CASE REPORT

Somatic symptom disorder, a new DSM-5 diagnosis of an old clinical challenge

Tea Rosic,[1] Sameer Kalra,[1] Zainab Samaan[2]

[1]McMaster University, Hamilton, Ontario, Canada
[2]Department of Psychiatry & Behavioural Neurosciences, McMaster University, Hamilton, Ontario, Canada

Correspondence to
Dr Zainab Samaan, samaanz@mcmaster.ca

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SUMMARY

Somatic symptom disorder (SSD) is characterised by a dysfunctional preoccupation with one or more physical symptoms. Patients with SSD often pursue excessive and unnecessary investigations, hospitalisations and treatments that significantly affect quality of life and drain healthcare resources. Thus, appropriate diagnosis and careful management are required to mitigate the patient's distress and to reduce the burden to the healthcare system. SSD is a new disorder defined in the Diagnostic and Statistical Manual Fifth Edition (DSM-5), replacing somatoform and related disorders in the DSM-4-Text Revision with diagnostic criteria that are inclusive of a broad array of presentations. This report presents a detailed clinical case of an elderly man with a history of frequent hospital visits presenting with SSD. We discuss diagnostic challenges and evidence-based management in acute inpatient as well as in outpatient settings. We also review data on healthcare utilisation associated with SSD.

BACKGROUND

Somatic symptom disorder (SSD) is characterised by preoccupation with one or more distressing physical symptoms, resulting in disruption of daily life.[1] According to Diagnostic and Statistical Manual Fifth Edition (DSM-5), a known medical condition explaining the somatic symptom does not preclude the diagnosis of SSD.[1] Rather, considering the medical diagnosis, the patient's distress and dysfunction is in excess of what would be expected.

SSD is a new disorder defined in the DSM-5, replacing somatoform and related disorders from the DSM-4-Text Revision (TR).[2] For example, criteria for somatisation disorder required the patient's constellation of somatic symptoms to include four different pain symptoms, two gastrointestinal symptoms, one sexual symptom and one pseudoneurological symptom.[2] In contrast, diagnosis of SSD requires the presence of just a single somatic symptom. Further, any one somatic symptom is not required to be continuously present, but rather the state of being symptomatic, with any variation of symptoms, is required for the diagnosis of SSD. These diagnostic criteria are inclusive of a broader array of presentations than the former DSM-4-TR diagnoses of somatisation disorder, undifferentiated somatoform disorder, somatoform disorder not otherwise specified (NOS) and pain disorder, which were not included in the DSM-5. Individuals previously meeting criteria for these disorders may now be diagnosed with SSD. Therefore, a re-examination of the disorder presentation in clinical practice is important to address. More specifically, we aim to:

1. Present a detailed account of a clinical case of SSD
2. Provide data on healthcare utilisation associated with SSD
3. Describe the current evidence-based guidelines for the management of SSD

In this report, we present a case of a 72-year-old man with SSD—a remarkably common and clinically demanding disorder. We discuss diagnostic challenges and recommendations for management in acute inpatient as well as in outpatient settings. Written informed consent to present this case was obtained from the patient prior to writing this report.

CASE PRESENTATION

A 72-year-old man was brought to the emergency room after reporting suicidal ideation to a local crisis outreach team. The patient was medically cleared in the emergency room, where an initial assessment revealed a 3-week history of increasingly depressed mood on a background of chronic pain. He was transferred to Psychiatry Emergency Services, where he told the medical team that he had held a knife on his person and endorsed suicidal ideation to his wife. He also revealed that he impulsively attempted to strangle himself with a belt the day before, but was able to stop himself from doing so and did not seek medical attention.

A collateral history obtained from his wife revealed at least two other suicide attempts by strangling with a belt, one of which occurred in recent months and the other several years prior. The patient did not seek medical attention for either event. She also reported his frequent suicidal ideation, low mood, increased agitation and poor sleep.

