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Early-Onset Somatic Delusional Presentation: A Case Report and Two-Decade Comparative Synthesis

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Abstract

Objective: Somatic-type delusional disorder is characterized by fixed false beliefs related to bodily changes or illness. This report describes an unusual early-onset case associated with significant functional decline and compares its features with previously published cases.
Method: Clinical information was collected through psychiatric assessment, family interviews, physical and neurological examinations, laboratory investigations, and six months of follow-up. A narrative review of relevant English-language case reports published between 2003 and 2025 was also undertaken.
Results: A 22-year-old woman developed a persistent belief that self-inflicted cuts on her feet had permanently altered her joints and skin. Despite repeated reassurance and normal medical findings, she remained convinced of the perceived deformity. The condition was associated with depressed mood, social isolation, poor self-care, reduced food intake, and academic impairment. Investigations did not identify an underlying medical cause. Treatment with olanzapine, psychoeducation, supportive psychotherapy, and family counselling resulted in gradual improvement. During follow-up, delusional conviction decreased substantially, accompanied by better self-care, social interaction, and academic functioning. Review of the literature indicated that severe impairment and early onset are relatively uncommon in somatic-type delusional disorder.
Conclusion: Early-onset somatic delusions can lead to marked psychosocial dysfunction and may differ from the traditionally described pattern of preserved functioning. Comprehensive assessment, ongoing diagnostic review, and combined pharmacological and psychosocial interventions are important for achieving favorable outcomes.

Key words: *Activities of Daily Living; Age of Onset; Cognitive Behavioral Therapy; Delusions; Diagnosis, Differential; Olanzapine; Patient Education as Topic; Psychotic Disorders*

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Somatic delusions are fixed false beliefs centered on bodily disease, deformity, infestation, or abnormal bodily functioning. In clinical practice, these presentations often lead patients to seek repeated evaluations in dermatology, internal medicine, surgery, neurology, and other non-psychiatric settings before receiving a psychiatric referral (1). Timely recognition is clinically important because repeated investigations and referrals can inadvertently strengthen conviction, extend functional disruption, and delay effective psychiatric intervention.

Within the schizophrenia-spectrum and related disorders, delusional disorder (DD) is characterized by the presence of one or more delusions lasting at least one month, without the prominent hallucinations, disorganized thought processes, or enduring negative symptoms that typify schizophrenia. Subtypes are defined by the predominant delusional theme, with the somatic type characterized by a persistent belief of bodily abnormality or disease. Closely related descriptions in earlier literature used the term monosymptomatic hypochondriacal psychosis (MHP) to emphasize the apparently single-theme, circumscribed nature of the delusion, particularly within liaison psychiatry and psychodermatology contexts (2).

Although DD has traditionally been described as involving relatively preserved functioning outside the delusional focus, accumulating evidence suggests that this distinction is not consistently robust. Comparative studies examining DD in relation to schizophrenia continue to debate over whether DD represents a categorically distinct disorder or falls along a spectrum with schizophrenia across symptom dimensions, cognitive profiles, and functional outcomes (3). Longitudinal data from first-episode psychosis cohorts further indicate that a subset of patients initially diagnosed with DD later meet the criteria for schizophrenia, reinforcing the importance of careful diagnostic formulation and ongoing reassessment over time rather than relying on a single cross-sectional evaluation (4).

Two considerations are particularly relevant when somatic delusions occur early in life and are associated with marked impairment. First, regarding age of onset: DD is more commonly described as emerging later than schizophrenia, often during adulthood; thus, early-onset cases warrant close scrutiny for alternative diagnoses, including schizophrenia-spectrum disorders and mood disorders with psychotic features (5). Second, regarding global functional impairment: severe self-neglect, a sustained inability to maintain basic self-care, the refusal of food or water, and pronounced psychomotor inactivity are difficult to reconcile with a strictly "monosymptomatic" formulation and may indicate broader psychopathology, even when the delusional content is narrow and well systematized (3). These clinical features affect risk assessment, the intensity of treatment planning, and the need for close follow-up.

The evidence base for the treatment of DD remains limited due to the condition's relative rarity and the predominance of case reports and small case series. Nevertheless, available reviews support the use of antipsychotic medications, with reported benefits for across both first-generation and second-generation agents, and frequently emphasize combining pharmacotherapy with psychoeducation, family engagement, and psychotherapy adapted to limited insight (6,7). Earlier summaries also noted that improvement may occur across different antipsychotic choices, while depressive symptoms and treatment adherence frequently influence outcomes—factors that remain relevant in interpreting the clinical response. Contemporary clinical discussions continue to support a multimodal approach and recommend a broad differential diagnosis in somatic presentations, including obsessive–compulsive and related disorders (such as body dysmorphic disorder), mood disorders with psychosis, substance / medication-induced psychosis, and psychosis due to medical conditions.

