კლინიკური შემთხვევა

SOMATIC SYMPTOM DISORDER AS A SURGICAL PROBLEM (CASE REPORT)

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Resiume

Somatic symptom disorder (SSD) is a psychiatric illness, with clinical manifestations that may mimic a surgical problem. Despite the overuse of numerous diagnostic modalities, it is not possible to find a responsible organic etiology of a patient's clinical complaints, which is why the surgeon may move to more invasive unnecessary interventions (eg, laparotomy). To avoid the latter problem, awareness about SSD, its diagnostic criteria, associations, and typical clinical presentation should be increased among surgeons as well as medical professionals of any other specialty. In this article, we report a case about an SSD patient admitted to a surgical department and discuss must-know details to help suspect and diagnose SSD.

A 58-year-old woman was hospitalized with spasmodic abdominal pain, with radiation to various abdominal and retrosternal areas. The patient's past medical history is significant for the resection of the sigmoid colon performed in 2019 when she was diagnosed with mega sigma. Despite the improved surgical intervention, the patient continued to have complaints of the same content, the cause of which could not be determined. After a series of visiting different doctors and nonsignificant laboratory and imaging results, the patient was consulted by a psychiatrist and diagnosed with an SSD.

Key points: Somatic symptom disorder, case report, Briquet's syndrome

INTRODUCTION

In 1959, French Pierre Briquet described patients with various somatic complaints, for which he could not find any medical basis and the condition was difficult to treat. Very soon, patients returned with the same or new, slightly changed complaints. For many years, this disorder was called Briquet's syndrome. Today, this condition is referred to as Somatic Symptom Disorder (SSD). SSD belongs to a mental condition defined as the presence of somatic symptoms not fully explained by any organic cause and the disproportionate thoughts, and behaviors related to them. The prevalence of SSD is estimated to be 6-7% in the general population in Europe as well as in the US with the female: male ratio of 10:1 [24, 12]. Considering the high prevalence and the diverse clinical manifestations mimicking an organic disorder of any body system, a medical professional of any specialty, including surgery, may encounter at least one case of SSD in their career [1]. Unrecognition of the SSD typically leads to healthcare overuse with possible iatrogenic complications and still no improvement in the patient's quality of life [1]. An analysis of individual patient data from nine community studies (total n >28,000 individuals) found that after controlling for potential confounding factors (eg, age, general medical illness, anxiety, and depression), a greater number of burdensome somatic symptoms were associated with greater health care use [13]. Another noteworthy association was identified between a delayed diagnosis of SSD and increased opioid prescription in patients presenting to the acute general surgical unit with pain as a somatic symptom. As the study found, early SSD diagnosis and adequate treatment administration may lower hospital admissions, presentations, and opioid prescriptions [26]. The recognition of SSD becomes especially troublesome when it affects the patient with a general medical condition, or suspicious findings in laboratory tests/imaging that may lead medical professionals to mistakenly attribute the complaints only to diagnostically identified abnormality. Eventually, this mistake may result in the treatment of the identified organic abnormality without managing the SSD - meaning no significant improvement in the patient's complaints and quality of life. In this paper, we discuss a clinical case of a patient presenting to a surgical department with a somatic symptom and a diagnostic challenge due to the presence of a general medical condition.

CASE DESCRIPTION

A 58-year-old female patient was admitted to the emergency surgery clinic with severe recurrent squeezing abdominal pain in the left lower quadrant for the last several years. She denied nausea/vomiting, constipation/diarrhea, melena/hematochezia. The pain radiated to multiple sites including the whole abdomen, back, and retrosternum.