The patient was admitted to inpatient psychiatry. On admission, he described a 3-week history of low mood and social withdrawal. He stated that he had called 911 to be brought to the hospital because he "could not see right, and felt as though he was going to die". This was in contrast to notes from the emergency room assessment. The patient did not report changes to his sleep, energy or appetite and denied suicidal ideation. He stated that his worsening mood was triggered by an episode of burning pain in his legs. He noted that this pain was long-standing over 30 years, but now began to worry that something could be seriously wrong with his health. He admitted then that he had expressed suicidal ideation in an attempt to be

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admitted to hospital and investigated for his pain condition. The patient also explained that he had recently heard that a previous tenant of his home had died of cancer, which made him very concerned that he might have cancer. He had recently advised his wife that they needed to move, as he feared he might contract cancer.

The patient was living independently with his wife. He had married in his late fifties and had had no children. He was the eldest of two siblings, and emigrated from Europe as a child. He had not entered into high school and hence had not completed any school education. He had been retired since the age of 45 years. He denied any current use of tobacco, alcohol or any illicit substances. Family history was positive for anxiety and possible alcohol abuse.

A Mental Status Examination was conducted on admission. It revealed an elderly man who was dressed casually. His behaviour was appropriate and he made good eye contact. His speech was normal and his thought form was mostly goal-directed and at times circumstantial. His mood was subjectively low, and his affect reactive. He denied any psychotic symptoms or suicidal ideation. Cognition appeared grossly normal. However, the Montreal Cognitive Assessment (MoCA) was administered on admission as a baseline screen, and the patient scored 19 out of 30, indicating mild cognitive impairment.[3] A second MoCA administered 3 weeks later revealed a score of 23, remaining within the range of mild cognitive impairment.

A chart review revealed eight psychiatric admissions from 2005 to 2014. Seven admissions identified chronic pain as a primary diagnosis, with additional diagnoses varying from major depressive disorder, persistent depressive disorder and anxiety disorder NOS. In later admissions, dependent and narcissistic personality disorder traits were documented, as were opioid dependency and marital conflict. Discharge summaries noted multiple emergency room presentations for pain over this 10-year period. One admission in 2013 was for Triskaidekaphobia (fear of the number 13, including the year 2013).

Chart review also revealed 71 local emergency room visits since 1994 (figure 1). For 20 of these visits, musculoskeletal pain was recorded as the 'reason for visit' in the electronic record, including leg, neck, shoulder and back pain. Sixteen visits resulted in referral to Psychiatry Emergency Services.

Other medical history included hypertension, hyperlipidaemia, benign prostatic hypertrophy and bilateral pulmonary emboli in 2010, as well as a motor vehicle accident in 2010, from which he sustained shoulder and knee fractures. The patient also had a previous diagnosis of disc degeneration. Preadmission medications included acetaminophen 650 mg four time a day, pregabalin 50 mg every bedtime and fentanyl 12 mcg patch q 2 days for pain control. He was also prescribed duloxetine 60 mg every morning, risperidone 2 mg every bedtime and clonazepam 2.5 mg daily, in divided doses. The patient was also taking docusate sodium 100 mg two times a day, fenofibrate 145 mg daily, furosemide 40 mg daily, alfuzosin 10 mg every bedtime, as well as warfarin 5 mg daily due to his history of pulmonary emboli.

In the past, he had been trialled on numerous other medications for pain control, including gabapentin, morphine, hydromorphone, oxycodone, carbamazepine and caudal epidural steroid injections. During his various psychiatric admissions, he had been trialled on paroxetine, venlafaxine, duloxetine, quetiapine, olanzapine, risperidone and clonazepam.

Early in the course of this admission, the patient expressed heightened concern about various somatic symptoms. He was initially preoccupied with upper respiratory symptoms. He feared coughing, which he worried could cause disc prolapse. He similarly feared sleeping, worrying he might choke. During this time, other pain symptoms were not communicated. Several days later, the patient once again began to express increasing concern about burning pain in his legs, and endorsed an acutely depressed mood and suicidal ideation. His mood and suicidal ideation were consistently reactive to somatic symptoms, and fluctuated daily. The patient was intensely preoccupied by the symptoms and eventually described his condition as 'paralysis' of his legs, despite his ability to walk. He was able to ambulate independently, but a walker was provided when his fentanyl dose was increased to 25 µg to avoid potential falls from excessive sedation.

