On a similar matter, we cannot accurately ascertain that her reported symptoms are not all or in part components of a somatoform disorder, such as somatic symptom disorder, conversion disorder, illness anxiety disorder, factitious disorder, or malingering. Such somatoform disorders also present with assorted, nonspecific symptoms consistent with our subject's presentation and are often associated with other mental health disorders, which our subject has a known history of [20].

Conclusion

We cannot definitively say that SARS-CoV-2 was inherently involved in the development of our subject's persistently abnormal thyroid function tests and ill-defined, multifarious symptoms that are often associated with fibromyalgia. However, these symptoms began and progressively worsened over a three-year course following her initial COVID-19 infection, and she meets several criteria that have been correlated to post-COVID-19 syndrome. With no other provoking factor identified during her ailments, we believe the exacerbation of her Hashimoto's thyroiditis in the setting of appropriate treatment and the development of her myriads of fibromyalgia-like symptoms may be, at least in part, a result of a COVID-19-related chronic disease process.

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