In 2019 the patient was hospitalized for a similar pain associated with constipation, mega sigma was identified (therefore the pain was attributed to mega sigma) and treated surgically (sigmoid colon was resected) without any complications and normal postoperative period. 1.5 years after the surgery she developed a postoperative incisional ventral hernia that was clinically presented with an anterior abdominal bulge with a similar left lower

quadrant discomfort and pain. Successful hernioplasty was done with a mesh placement. Although the hernia repair was done 1.5 years ago she still uses an abdominal bandage as she feels safer and more comfortable with it. After the surgery, the pain persisted with the same recurrent pattern. The patient has undergone multiple diagnostic procedures during the last several years including abdominal CT, colonoscopy with biopsy, and numerous abdominal/pelvic ultrasounds. Focal thickening of the colonic wall was found on CT with nonsignificant lymphocytic infiltrate on biopsy, features of colitis, but the severity and diffuse pain radiation sites she experiences could not only be explained by these findings and diagnosis of colitis.

The patient also recalled the onset of anxiety attacks 2-3 months ago; Her first severe anxiety attack was accompanied by hypertension, tachycardia, paresthesias, and an unusual feeling in her bilateral upper extremities ("felt like something was eating my hands" - describing severe paresthesias); She was hospitalized, head CT was done without any abnormal findings. Around the same period, she had the most severe episode of abdominal pain with the same pattern mentioned above. After the consultation with the neurologist, she was prescribed anxiolytic Buspirone but due to severe side effects, it was changed to another anxiolytic/anticonvulsant Etifoxine 50mg twice a day to prevent anxiety attacks. The patient felt better with the medication but the abdominal pain was still disturbing. During the consultation with the psychiatrist, she also mentioned a swinging mood, easy irritability, and fears over minimal issues for the last few years. Despite the normal duration, she complained of low quality of sleep due to frequent nightmares.

She has a history of surgically corrected sialolithiasis, resected fibroadenoma of the breast, and hysterectomy with unilateral salpingo-oophorectomy due to leiomyoma.

Physical examination of the patient showed mild tenderness in the LLQ without rebound tenderness, and negative Rovsing, Psoas, Murphy, and Ortner signs. An intermittent focal colonic wall spasm was palpated at the tender point. Examination of other systems revealed no abnormalities. The patient's mental status was alert, and oriented with adequate responses. Despite having no family history of cancer, she underlined her fear towards cancer several times during the interview ("They have told me there are no signs of malignancy, but I am still afraid, every other person has cancer today"). CBC, CRP, and serum lipase were within normal limits.

The decision to hospitalization was a great relief for the patient as she demanded to stay as long as possible. A day before the hospitalization she discontinued Etifoxine on her own. Conservative management of the patient included normal saline infusion and spasmolytics. On morning rounds the patient every day complained of an episode of severe abdominal pain with diffuse radiation mostly started in the previous evening or night. The pain was accompanied by nausea but no vomiting was reported. She felt better during the day under more attention from the medical staff. On the 2nd day of admission, the patient developed tremors and hand/wrist muscle spasms - flexion of the wrist and metacarpophalangeal joints, an extension of the DIP (distal interphalangeal) and PIP (proximal interphalangeal) joints, and abduction of fingers (Main d'accoucheur - French for "hand of the obstetrician" sign) without any electrolyte abnormalities evidenced on laboratory tests. Due to worsening and frequently occurring episodes, she was advised to continue Etifoxine. Cardiologist consultation was required due to retrosternal chest pain, but ECG during the pain episode showed no abnormalities. Neurologist consultation yielded no significant information except for the advice to consult a psychiatrist. The psychiatrist made a diagnosis of SSD and prescribed the antidepressant Sertraline 25mg a day.