The patient requested various investigations including X-ray, myelography and MRI, despite being aware of the results of numerous previous investigations and of his diagnosis of mild disc degeneration. He reported that he visited the emergency room five times in the preceding 2 months to investigate his leg pain. He also made requests to be transferred to a different hospital to further investigate his pain symptoms. At other times, he perseveratively requested euthanasia or bilateral leg amputation. He also requested a colostomy to avoid constipation in order to take morphine for pain. The patient had unattainable demands and was not amenable to discussion.

INVESTIGATIONS

Laboratory investigations on admission, including complete blood count, liver enzymes, B_{12} and thyroid function, were within normal range. Renal function was reduced with elevated creatinine (121 µmol/L; local laboratory normal range 64–111 µmol/L) and estimated glomerular filtration rate at 51 (>60). Urea was elevated at 9.8 mmol/L (3.0–9.2 mmol/L). Calcium was slightly elevated at 2.59 mmol/L (2.15–2.55 mmol/L). Fasting glucose was high at 7.4 mmol/L (3.8–6.0 mmol/L), as was glycated haemoglobin at 7.8% (4.0–6.0%), and diabetes mellitus type 2 was subsequently diagnosed. Triglycerides were elevated at 1.77 mmol/L (<1.7 mmol/L). International Normalised Ratio was elevated at 1.7, as expected with warfarin treatment.

MRI of the lumbar spine revealed mild degenerative changes, with minimal progression from the previous, of the neural foraminal stenosis on the left side at L5-S1. Neither nerve root compression nor evidence of cauda equina was seen. MRI of the brain was reported to show generalised atrophy, more pronounced within the frontal and anterior temporal lobes (figure 2). There were no acute intracranial findings and no signs of a vascular dementia.

Neuropsychological testing was carried out to corroborate the atrophy seen on MRI, and to further investigate the mild cognitive impairment detected on repeated MoCAs. The testing revealed cognitive decline in keeping with age and mental status, and no evidence of frontal lobe dementia. Memory testing revealed mild encoding problems in retaining verbal information, though visual memory was intact. The patient scored as having verbal intellect in the average range, and non-verbal intellect in the borderline range. His relative strengths included orientation, language (comprehension, confrontation naming, fluency, verbal abstraction), basic attention, working memory, set shifting, visuoconstruction and visual memory. Relative weaknesses were detected in processing speed, mental flexibility, angle perception and three-dimensional visuoconstruction. Psychological testing was also carried out; however, the results were found to be invalid as the patient over-inflated his responses to the questionnaires.

Prior investigations included electromyography (EMG), performed in 2005, which was reported as within normal limits and demonstrated no evidence of focal entrapment neuropathy,

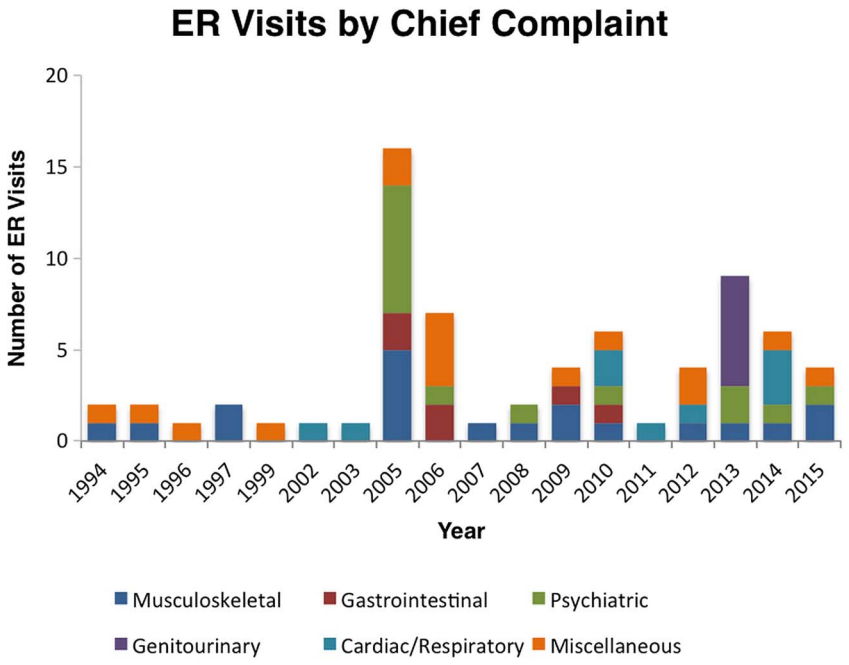

Figure 1 Patient's emergency room visits from 1994 to 2015.

