Case report

# Somatic symptom disorder: a diagnostic dilemma

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## SUMMARY

Somatic symptom disorder (SSD) is a diagnosis that was introduced with publication of the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) in 2013. It eliminated the diagnoses of somatisation disorder, undifferentiated somatoform disorder, hypochondriasis and pain disorder; most of the patients who previously received these diagnoses are now diagnosed in DSM-5 with SSD. The main feature of this disorder is a patient's concern with physical symptoms for which no biological cause is found. It requires psychiatric assessment to exclude comorbid psychiatric disease. Failure to recognise this disorder may lead the unwary physician or surgeon to embark on investigations or diagnostic procedures which may result in iatrogenic complications. It also poses a significant financial burden on the healthcare service. Patients with non-specific abdominal pain have a poor symptomatic prognosis with continuing use of medical services. Proven treatments include cognitive behavioural therapy, mindfulness therapy and pharmacological treatment using selective serotonin reuptake inhibitors or tricyclic antidepressants. The authors describe the case of a 31-year-old woman with an emotionally unstable personality disorder and comorbid disease presenting to the emergency department with a 3-week history of left-sided abdominal and leg pain. Despite a plethora of investigations, no organic cause for her pain was found. She was reviewed by the multidisciplinary team including surgeons, physicians, neurologists and psychiatrists. A diagnosis of somatoform symptom disorder was subsequently rendered. As patients with SSD will present to general practice and the emergency department rather than psychiatric settings, this case provides a cautionary reminder of furthering the need for appropriate recognition of this condition.

## BACKGROUND

Somatisation is present when emotional or psychological distress is manifested as physical symptoms with no biological cause found. To better define these disorders and make them more relevant to the primary setting, the nomenclature for the diagnostic category Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5), previously known as somatoform disorders, was changed to somatic symptom and related disorders from the DSM-4 Text Revision.[1] In contrast to the criteria for somatisation disorder which required a constellation of somatic symptoms including four different pain symptoms, for example, two gastrointestinal, one sexual and one pseudo-neurologic, somatic symptom disorder (SSD) requires the presence of just a single somatic symptom. Subsets of SSD include the following: conversion disorder, factitious disorder, illness anxiety disorder, psychological factors affecting other medical conditions, other specified somatic symptoms and related disorders and unspecified somatic symptoms and related disorders. Increased healthcare utilisation is a significant concern in this patient cohort.

## PRESENTATION

A 31-year-old woman with a high Body Mass Index (BMI) presented to the emergency department with a 3-week history of left-sided abdominal and leg pain. She described an episode of nausea and vomiting. Her bowels opened 2 days previously and she denied rectal bleeding or mucus.

She described her abdominal pain as 'sharp, shooting and stabbing' in nature and radiating down her left leg. Her medical history included chronic depression and an emotionally unstable personality disorder. This was her fifth emergency presentation requiring inpatient admission with headache, diplopia, non-specific abdominal and left leg pain over a 12-month period. All investigations including a lumbar puncture and radiology were normal. She reported suicidal ideation in January 2013 resulting in an impulsive opiate overdose. In 2008, she started mobilising with crutches despite no abnormal pathology and progressed to a wheelchair in 2015 despite a normal MRI spine. She reported a 6-year history of intermittent left leg weakness, chronic back pain and constipation. She had a provisional diagnosis of epilepsy with recurrent partial seizures. She declined investigation with an electroencephalogram (EEG). In addition, she described a history of temporary visual loss of her right eye. Ophthalmology review noted normal visual evoked potentials. The clinical impression was of functional reduced visual acuity. She was also diagnosed with asthma, anxiety and migraine. Her surgical history included an appendectomy in 2009. She underwent an examination under anaesthesia and botox injection for an anal fissure. Postoperatively, she developed a seizure and was admitted to Intensive Care Unit (ICU) for observation.

She did not require intubation. She was allergic to aspirin, paracetamol and non-steroidal anti-inflammatory drugs (NSAIDs). Her medications included dihydrocodeine, phenytoin, lamotrigine, sertraline, folic acid, salbutamol and seretide. She was an ex-smoker with a 4.5 pack-year history. She did not consume alcohol. She completed



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second-level education and was currently unemployed. In addition, she required assistance from her partner to help her dress.