DISCUSSION

Somatic symptom disorder is a diagnosis that was introduced in 2013 with the publication of DSM-5. It consolidated multiple previous diagnosis of somatoform disorder, somatization, hypochondriasis, and pain disorder [13]. SSD has 2 major clinical characteristics: the presence of 1 or more somatic symptoms causing significant disruption in normal daily activities and persistently elevated levels of anxiety about the symptoms/health issues [12]. Even though there is no specific pathophysiology of the disorder identified, numerous risk factors are investigated to play a role in the development of SSD such as fewer than 12 years of education, separated, widowed or divorced status, reported psychological or sexual abuse during childhood, co-existing medical illnesses (especially in elderly), anxiety and depression [7]. One study demonstrated a correlation between the severity of SSD and conflicts within the family, which concluded that individuals who had stable low family conflict were most likely to follow a stable low somatic symptom trajectory [6]. Age was identified to be one of the predictors of SSD prevalence as well as its severity. Patients with age>60 y/o suffer more severe SSD with higher prevalence [25]. The genetic component was also found to have an effect based on a study conducted on monozygotic and dizygotic twins (n=28 531) that concluded about 7-29% attribution of genetic factors, the rest was influenced by the environmental factors [10]. Patients with SSD can be presented in 2 patterns: patients that have somatic symptoms without any organic cause and those that may have an organic abnormality but the perception of the problem is exaggerated due to a comorbid psychiatric condition. Previously this phenomenon was referred to as hypochondriasis which is now a part of SSD. The basis of this is the possible cognitive and perceptual distortions of the patients with increased sensitivity and decreased threshold for symptom perception (eg, pain) as well as abnormally increased attention to bodily sensations.

As the data from the nine population-based studies (n=28377) showed, the most common and burdensome symptom among the SSD patients was pain including back, joint, head, and extremity pains reported by 11-77% of responders [21]. Pain localization or radiation can vary and

involve any part of the body. Other possible complaints may originate from GI (nausea, vomiting, abdominal pain, bloating, gas, diarrhea), cardiopulmonary (shortness of breath, chest pain, palpitations), neurologic (movement disorders, sensory loss, weakness, paralysis), reproductive (dyspareunia, dysmenorrhea, erectile dysfunction) systems or be more nonspecific (fatigue, syncope, dizziness) [15, 22, 17, 18]. Several organ system complaints may be found in the same patient. Associated signs/symptoms can be related to somatization itself or be a manifestation of coexistent medical disorders (e.g. anxiety/depression) in our case - the primary somatic symptom (abdominal pain with diffuse radiations) was accompanied by episodes of neurologic signs (obstetrician's hand, paresthesia of bilateral upper extremities). This pattern was also reflected in a case report from the United Kingdom which presented an SSD patient with a chief complaint of abdominal pain and associated neurologic symptoms of headache, diplopia, and temporary visual loss, the patient also suffered from comorbid anxiety [8]. Another case report described an SSD patient with predominant left-sided chest pain for 3 years associated with facial paresthesias, and numbness and tingling sensation in his left arm and ankle [16]. Clinically, SSD can be divided into two categories, multisomatoform disorder (also known as undifferentiated SSD) which was defined as 'three or more medically unexplained symptoms ongoing or chronic for two or more years. The other category was specific SSD, diagnosed when the patient presents with a single medically unexplained symptom [1]. The number of symptoms does not influence the diagnosis of SSD but the prognosis and severity can be predicted. An increasing number of symptoms were found to be associated with poorer outcomes (eg, poor physical functioning - they may avoid physical activity) [15, 22, 9]. As mentioned, SSD can be coexistent with other psychiatric conditions, commonly anxiety and depression. This may lead to difficulty differentiating between SSD with anxiety and illness anxiety disorder which also presents as excessive thoughts and worries about health. These 2 can be separated by the presence of pronounced severe somatic symptoms (in SSD) that are not typical in illness anxiety (they have no or mild symptoms). Along with anxiety and depression, approximately two in three patients with an SSD meet the criteria for a personality disorder. The four most frequently identified personality disorders were avoidance 26.7%, paranoia 21.3%, self-defeating 19.1%, and obsessive-compulsive 17.1%. Interestingly histrionic personality disorder was identified in only 12.8% of the sample and antisocial personality disorder in 7.4% [5, 20]. Comorbid psychiatric disorders (especially depression) significantly worsen the mental condition of the patient and may lead to devastating outcomes including suicidal ideation. In a study of 142 SSD patients, 23.9% had active suicidal ideation in the past 6 months [23].