Rationale and objectives: We present the case of a young woman with a persistent somatic delusion beginning in late adolescence, accompanied by depressive symptoms and severe functional deterioration. Because both (i) early onset and (ii) global impairment are atypical in classical monosymptomatic descriptions, this case raises an important diagnostic question regarding the boundaries with schizophrenia-spectrum and psychotic mood disorders. Accordingly, our aims are: (1) to describe the clinical course, investigations, treatment, and outcome; (2) to provide a two-decade PubMed-based comparative synthesis (2003–2025) of similar published cases with attention to age at onset, functional outcomes, and treatment strategies; and (3) to discuss the differential diagnostic implications and the value of longitudinal follow-up when diagnostic stability is uncertain.

Materials and Methods

Study Design

This article reports on a single clinical case with longitudinal follow-up. A case-report format was used to document the presenting complaints, symptom chronology, mental status findings, investigations, diagnostic formulation (including differential diagnosis), treatment, and clinical outcomes over time. The methodological workflow is summarized in Figure 1.

Comparative Literature Synthesis

In addition to the clinical case description, a focused PubMed-based narrative comparative synthesis was undertaken to contextualize the present case. English-language case reports and closely comparable case-based

publications from 2003 to 2025 were identified using various combinations of the search terms "somatic-type delusional disorder," "somatic delusional disorder," "monosymptomatic hypochondriacal psychosis," "delusional infestation," and related somatic-delusion terms. Reports were reviewed narratively to extract the age at onset or presentation, core delusional themes, key medical work-up, treatments, and functional outcomes. This synthesis was designed to provide clinical context for Table 3 and was not intended as a formal systematic review.

Patient and Setting

The patient was a 22-year-old unmarried postgraduate student who was referred for psychiatric assessment after repeatedly seeking help for bodily concerns in non-psychiatric settings. Clinical data were obtained through direct interviews with the patient and supplemented with collateral information from close family members who accompanied her and provided observations regarding symptom evolution, functional changes, and adherence to treatment.

Clinical Assessment Procedure

The assessment was based on serial, semi-structured psychiatric interviews conducted at baseline and during follow-up. Interviews explored the onset, progression, and stability of the somatic belief, the degree of conviction, and behavioral consequences such as reassurance seeking, avoidance, and withdrawal from daily activities. Psychotic symptoms beyond the somatic delusion—including hallucinations, disturbances in thought form, and negative symptoms—were systematically assessed. Mood and anxiety symptoms were evaluated by focusing on their timing in relation to the somatic belief, and risk assessment included screening for suicidal ideation and behaviors that could compromise physical safety. Functioning was assessed clinically through the documentation of academic engagement, social interaction, daily routines, sleep and appetite, and capacity for self-care, with particular attention to periods of self-neglect and refusal of food or water. A full mental status examination was recorded at baseline and reviewed across follow-up visits to track changes in affect, thought content, insight, judgement, and overall clinical stability.

Measurement of Delusional Conviction and Symptom Change

Validated psychometric instruments for psychosis severity, mood symptoms, or functioning were not administered because this was a routine clinical case report, which is recognized as a limitation. The "percentage of delusional conviction" was reported as a clinician-estimated descriptive indicator derived from repeated interviews using consistent questioning regarding the patient's certainty in the belief and willingness to consider alternative explanations. These estimates are presented to illustrate longitudinal change and are not intended to represent standardized measurements.

Medical and Neurological Evaluation

A medical evaluation was conducted to exclude organic conditions that might account for the patient's somatic complaints or contribute to her psychiatric symptoms. This included general physical examination with attention to dermatological findings and signs of nutritional compromise, review of laboratory investigations (summarized in Table 1), and neuroimaging to rule out structural brain pathology. Any abnormal findings were interpreted within the clinical context, particularly whether they were more consistent with the consequences of reduced intake and self-neglect or suggestive of a primary medical disorder. When clinically indicated, substance-related causes were also considered to reduce the likelihood of substance- or medication-induced psychotic symptoms.