## INVESTIGATIONS

Her observations were as follows: heart rate 87 beats/min, blood pressure 127/79 mm Hg, respiratory rate 19 breaths/min, oxygen saturation 95% and temperature 36.4°C. Physical examination confirmed left-sided abdominal tenderness with no evidence of guarding or peritonism. Bowel sounds were present on auscultation. Digital rectal examination was unremarkable. Neurological examination confirmed normal power, tone and reflexes of her upper and lower limbs. Her cranial nerves were intact. Laboratory investigations showed a normal full blood count and a mildly elevated C reactive protein (CRP) (9 mg/L). Urinalysis and her beta human chorionic gonadotropin (bHCG) were negative. A blood gas confirmed normal physiology. Further investigation with a CT abdomen and pelvis with contrast showed no evidence of free air or fluid in the abdomen. The liver, gallbladder and pancreas were unremarkable. Prominent adnexa with multiple cystic lesions were noted bilaterally. No bowel loop distension was observed. The fat halo sign of the colon was non-specific. Normal alignment of the spine and normal height of the vertebral bodies were noted. A transabdominal and transvaginal ultrasound scan (USS) showed a normal sized, shape and position of her uterus. Both ovaries appeared normal and there was no evidence of an adnexal cyst, mass or free fluid. An USS abdomen was unremarkable. A repeat CT of her abdomen due to increased symptom severity failed to identify a cause for her persistent symptoms. She declined further investigation with flexible sigmoidoscopy. She developed weakness and an altered sensation of her left leg. She was reviewed by the neurologist, who reported a 6-year history of recurrent similar presentations, thought functional in origin.

Physical examination confirmed reduced power 3/5 of her left leg. Bilateral symmetrical reflexes and down-going plantars were observed. Cerebellar examination was normal. Cranial nerves I–XII were intact. An MRI head showed normal cerebral and cerebellar parenchyma, ventricles and midline/posterior fossa. An MRI of her thorax and spine showed a prominent central canal/syringomyelia which was unchanged from her MRI in 2017. She developed right-sided myoclonic jerks with increased left leg pain. This event was self-terminated and her lactate was 0.9. She developed severe headache and photophobia, however, a CT head showed no evidence of intracranial haemorrhage, acute infarction, space occupying lesion, brain oedema or hydrocephalus. Her behaviour became aggressive towards medical staff, demanding further investigations, higher dose opioids and an operation to alleviate her abdominal pain.

## DIFFERENTIAL DIAGNOSIS

This patient presented with a constellation of symptoms representing features of possible SSD and comorbid psychiatric conditions. She was reviewed by psychiatry. She described a 3-week history of low mood and social withdrawal. She reported changes to her sleep, energy and appetite. She denied suicidal ideation. She stated that her low mood was triggered by her abdominal pain and leg weakness and she was concerned she had a serious health problem. She continued to demand an operation to 'fix her pain'. No change was made to her medications and a psychology review was sought. She was reviewed by the pain team as she was demanding higher dose opioids for pain control.

## TREATMENT

She refused to engage with the physiotherapists opting to stay in bed rather than make an effort to mobilise. As there is now global recognition that obesity is a distinct disease that warrants metabolic investigations and management, she was referred to the dietician. She failed to engage with this service and expressed no desire to lose any weight or understand the implications of her high BMI on her health.

## OUTCOME

Our patient fulfilled the criteria to render a diagnosis of SSD. She reported a history of distressful symptoms over a period of 6 years resulting in periods of dysfunction and low mood. During this admission, she exhibited disproportionate thoughts and behaviours in response to her symptoms, most notably for her abdominal pain. She would persistently request further investigations including demanding a laparoscopy to 'find something to fix' despite having multiple USS, CT and MRIs which were unchanged from previous admissions.

Following psychological review, she was referred to the 'medically unexplained symptoms' Clinic in the community. After 3 weeks of supportive care and extensive investigations, she was discharged home uneventfully. This case demonstrates the practical and ethical dilemma that confronts physicians and surgeons when such cases are encountered.