In addition to psychiatric conditions, SSD is often comorbid with general medical conditions. This can make the diagnosis even more challenging, especially when the patient is admitted to a department unrelated to neuropsychiatric care (eg, surgery, cardiology, urology, gastroenterology) where the awareness about SSD may not be sufficiently high. This diagnostic challenge was presented in the case reported in this article. The patient's assessment with multiple diagnostic tools was consistent with a mild form of colitis but the presence of disproportionately severe clinical manifestation and comorbid anxiety attack episodes led to consideration of SSD as a coexisting problem. In order to solve this challenge and establish if the patient with a general medical condition may also have SSD, medical professionals should determine whether the cognitive, emotional, and behavioral responses to the medical disease are excessive compared with most other patients with that medical disorder [15]. The level of functional impairment (eg, interpersonal, occupational, physical) caused by the condition should be assessed as it is typically higher in patients with a medical illness plus SSD. The risk factors (comorbidities, family history, social history) must be carefully evaluated to modify the level of suspicion. Moreover, multiple patient characteristics and details from the history of SSD can help the differentiation. The patients are not at all or only temporarily reassured by the normal/benign results of the diagnostic tests and soon they demand/repeat the same or even more invasive tools to search for the reasons for their health issues. Non-reassurance may further cause the frustration about the doctors leading to "doctor-shopping", visiting different clinics, and doctors, and taking laboratory tests or imaging without professional medical advice [19]. Other possible positive signs include vague/inconsistent history of present illness, no alleviating factors for the symptoms, multiple courses of standard treatment without a result, attributing normal physical sensations to medical illnesses, repeatedly checking one's body for abnormalities, and unusually high sensitivity to medication side effects. Adverse effects produced by medications can have amplifying effects on symptom perceptions, particularly in people focusing on somatic symptoms without medical causes [11]. The environment surrounding the condition, which includes sensationalized media coverage, intense mistrust of medical knowledge and practitioners, and a treatment strategy that overemphasizes the biological and disregards psychosocial components, can drive SSD symptoms in a cycle. All of these factors heighten patients' worries and negative expectations, extend their disability, and reinforce their position as "sick" [4].

General approach to the patient with suspected SSD includes careful history taking covering the history of present illness, and past medical, family, and social history. Specific clues should be obtained from the history to risk stratify and diagnose the patient accordingly. For example, it is essential to understand which somatic symptoms trigger health anxiety and how the patient responds to it, if they devote too much time and energy thinking about their illness and anxiety, whether they are undergoing other current medical evaluations, etc. Past events should also be clarified to understand whether there is a pattern of multiple physical symptoms, increased healthcare utiliza-

tion, and greater psychosocial impairment than expected. To think retrospectively about our case, thorough patient history already had some clues to think about the diagnosis of SSD. This includes constant worrying about the seriousness of the illness, visiting different doctors, and conducting multiple diagnostic tests and interventions. Besides proper history, laboratory, imaging and other diagnostic tests should be obtained to exclude all the possible medical conditions explaining the patient's chief complaint. However, the precise diagnosis of SSD should be made according to the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) criteria [3].

Initial treatment for SSD involves scheduling regular outpatient visits with a primary care physician. Patient education about their condition and its management is also a part of treatment and it is necessary to acknowledge that their symptoms are real. Laboratory tests and imaging studies should be minimized as much as possible. Patients must be informed clearly that all the life-threatening causes are ruled out. For treatment-resistant patients with somatic symptom disorder who do not respond satisfactorily to initial treatment, primary care clinicians should continue initial treatment and in addition, discuss the case with a psychiatrist, meet with the patient and family, and administer antidepressants, psychoeducation, and relaxation training. Use of antidepressants is associated with a risk of adverse effects that can worsen the already existing somatic symptom so they are only used when a patient with SSD has comorbid symptoms of anxiety or depressive disorders [2, 12, 14].

CONCLUSION

In this article, we discussed a diagnostic challenge encountered in a surgical department. This case highlights the struggles of identifying psychiatric illness in a patient presenting with a physical symptom (abdominal pain). In order to avoid misdiagnosis followed by unnecessary interventions including invasive techniques (eg, laparotomy), awareness about SSD should be increased among doctors of different specialties. Even though the patient had imaging results consistent with mild colitis, the major contribution to the disturbing symptoms was related to her mental condition. Therefore, managing the SSD would be much more beneficial in alleviating the symptoms than treating colitis itself.