lumbosacral radiculopathy or peripheral polyneuropathy. The EMG report indicated that the symptoms were unlikely to be related to a spinal issue due to the bilateral sock-type distribution, and that though early distal small fibre involvement could not be excluded, the time sequence would be atypical for this type of neuropathy. MRI of the lumbar spine from 2005 showed mild degenerative disc disease and minimal facet osteoarthritis in the lower lumbar spine, without focal neural compromise. MRI of the lumbar spine from 2008 showed minimal degenerative disc disease with no change compared to 2005.

DIFFERENTIAL DIAGNOSIS

This patient presented with a constellation of symptoms representing features of possible SSD, depression, anxiety, personality disorder traits and cognitive decline. These included low mood, suicidal ideation, poor sleep, irritability, social withdrawal, excessive worry and preoccupation with distressful physical symptoms. As such, a broad differential diagnosis was considered, including major depressive disorder with somatic concerns, delusional disorder with somatic concerns, generalised anxiety disorder, dependent personality disorder and frontotemporal dementia, in addition to SSD. He was diagnosed with comorbid SSD and depression, meeting DSM-5 criteria for both disorders. Neuropsychological testing ruled out the diagnosis of frontotemporal dementia as the aetiology of his presentation.

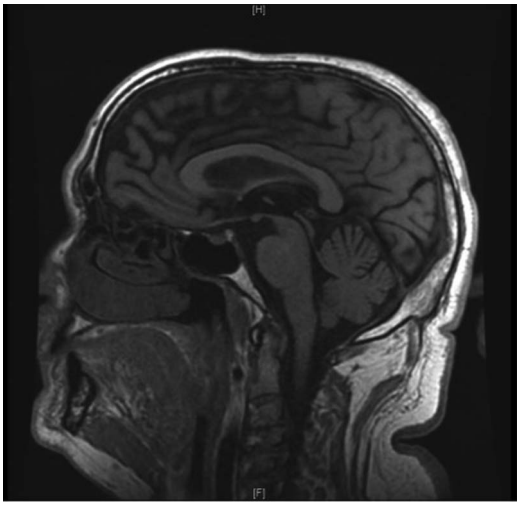

Figure 2 MRI of the brain showing generalised atrophy, more pronounced within the frontal and anterior temporal lobes.

TREATMENT

The patient was continued on some of his home medications, including warfarin 5 mg every bedtime, alfuzosin 10 mg every bedtime, docusate sodium 100 mg and clonazepam 2.5 mg daily in divided doses.

To address his acute presentation, several medication changes were made. For pain management, pregabalin was increased gradually to 250 mg daily in divided doses, and acetaminophen 650 mg four times a day and hydromorphone 0.5 mg three times a day were started as *pro re nata* medications. Fentanyl 25 µg/h patch q 3 days was ordered in place of his home fentanyl 12 µg/h patch q 2 days. Polyethylene glycol 17 g daily and senna 17.2 mg every bedtime were added to manage constipation.

Duloxetine was discontinued and substituted by venlafaxine, starting at 75 mg and increased to 150 mg daily after 2 weeks. Risperidone was discontinued. Quetiapine 50 mg every bedtime was started. Lorazepam 0.5–1 mg q4 h PRN up to 4 mg daily was added.

To optimise management of hypertension and hyperlipidaemia, ramipril 10 mg daily and rosuvastatin 20 mg daily were added. Additionally, sodium chloride 0.9% nasal spray 2 sprays PRN and amylmetacresol-dichlorobenzyl lozenge q4 h PRN were added. Metformin 250 mg two times a day with meals was started following the diagnosis of type 2 diabetes.

In-hospital referrals to occupational therapy and physiotherapy were made. The patient declined physiotherapy throughout the admission. A consultation for electroconvulsive therapy (ECT) was completed, but the patient declined to pursue this.

OUTCOME AND FOLLOW-UP

The patient was discharged after 43 days in hospital. He reported feeling 'stabilised', with improved pain and elevated

mood. Outpatient follow-up was arranged with geriatric psychiatry and telephone counselling. The family was provided with information about retirement communities and options for respite care.