Diagnostic Formulation and Differential Diagnosis

The diagnostic formulation was guided by DSM-5-TR criteria and a structured differential diagnostic process. A working diagnosis of delusional disorder, somatic type (historically referred to as monosymptomatic hypochondriacal psychosis) was established because the psychotic content was dominated by a persistent, well-systematized somatic belief and there was no evidence of prominent hallucinations, disorganized thought processes, or sustained negative symptoms at the time assessment. However, because the presentation included atypical features—particularly an onset in late adolescence and marked functional decline with self-neglect and intermittent refusal of basic needs—so the formulation was maintained provisionally and interpreted cautiously. Schizophrenia-spectrum disorders were considered in view of the early onset and functional impairment, while psychotic mood disorders were evaluated by examining whether the mood symptoms preceded the delusional belief or represented a distinct mood episode. Disorders characterized by excessive health or appearance concerns without delusional intensity were also considered but were less consistent with the fixed and reassurance-resistant nature of the belief. The diagnostic plan therefore included longitudinal reassessment to monitor whether the syndrome remained circumscribed or evolved into a broader psychotic disorder over time.

Treatment and Follow-up Monitoring

Management involved a combination of pharmacological and psychosocial interventions. Antipsychotic treatment (olanzapine; dose specified in the case description) was initiated with clinical monitoring for tolerability and adverse effects. Psychoeducation was provided to the patient and her family to promote engagement and treatment adherence and to reduce misunderstandings that could reinforce distress. Supportive psychotherapy

addressed her distress and coping mechanisms in the context of the precipitating stressor, while cognitive-behavioral strategies were introduced in a manner appropriate for her limited insight. Family counselling focused on reducing repeated confrontations concerning the belief and strengthening supportive, non-confrontational responses that encouraged adaptive functioning. Follow-up visits were conducted monthly for six months, documenting changes in clinician-estimated delusional conviction, mood symptoms, self-care, eating and drinking behavior, academic functioning, social participation, and safety indicators.

Ethics and Consent

Written informed consent was obtained from the patient for the publication of this case report and the associated clinical images. Identifying details were removed or anonymized to protect confidentiality, and ethical requirements were followed in accordance with local institutional policy for anonymized single-case reports. The authors confirm that the clinical photographs, magnetic resonance images, flow diagram, and clinical progress figure were generated from the patient clinical material and the authors own data analysis and were not copied from external sources.

Case Presentation

History of Present Illness

Ms. J, a 22-year-old unmarried postgraduate student, presented with a four-year history of persistent preoccupation with perceived bodily deformities. Her symptoms began at age 18, when she intentionally incised the sole of her foot in an attempt to alter her "destiny lines," believing this would change her future trajectory. In subsequent years, she developed a fixed conviction that her joints were deformed and her skin permanently scarred as a result of these injuries. Despite multiple normal medical evaluations and repeated reassurance from family members and physicians, her belief remained unshaken.

Approximately seven months before presentation, her condition worsened markedly following the abrupt cancellation of her engagement—a significant psychosocial stressor. She developed a sleep disturbance characterized by sleeping three to four hours nightly, a poor appetite accompanied by an approximately 5 kg weight loss over two months, frequent crying spells, and increased irritability. She became socially withdrawn, neglected personal hygiene, and occasionally refused food and water. Family members observed her sitting motionless for prolonged periods, ruminating on her bodily appearance. Attempts to challenge her beliefs often provoked intense anger and emotional outbursts.

Past, Family, and Personal History

Premorbidly, Ms. J was well-adjusted, academically successful, and socially active. There was no history of psychiatric illness, substance use, or significant medical comorbidity. Her family history was negative for psychotic, affective, or substance-related disorders, and all of her developmental milestones had been achieved appropriately.