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სომატური სიმპტომური აშლილობა ქირურგიაში (შემთხვევის აღწერა)

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რეზიუმე

1959 წელს ფრანგმა პიერ ბრიქემ (Pierre Briquet) აღწერა შემთხვევები, როცა პაციენტები მასთან მიდიოდნენ მრავალფეროვანი სომატური ჩივილებით, რომელსაც იგი ვერანაირ სამედიცინო საფუძველს ვერ უძებნიდა და მდგომარეობაც ძნელად ექვემდებარებოდა მკურნალობას. ძალიან მალე პაციენტები იგივე, ან ახალი, მცირედ შეცვლილი ჩივილებით ბრუნდებოდნენ. მრავალი წლის განმავლობაში ამ აშლილობას ბრიქეს სინდრომს უწოდებდნენ. დღეს ეს მდგომარეობა სომატურ სიმპტომურ აშლილობად (SSD) მოიხსენიება.

სომატური სიმპტომური აშლილობა ფსიქიატრიული დაავადებაა, რომელიც კლინიკური გამოვლინებებით შესაძლოა ძალიან ჰგავდეს ქირურგიულ პრობლემას. უამრავი დიაგნოსტიკური მეთოდის გამოყენების მიუხედავად, კლინიკურ ჩივილებზე პასუხისმგებელი მნიშვნელოვანი ორგანული ეტიოლოგიის აღმოჩენა ვერ ხერხდება, რის გამოც ქირურგმა შესაძლოა მეტად ინვაზიურ ჩარევებს (მაგ, ლაპაროტომიას) მიმართოს. ამ უკანასკნელის თავიდან აცილების მიზნით, სასურველია ქირურგებში, ისევე, როგორც სხვა ვიწრო სპეციალობის ექიმებში, ამაღლდეს ინფორმირებულობა სომატური სიმპტომური აშლილობის, მისი დიაგნოსტიკური კრიტერიუმების და დამახასიათებელი კლინიკური გამოვლინების შესახებ. ამ სტატიაში, ჩვენ აღვწერთ, კლინიკურ შემთხვევას ქირურგიულ განყოფილებაში მოთავსებული სომატური სიმპტომური აშლილობის მქონე პაციენტის შესახებ და განვიხილავთ იმ მნიშვნელოვან დეტალებს, რომლებიც დაგვეხმარება ამ დაავადების ამოცნობაში.

58 წლის პაციენტი, ქალი, კლინიკაში მოთავსდა სპაზმური ხასიათის მუცლის ტკივილით, ირადიაციით მუცლის სხვადასხვა არესა და გულმკერდში, რეტროსტერნულად. პაციენტის ისტორიაში აღსანიშნავია 2019 წელს ჩატარებული სიგმოიდური ნაწლავის რეზექცია, როდესაც მსგავსი ჩივილების გამო მას დაუსვეს მეგა სიგმის დიაგნოზი. წარმატებული ქირურგიული ინტერვენციის მიუხედავად, პაციენტს შეუნარჩუნდა იმავე ხასიათის ჩივილები, რომელთა მიზეზის დადგენაც ვერ ხერხდებოდა. სხვადასხვა სპეციალობის ექიმებთან ვიზიტების, ლაბორატორიული და რადიოლოგიური კვლევების შემდეგ, პაციენტს გაეწია ფსიქიატრის კონსულტაცია და დაისვა სომატური სიმპტომური აშლილობის დიაგნოზი. აღნიშნული დიაგნოზის შესაფერისი მკურნალობის ფონზე პაციენტის მდგომარეობა მნიშვნელოვნად გაუმჯობესდა, თუმცა პერიოდულად ადგილი აქვს მისთვის ჩვეული ჩივილების რეციდივს.

საკვანძო სიტყვები: სომატური სიმპტომური აშლილობა ქირურგიაში, ბრიქეს სინდრომი, შემთხვევის აღწერა