DISCUSSION

Diagnosis

The criteria for SSD have been expanded in DSM-5 to capture a broader array of presentations than in previous classifications of somatoform disorders. This also affords earlier diagnosis. Diagnosis relies on the presence of somatic symptoms with a 6-month persistent duration and disproportionate thoughts, feelings and behaviours related to the somatic symptoms.[1] In comparison to previous editions of the DSM, the emphasis now lies on the consequences of abnormal thoughts, feelings and behaviours in response to the somatic symptoms rather than whether or not they have a medical explanation. This change increases the sensitivity of the diagnostic criteria for SSD compared to previous diagnoses. It also begets a degree of clinical judgment when deciding what constitutes a 'significant disruption in daily life', which can help to allay concerns of overpathologising all individuals with somatic symptoms.[4] In addition to somatisation disorder, the DSM-4 included diagnostic criteria for undifferentiated somatoform disorder, pain disorder and somatoform disorder NOS, none of which were included in the DSM-5 classification.[2] Individuals previously meeting criteria for these disorders may well be diagnosed with SSD, further increasing the prevalence of SSD.

Given the presence of somatic symptoms, many patients with SSD will present to family medicine clinics rather than psychiatric settings, furthering the need for appropriate recognition of the condition.[5] Despite studies suggesting that somatic symptom disorders are quite common, the condition remains less recognised and its diagnosis is rarely documented.[6] Often, non-psychiatric clinicians will apply symptom diagnoses such as 'headache' or 'atypical chest pain', whereas psychiatrists may apply more ambiguous diagnoses such as 'Adjustment Disorder' or 'Major Depression NOS' to describe the same clinical phenomenon.[4] Somatic symptoms are also frequent manifestations of mood disorders—in elderly populations in particular, MDD may initially present with somatic symptoms.[7] Although rare, delusional disorder with somatic symptoms must also be considered in the differential diagnosis. Given the potential overlap with other conditions, the astute clinician must carefully clarify the diagnosis for the appropriate triage and management of patients.

It is quite clear that our patient met criteria for SSD—he reported of numerous distressful symptoms within the span of several years that were marked with significant periods of dysfunction. In this hospital visit, he exhibited disproportionate thoughts, feelings and behaviours in response to his symptoms, most notably for leg pain. The patient would persistently request further investigations to 'find something to fix', despite having multiple MRIs that were unchanged from previous results. He would often report of paralysis, despite being able to walk. In response to his symptoms, he adamantly requested bilateral leg amputation and euthanasia, and expressed suicidal ideation, all due to his experience of pain.

Over his 20-year history of ER visits and hospital admissions, his chief problems consisted of a number of symptoms that were medically explained (eg, bilateral neuropathic leg pain secondary to diabetes and disc degeneration) and others that were investigated with no definitive aetiology. Importantly, this presence of multiple diagnosed medical problems, including diabetes and disc degeneration, does not preclude a diagnosis of SSD, which he most certainly meets criteria for. This highlights one of the most significant changes in diagnostic criteria from previous somatoform classifications. The large quantity and character of his visits is also significant, as demonstrated in figure 1. They demonstrate a consistent pattern of significant distress and are associated with psychiatric diagnoses. As well, his experience of pain was correlated with his levels of distress and dysfunction, both during this admission and in previous situations recounted by his wife. Limited epidemiological data on DSM-4 somatoform disorders support the notion that the patient had several risk factors for developing this diagnosis, including older age, low education, unemployment and concurrent chronic physical illness.[8]

It is important to note that somatoform disorders are often present with comorbid psychiatric conditions, most commonly depression and anxiety. Prevalence of depression and anxiety has been reported at 58%, or six times higher than in patients without somatisation.[9 10] This patient, too, was diagnosed with comorbid depression. Consequently, it is important to screen for mood, anxiety, personality, psychotic and substance use diagnoses, as treating these conditions may diminish the severity of the somatic symptom disorder. Notably though, somatic symptom disorders do not typically resolve with treatment of a concurrent psychiatric diagnosis—if they do, then a diagnosis of SSD is less likely.[6]