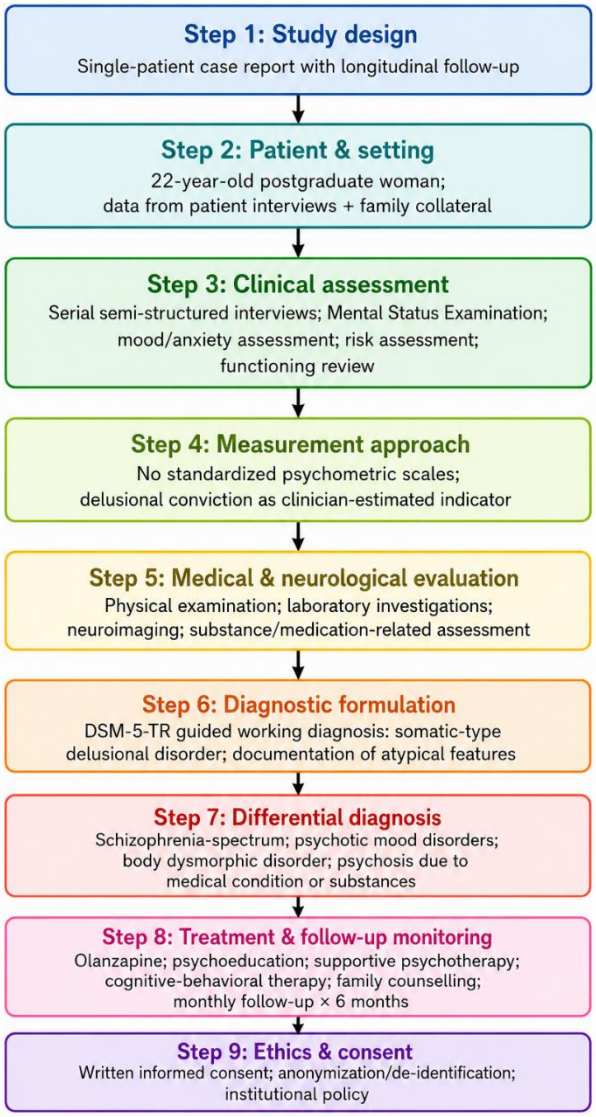

Figure 1. Methodological Workflow for the Clinical Case Report and Two-Decade Comparative Synthesis

Sequential overview of the clinical workflow used in this study: case-report design and data sources (patient interviews and family collateral data), serial psychiatric assessments (including the mental status examination, mood / anxiety and risk evaluations, and functional assessment), descriptive symptom tracking (clinician-estimated delusional conviction in the absence of standardized scales), medical and neurological investigations (physical examination, laboratory tests, and neuroimaging), a DSM-5-TR–guided diagnostic formulation with a structured differential diagnosis,

combined pharmacological and psychosocial treatment with monthly follow-ups over six months, and ethical procedures including written informed consent and anonymization.

Mental Status Examination

General physical examination revealed mild pallor with a body mass index (BMI) of 19.2 kg/m². Vital signs were stable (BP 108/70 mmHg, HR 82/min, and she was afebrile). Cardiovascular, respiratory, abdominal, and neurological assessments were unremarkable. Dermatological examination demonstrated multiple superficial, well-healed linear scars over the forearms, hands, and feet, consistent with self-inflicted injuries and without evidence of active inflammation or infection (Figure 2).

Comprehensive laboratory investigations were largely within normal limits, except for mild microcytic anemia and vitamin D insufficiency, as summarized in Table 1. A T1-weighted brain MRI revealed normal cortical and subcortical structures with no frontal or temporal pathology (Figure 3), thereby excluding structural neurological causes for her somatic preoccupations.

Investigations

A comprehensive laboratory evaluation was undertaken to exclude organic pathology. The most notable abnormality was mild microcytic anemia, with a hemoglobin level measured at 9.2 g/dL. Other hematological parameters, renal and liver function tests, fasting glucose, and electrolytes were within normal limits. The thyroid profile and vitamin B12 levels were normal, while 25-hydroxy vitamin D levels were reduced at 21 ng/mL, consistent with insufficiency. A detailed summary of laboratory findings is provided in Table 1.

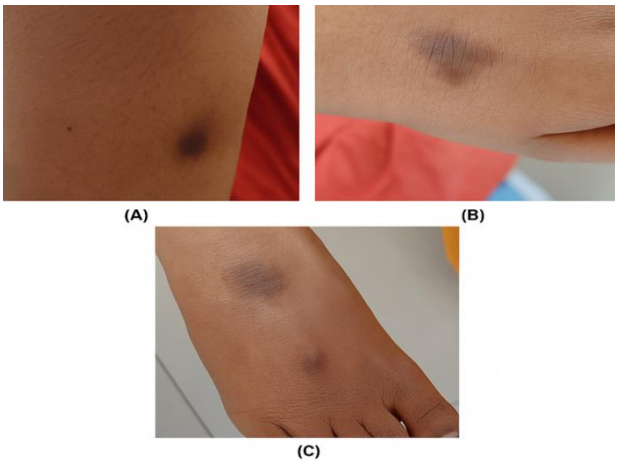

Figure 2. Cutaneous Findings Showing Stable Self-Inflicted Scars and Hyperpigmented Lesions in a Patient with Somatic Delusional Presentation