## DISCUSSION

John Bonica revolutionised the study of pain through his insistence that pain warranted attention as a symptom and not only as an indicator of underlying disease. Historically, somatisation is akin to hysteria and hypochondriasis. In 1908, Stekel introduced the term as 'a deep seated neurosis akin to the mental mechanism of conversion'.[2] Somatisation is an important public health problem because it accounts for significant functional disability and healthcare utilisation. Somatisation describes a constellation of clinical and behavioural features indicating that a patient is experiencing and communicating psychological distress through physical (somatic) symptoms not accounted for by pathological findings. Its aetiology is multifactorial and individual, family and environmental factors have been proposed as predisposing, precipitating or perpetuating in somatisation. Individuals present to primary care or to the emergency department with physical symptoms without an organic cause which may be labelled functional, somatic or medically unexplained symptoms and may prove challenging for physicians to address.

The term 'somatic symptom disorder' was introduced in 2013 with publication of the DSM-5 in 2013. It has a female predilection with an estimated prevalence of 5% in the general population and 25% of individuals develop a chronic somatic illness. Prevalence rates are higher in patients with functional disorders such as fibromyalgia, irritable bowel syndrome and chronic fatigue syndrome. The diagnosis of SSD is not a diagnosis of exclusion, on the contrary, it is a positive diagnosis, based on solid criteria.[3] Diagnostic criteria for SSD include one or more somatic symptoms that are distressing or result in significant disruption of daily life and excessive thoughts, feelings or behaviours related to the somatic symptoms or associated health concerns.[4] Affected individuals experience substantial impairment in social functioning with the risk of progressive social withdrawal and hence a negative impact on the functioning of the entire family.

Subsets of SSD include the following: conversion disorder, factitious disorder, illness anxiety disorder, psychological factors

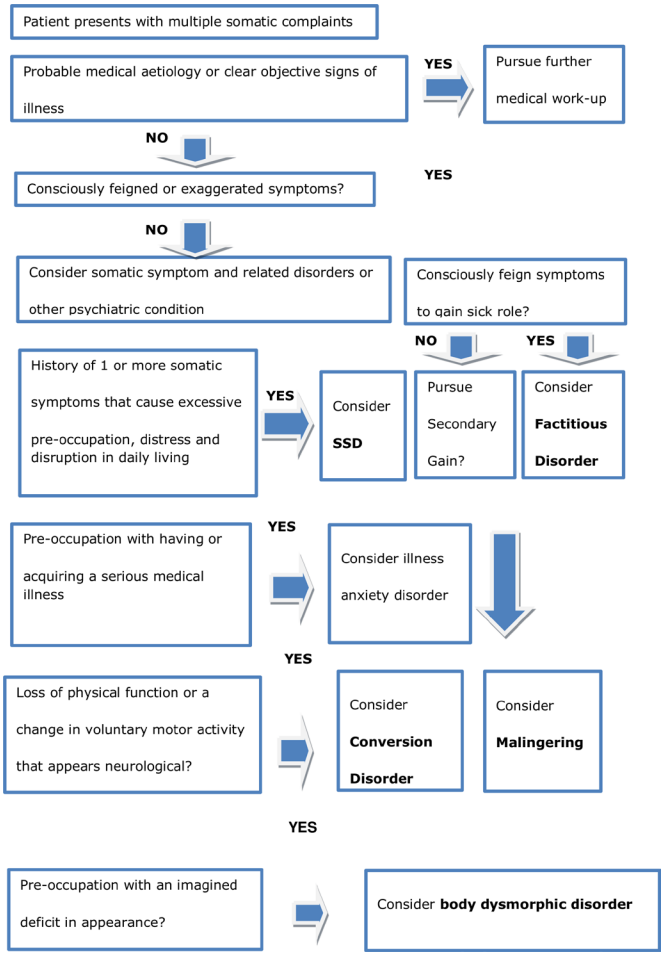


**Figure 1** Diagnostic considerations in the patient with multiple unexplained symptoms.

affecting other medical conditions, other specified somatic symptoms and related disorders and unspecified somatic symptoms and related disorders.[4] Medically unexplained symptoms present to most hospital specialities and account for a considerable proportion of consultations by frequent attenders in secondary care.[5] This group of disorders account for one-fifth of presentations to general physicians with headache, fatigue and abdominal pain, for which no biochemical cause can be detected.[6] It has been suggested that patients suffering from medically unexplained symptoms are less aware of their symptoms and use maladaptive coping strategies when coping with problems (figure 1).[7]