Healthcare utilisation

Increased healthcare utilisation is a significant concern in SSD. Precise calculations of the impact of somatoform disorders on healthcare utilisation are rare in the literature. One study found patients with somatisation to have 1.4-fold more primary care visits, 1.65-fold more specialist visits, 2.6-fold more emergency room visits and threefold more hospital admissions.[9] Another study found that patients with somatoform disorders utilised outpatient and inpatient services 2.2-fold higher than individuals without these disorders during the 2 years prior to treatment, and found treatment with cognitive-behavioural therapy (CBT)-based psychotherapy to result in a 24.5% decrease in cost of outpatient care and a 36.7% decrease in cost of inpatient care.[11]

Our patient had 71 local emergency room visits since 1994, with 53 visits in the past 10 years since his first psychiatric admission in 2005. The average cost of a single emergency room visit at the local hospital was $C170 in 2013–2014. Cost estimates were obtained from the local hospital administration as an approximate cost for services provided at the hospital. The average cost for seniors visiting the emergency department, however, is estimated to be higher, at $C386 for each visit.[12] Using these figures, the conservative estimated cost for the patient's 53 emergency room visits since 2005 ranges from $C9010 to $C20 458.

Further, the patient spent a total of 87 days as an inpatient in psychiatric units during the eight admissions since 2005, not including the present admission. The average daily cost for an inpatient bed in a mental health unit at this hospital was $C624 in 2014–2015. This amounts to an estimated minimum cost of $C54 288 for the patient's previous psychiatric admissions. For the current admission of 43 days, the cost is estimated to be about $C26 832, not including costs of investigations or medications.

Considered altogether, a very conservative estimate of the total cost to the healthcare system over 10 years amounts to $C90 130. This does not include the cost of primary care

appointments, outpatient subspecialty appointments or investigations, or medications dispensed outside of the hospital.

Evidence-based management of SSD

Management of SSD involves a combination of pharmacological therapies and psychological interventions. Treatment regimens should be selected based on evidence and tailored to each individual patient, accounting for their particular presentation, and psychiatric and medical comorbidities. For example, a single drug may be chosen to reduce symptom severity and to treat a comorbid psychiatric disorder. As well, the use of psychological therapy depends on consent and a patient's psychological mindedness.

Currently, several of the most studied medicines in the treatment of somatisation disorder and major depressive disorder with somatic symptoms are duloxetine, venlafaxine, mirtazapine and St John's wort. Randomised controlled trials have found some benefit over placebo for all of these medications. A Cochrane Review of evidence for the efficacy of duloxetine in treating pain syndromes revealed benefit in treatment of painful diabetic peripheral neuropathy (number needed to benefit (NNTB) is 5), and lower quality evidence for treatment of painful physical symptoms in depression and fibromyalgia (NNTB is 8).[13] In an open observational study of patients with a primary diagnosis of at least one chronic pain syndrome and concomitant depression, treatment with mirtazapine was found to significantly reduce pain from baseline irrespective of pain syndrome, as well as improvement from baseline in sleep disturbance, irritability and exhaustion.[14] A randomised, open-label trial evaluating mirtazapine versus venlafaxine in treating somatic symptoms associated with major depressive disorder found the drugs to be equal in response and remission rates (57.5% vs 43.4% and 24.7% vs 15.1%, respectively).[15] Two randomised controlled trials of St John's wort have found it to be superior to placebo in reducing somatoform symptoms in patients diagnosed with somatisation disorder.[16 17]

However, a Cochrane Review of 26 randomised controlled trials comparing the efficacy of various antidepressants, antipsychotics and natural products in treatment of somatoform disorders, concluded that the evidence is of low quality in studies suggesting efficacy of amitriptyline, citalopram, clomipramine, fluoxetine, flupentixol, milnacipran, mirtazapine, paliperidone, paroxetine, reboxetine, sertraline, trazodone, quetiapine, venlafaxine, butterbur root, lemon balm leaf-Ze 185, passionflower herb, St John's wort or valerian root.[18] The authors noted that current evidence is limited by small sample sizes, high risk of bias and lack of follow-up assessment.[18]