Figure 2. (A) Hyperpigmented macule on the forearm. (B) Lichenified hyperpigmented patch on the dorsum of the hand. (C) Well-healed hyperpigmented plaque with scarring on the dorsum of the foot. All lesions were stable, non-tender, and without signs of active inflammation, consistent with a psychogenic/self-inflicted origin in the context of a somatic delusional presentation

Figure 3. Normal T1-Weighted Brain Magnetic Resonance Imaging Excluding Structural Neurological Pathology in a Patient with Somatic Delusional Presentation

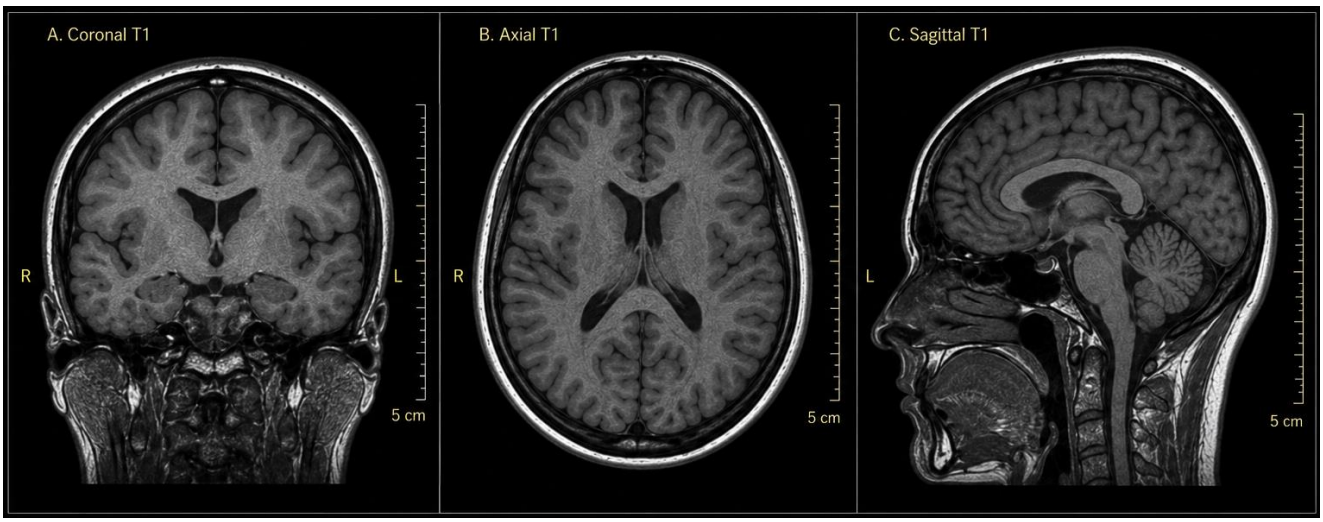

Figure 3. Coronal (A), axial (B), and sagittal (C) T1-weighted MRI views of the brain demonstrating normal cortical and subcortical anatomy. The cerebral hemispheres were symmetrical, the ventricles were of normal size and configuration, and no structural abnormalities were evident in the frontal or temporal lobes. These findings support the absence of organic pathology contributing to the patient's somatic delusions.

Table 1. Comprehensive Laboratory and Clinical Investigations in a Patient with an Early-Onset Somatic Delusional Presentation

Test Category	Parameter	Patient Result	Reference Range	Units	Clinical Significance & Rationale
Complete Blood Count (CBC)	Hemoglobin (Hb)	9.2	12.0–15.5	g/dL	Mild anemia, most consistent with nutritional deficiency and self-neglect; common in psychiatric inpatients.
	Hematocrit (Hct)	35	36–46	%	Slightly reduced; confirms anemia.
	Red Blood Cell Count (RBC)	4.4	4.0–5.2	$\times 10^{12}$/L	Normal range; excludes bone marrow suppression.
	Mean Cell Volume (MCV)	79	80–100	fL	Borderline low; supports iron-deficiency anemia.
	Mean Cell Hemoglobin (MCH)	26	27–32	pg	Slightly decreased; reinforces microcytic anemia pattern.
	Mean Cell Hemoglobin Concentration (MCHC)	310	320–360	g/L	Mildly reduced; consistent with iron deficiency.
	Red Cell Distribution Width (RDW)	15.2	11.5–14.5	%	Elevated; indicates anisocytosis, typical in early nutritional anemia.
	White Blood Cell Count (WBC)	6.1	4.0–11.0	$\times 10^{9}$/L	Within normal range; excludes leukocytosis or infection.
	Neutrophils (absolute)	3.4	2.0–7.5	$\times 10^{9}$/L	Normal; no evidence of bacterial infection.
	Lymphocytes (absolute)	2.1	1.0–4.0	$\times 10^{9}$/L	Within normal range; intact immune profile.
	Platelet Count (PLT)	310	150–400	$\times 10^{9}$/L	Normal hemostatic function.
Comprehensive Metabolic Panel	Fasting Glucose	92	70–110	mg/dL	Normoglycemi; excludes diabetes mellitus.
	Blood Urea Nitrogen (BUN)	14	7–20	mg/dL	Normal renal excretory function.
	Creatinine	0.7	0.6–1.2	mg/dL	Normal; excludes renal impairment.
	Estimated GFR	>90	>60	mL/min/1.73m^2	Preserved renal function.
	Sodium (Na^+)	139	135–145	mmol/L	Normal; excludes hyponatremia or dehydration.
	Potassium (K^+)	4.1	3.5–5.0	mmol/L	Normal; excludes hypokalemia, which can mimic psychiatric symptoms.
	Chloride (Cl^-)	103	98–107	mmol/L	Normal electrolyte status.