Pain is an inherently subjective phenomenon and Henry Beecher's pain research has shown that there is no direct correlation between the amount of tissue damage and the level of pain experienced. Clinical somatisation arises from patients situated at various points along a continuum of distress severity; this distress is manifested in heightened symptom perception and illness behaviour. Several psychosocial factors may be involved in the pathogenesis of SSD, such as developmental factors, physical and sexual abuse, cognitive and perceptual distortions and behavioural abnormalities as well as difficulties with self-expression. Life change has been correlated with the onset of physical and psychological disorders. Family conflict is another recognised risk factor for its pathogenesis. Somatic symptoms manifesting as gastrointestinal symptoms such as nausea, vomiting, abdominal pain, bloating and diarrhoea are common in individuals with a depressive disorder. Pain symptoms include joint pain, back pain, headache and chest pain. Neurologic symptoms include movement disorders, sensory loss, weakness and paralysis. Distress and somatisation are highly correlated.[8] Hypochondriasis often occurs with somatisation. Increasing levels of anxiety, depression and somatisation are associated with higher preprandial and/or postprandial gastrointestinal symptom levels in inflammatory bowel disease. The natural course of the disorder can be variable with periods of flare-ups associated with previous substance abuse, anxiety and affective disorder.[9] There is growing evidence of childhood abuse in women with somatisation disorder and borderline personality disorder. Typically, functional abdominal pain presents as a diffuse or periumbilical pain. Children may present with loss of vision, hearing or mutism. Poloni performed an observational retrospective study to profile clinical and sociodemographic characteristics of patients with medically unexplained physical symptoms.[10] Reported symptoms included headache, seizures, vertigo, fibromyalgia, paraesthesia, visual disturbance and amnesia. The diagnosis was somatoform disorder in 6.3%, conversion disorder in 2.7% and somatoform disorder in 6.3%.[10] Comorbid psychiatric disorders may precede the development of somatic symptoms but often develop during the course of the somatoform disorder. Depression, panic disorder, generalised anxiety disorder, substance misuse and non-psychiatric medical conditions should be considered in the differential diagnosis. The Somatic Symptom Scale shows promise in measuring somatic symptom burden. A common feature of these patients is that they often undergo an extensive, repeated and poorly justified diagnostic work-up, sometimes magnified by 'doctor shopping' in the constant search for different medical opinions.[11]

While it is acknowledged that it is difficult to render an exact diagnosis in this complex group of SSDs, it is important to support effective recognition and response to the needs of this complex patient group. Once patients develop SSD, it may be perpetuated by chronic stressors and maladaptive coping skills. In addition, behaviour related to the symptoms and sick role add another psychological dimension that maintain the disorder.[12] Management requires a multifaceted approach tailored to the individual patient. SSDs are generally chronic, however, 50%–75% of individuals show improvement and 10%–30% deteriorate. Positive prognostic indicators include fewer physical symptoms and better functioning at baseline. Training in the diagnosis and management of this disorder is inadequate in the postgraduate

## Learning points

- Somatisation describes a constellation of clinical and behavioural features indicating that a patient is experiencing and communicating psychological distress through physical (somatic) symptoms not accounted for by pathological findings.
- Somatic symptom disorder (SSD) was derived in part from the somatoform disorders (somatisation disorder, hypochondriasis) which were eliminated from Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition.
- Anxiety disorders and/or depressive disorders are common in SSD. The key to establishing whether the patient with a medical disorder also has SSD is determining whether the cognitive, emotional and behavioural responses to the medical condition is excessive compared with most other patients with that medical disorder.

curriculum. Scientific research in this field is required. Rehabilitation modelling and behavioural intervention is effective and a bio-psycho-social framework should be used for assessment and intervention. Techniques can be developed to deal with specific symptoms and impairments, for example, distraction, muscular relaxation for headaches, graded physical exercise for muscular problems and fatigue as well as practical management of pseudo-seizures.

Psychological interventions, such as cognitive behavioural therapy is recommended for the management of co-morbid emotional disorders.[13]

A Cochrane review of 21 studies found that all psychological therapies included in the review (cognitive behavioural therapy (CBT), mindfulness, psychodynamic and integrative therapy) were superior in reduction of symptom severity.[14] Longitudinal studies have shown that 90% of SSDs last longer than 5 years.[15] Systematic reviews and meta-analysis have revealed that therapeutic interventions only demonstrate a small-to-moderate effect.[16]

Individuals affected by SSD frequently attend the emergency department and physicians have the unique opportunity to identify this condition according to the DSM criteria and have a positive effect on the patient's prognosis.

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