Treatment with electroconvulsive therapy (ECT) has been reported effective in case reports of patients with somatoform disorders. However, there is no other evidence supporting its effectiveness.[19 20] Other studies have found somatisation to predict low likelihood of sustained remission with ECT.[21]

Numerous psychotherapies have also been studied in the treatment of somatoform disorders. A meta-analysis of 16 studies that compared psychotherapy (CBT or psychodynamic therapy) with treatment as usual for chronic somatoform disorder (mean length of physical symptoms was >8 years) found that improvement of physical symptoms and functioning was superior with both CBT and psychodynamic psychotherapy.[22] Improvement of psychological symptoms (eg, depression, anxiety, anger), however, was comparable between psychotherapy and treatment as usual.[22] Other therapies considered effective in the treatment of somatoform disorders are mindfulness therapy and relaxation training.[23 24]

A Cochrane review of 21 studies found that all psychological therapies included in the review (CBT, mindfulness, psychodynamic and integrative therapy) were superior to usual care or waiting list in reduction of symptom severity.[25] However, the authors noted effect sizes were small and all studies included only participants who were agreeable to receive psychological treatment, thus limiting the studies' external validity.[25] A significant challenge to treatment with psychotherapy arises if the patient is not psychologically minded, and cannot identify and acknowledge the emotions and cognitive distortions underlying their somatic symptoms. Despite stating many times that he was depressed and suicidal, our patient exhibited alexithymia—an inability to identify and describe one's emotions. He was unable to elaborate beyond feeling 'depressed' and being in physical pain. Alexithymia is negatively associated with psychotherapeutic outcome, though alexithymia itself may be improved with psychotherapeutic interventions.[26–28]

In addition to selecting a therapeutic regimen, practitioners may find it beneficial to schedule time-limited appointments with these patients on a regular basis, rather than seeing them sporadically as symptoms arise, in order to proactively address patients' symptoms.[29] Regular dosing of pain medications, rather than as needed analgaesics, are recommended.[29] Management should be a collaborative process with the patient to help reduce distress from symptoms, with the goal to restore function as much as possible.[29]

Patient's perspective

"Many, many years of pain. It gradually goes away. The reason for my pain are the discs in my back and they need to be fused. I say to the ER doctors that the problem is in my back. It's L4, L5 and S1, and that has got to be rebuilt. They say 'go home, nothing we can do'. They don't listen. None of them listen. I want a consult with the orthopaedic surgeon. I want them to take a myelogram. The myelogram shows everything. There is something that they are not seeing. I have 4–5 months with no pain. Doing the wrong thing makes it come back—lifting, pulling, running, sneezing very hard. I feel so terrible I want to commit suicide because that's now terrible pain. I do want to live if I have no pain. I went 2–3 times to ER last month. Maybe 50 times over the past two years, mostly for this problem. I'm hoping that someone will change their mind and do something. Nobody listens. The guy that did the MRI here said it is like the last one and I wanted to scream. They said there is nothing they can do. I was hoping they would find something that they could fix. I hope the pain goes away, that's what I'm hoping for."

Spouse's perspective

"He says, I don't feel right and I don't know what's wrong with me and I need to go to the hospital. And the hospital doesn't find anything that's wrong. It's always the same type of pain in his legs. When the pain isn't there he is a totally different person."

Learning points

- Somatic symptom disorder (SSD) is common. The prevalence of somatisation disorder of the Diagnostic and Statistical Manual (DSM-4)-Text Revision (TR) has been estimated to be 4–6% in the general population.[30 31] As the new DSM-5 diagnostic criteria for SSD are more sensitive and encompass the DSM-4V-TR diagnoses of somatisation disorder, undifferentiated somatoform disorder, somatoform disorder not otherwise specified and pain disorder, the prevalence of SSD should be higher.
- Diagnostically, SSD should be differentiated from primary mood, anxiety and delusional disorders with somatic symptoms. Somatoform disorders are often comorbid with depression and anxiety.
- SSD leads to considerable strain on healthcare resources through increased healthcare contacts, including diagnostic investigations, emergency room visits and hospitalisations.
- Response to treatment is limited. Evidence suggesting benefit with psychopharmacological treatment is considered to be of low quality. Several psychotherapies have been found to be effective treatments, but are of limited use for psychologically minded patients only.

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Competing interests None declared.

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