Test Category	Parameter	Patient Result	Reference Range	Units	Clinical Significance & Rationale
	Carbon Dioxide (CO_2)	24	22–28	mmol/L	Normal acid–base balance.
	Calcium (total)	9.0	8.5–10.5	mg/dL	Normal; excludes metabolic bone disease.
	Albumin	4.1	3.5–5.0	g/dL	Normal protein synthesis; nutritional status relatively preserved.
	Total Protein	7.3	6.0–8.3	g/dL	Normal total protein status.
Hepatic Function Panel	ALT	22	7–35	U/L	Normal hepatocellular integrity.
	AST	26	8–35	U/L	Normal liver and cardiac enzyme profile.
	Alkaline Phosphatase (ALP)	92	44–147	U/L	Normal; excludes cholestasis or bone disease.
	Total Bilirubin	0.6	0.2–1.2	mg/dL	Within normal limits; excludes hepatic dysfunction or hemolysis.
	Gamma-Glutamyl Transferase (GGT)	20	9–48	U/L	Normal hepatobiliary function.
Endocrine Screening	Thyroid Stimulating Hormone (TSH)	2.1	0.4–4.0	mIU/L	Normal thyroid profile; excludes thyroid-induced psychiatric symptoms.
	Free T4	Not performed	0.9–1.7	ng/dL	Not indicated due to normal TSH.
Nutritional Assessment	Vitamin B_{12}	315	200–900	pg/mL	Adequate; excludes B_{12} deficiency psychosis.
	Folate (serum)	7.8	3–17	ng/mL	Within normal range.
	25-Hydroxy Vitamin D	21	30–100	ng/mL	Insufficiency; may contribute to low mood and fatigue.
Toxicology / Substance Screening	Urine Drug Screen	Negative			Excludes substance-induced psychosis.
	Serum Ethanol	<10		mg/dL	Within safe range; excludes alcohol intoxication.
Specialized Assessments	Physical Examination	Mild pallor, otherwise normal			No systemic organic pathology detected.
	Vital Signs	Stable			No acute medical illness.
	Dermatology Consultation	Self-inflicted, well-healed			Confirms non-organic etiology of cutaneous findings.

Test Category	Parameter	Patient Result	Reference Range	Units	Clinical Significance & Rationale
		scars (Figure 2)			

Based on DSM-5-TR criteria, the presentation was most consistent with a working diagnosis of delusional disorder, somatic type (historically described as monosymptomatic hypochondriacal psychosis). This formulation was retained provisionally because the belief had been persistent, well systematized, reassurance-resistant, and present for more than four years, without prominent hallucinations, formal thought disorder, or sustained negative symptoms on assessment. However, the early age of onset and the degree of functional deterioration required ongoing differential diagnostic consideration, particularly regarding schizophrenia-spectrum disorders and psychotic mood disorders. Although cognitive functions were preserved, diagnostic stability required longitudinal follow-up rather than categorical certainty based on a single cross-sectional assessment.

Secondary features included depressive symptoms, such as pervasive low mood, anhedonia, irritability, crying spells, and disturbed sleep and appetite, all of which were precipitated by the cancellation of her engagement. Laboratory evaluation also revealed nutritional anemia (Hb 9.2 g/dL, with borderline microcytosis) and vitamin D insufficiency (21 ng/mL), which were plausibly attributable to poor nutrition and the neglect of self-care. Functionally, she exhibited severe impairment, marked by withdrawal from academic and social roles and a significant deterioration in personal hygiene. These features underscored both the clinical burden of the presentation and the need for a cautious longitudinal reassessment of the diagnosis.

Pharmacotherapy

Treatment was initiated with olanzapine 10 mg nightly, selected on the basis its reported benefit in prior case-based literature on somatic delusional presentations and a favourable tolerability profile relative to some first-generation antipsychotics such as pimozide. The dose was maintained within the therapeutic range. The patient experienced mild sedation during the initial weeks, which resolved spontaneously. No extrapyramidal or metabolic side effects were noted during the six-month follow-up period.

Psychoeducation

A structured psychoeducation program was delivered to both the patient and her family. This focused on the neurobiological basis of her symptoms, the importance of adherence to pharmacotherapy, the early recognition of relapse, and the need to dispel misconceptions that her skin and joints were permanently deformed. Psychoeducation fostered a therapeutic alliance and improved the family's understanding of her illness.

Psychotherapy

She engaged in supportive psychotherapy, emphasizing stress management, emotional regulation, and gradual reality testing. Direct confrontation of delusional beliefs was avoided to reduce resistance. From the third month onward, cognitive-behavioral therapy (CBT) strategies were incorporated, including behavioral experiments and structured cognitive reframing, leading to progressive reduction in conviction strength.

Family Counselling

Given the family's initial tendency to confront her beliefs, family counselling was prioritized. Sessions emphasized empathic listening, non-confrontational communication, and the reinforcement of adaptive behaviors. This intervention significantly improved the home environment and reduced interpersonal conflict.

Clinical Progress

Over the six-month follow-up period, the patient demonstrated steady improvement in both psychopathology and functioning. Her delusional conviction decreased from 90% at baseline to 20% at the end of follow-up, with a concurrent restoration of academic participation and social engagement. Clinical progress was monitored monthly and is summarized in Table 2 and Figure 4.

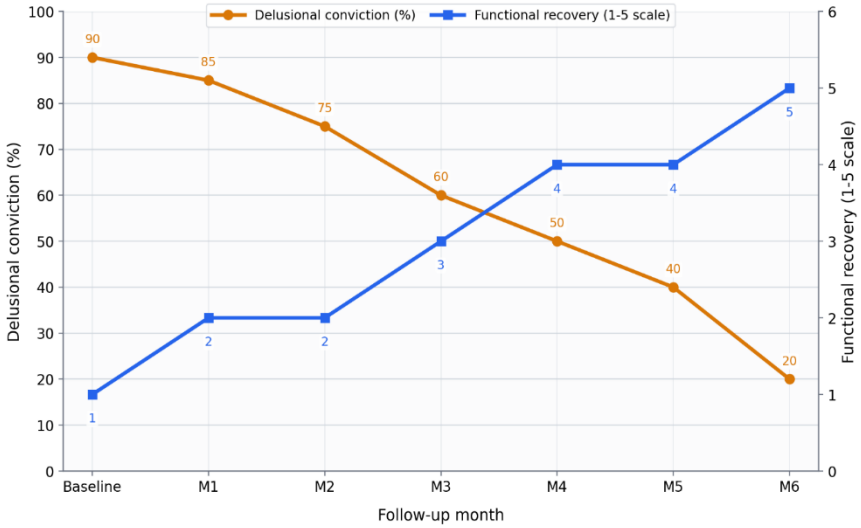

Figure 4. Six-Month Clinical Course Showing Reduced Delusional Conviction and Improved Functional Recovery After Integrated Treatment

Table 2. Six-Month Treatment Adherence, Psychotherapy Engagement, Delusional Conviction, Functional Improvement, and Family Support in a Patient with Early-Onset Somatic Delusional Presentation

Month	Medication Adherence & Side Effects	Psychotherapy & Counseling Engagement	Delusional Conviction (%)	Functional Improvements	Family Support & Dynamics
Baseline	Not on treatment	None	90	Withdrew from academics; poor hygiene	Frequent invalidation, conflict
Month 1	Adherent; mild sedation (resolved)	Initiated supportive psychotherapy; family psychoeducation	85	Sleep improved; began eating regularly	Less confrontational responses
Month 2	Stable adherence; no side effects	Weekly sessions; CBT elements introduced	75	Attended 2–3 classes weekly	Family practiced empathic communication
Month 3	Continued adherence	Insight-building and family joint sessions	60	Daily college attendance; peer interactions resumed	Greater emotional validation
Month 4	Maintained adherence; normal labs	Behavioral experiments, relaxation training	50	Reduced body-checking; joined book club	Family reinforced positive coping
Month 5	Consistent adherence; no adverse effects	Relapse-prevention strategies introduced	40	Regular self-care; daily social activities	Consistent positive reinforcement
Month 6	Continued adherence	Consolidation sessions; family review	20	Full academic reintegration; hopeful outlook	Family prepared for long-term support

Figure 4. Longitudinal trend showing symptomatic and functional improvement. Delusional conviction (orange line, left axis) decreased steadily from 90% at baseline to 20% at Month 6. Functional recovery (blue line, right axis, 1–5 scale) improved in parallel, with the patient progressing from withdrawal and neglect at baseline to full academic and social reintegration by Month 6. These improvements corresponded with consistent adherence to

olanzapine, supportive psychotherapy with CBT elements, structured psychoeducation, and family counseling.

Discussion

This case describes a somatic delusional presentation that remained clinically coherent in its narrow, well-systematized somatic theme and its persistence despite reassurance, while also showing two features with clear diagnostic relevance: an onset in late adolescence and a phase of marked global functional deterioration (8,9). Classical accounts of monosymptomatic hypochondriacal psychosis (MHP) and somatic-type delusional disorder often emphasize circumscribed delusional content with comparatively preserved functioning outside of the delusional focus. In contrast, the present case illustrates how somatic delusional phenomena may present near the boundary between delusional disorder and schizophrenia-spectrum or psychotic mood disorders, thereby supporting a formulation that remains open to diagnostic evolution and that prioritizes longitudinal reassessment (10,11).

Comparative synthesis of published cases (Table 3).

Table 3. Representative Published Case Reports and Diagnostic Reports from the Two-Decade Comparative Synthesis (2003-2025)

Author(s), Year	Age/Sex	Core Somatic Delusion / Theme	Setting & Key Work-up	Treatment	Outcome / Follow-up
Uğuz *et al*., 2003 (Turkish J Clin Psychiatry)	40 M	"Nerve spasm" causing chronic pain → disc surgery and 16 tooth extractions despite normal tests	Repeated normal imaging/labs; no organic cause	Haloperidol + amitriptyline	Partial symptom control; persistent fixed belief; major functional impact.
Kępska *et al*., 2011 (Acta Derm-Venereol)	59 F	"Purulent back lesions spreading internally"; tactile/proprioceptive sensations	Dermatology admission; normal exam except scratch scar; broad labs normal	Sulpiride 200 mg/day + emollients; psych liaison	Slight improvement in 6 months (lessened body sensations); continued outpatient derm + periodic psych care.
Ajiboye & Yusuf, 2013 (Afr J Psychiatry) – two illustrative MHP cases	29 F; 45 M	(1) Olfactory reference (bad body/mouth odour) with social withdrawal; (2) Infestation sensations after sexual contact	Medical work-ups negative; significant psychosocial impairment	(1) Haloperidol; (2) Trifluoperazine+ antidepressant; ECT used during relapse	(1) Maintained on low-dose haloperidol; (2) Recurrent course; partial response; ECT helpful during admission
Barone *et al.*, 2014/15 (J Psychopathology) – Ekbom/infestation	69 F	Worms/eggs under skin, eyes, genitals (matchbox sign)	Normal labs/CT; primary somatic-type delusional disorder	Olanzapine 5→20 mg/day	Marked improvement in ~4 weeks; better sleep/appetite; good alliance.
Ranjan *et al*., 2021 (Psychiatria Danubina) – ECT case	55 F	Food pipe "narrowed," can't swallow	Normal MRI/labs; failed risperidone/olanzapine trials	Bitemporal ECT ×8 + olanzapine	Substantial resolution of delusional conviction; functional recovery; literature summary of prior ECT-responsive somatic delusions included.
Vouk-Kamenski *et al.*, 2021 (Alpha Psychiatry) – Depot SGA	Adult M	Delusional infestation; severe non-adherence, substance use	Dermatology overuse labs/CT/EEG largely normal	Olanzapine pamoate LAIafter oral trial	Delusion faded with improved adherence—first report of olanzapine depot success in DI.
Lochner and Stein, 2003 (Journal of Postgraduate	Not applicable (diagnos	Persistent belief of emitting offensive body odor with referential interpretations;	Differential diagnosis discussed, including psychotic disorders, body dysmorphic disorder, social	Treatment approach depends on formulation; pharmacotherapy	Included to support diagnostic criteria and differential diagnosis rather than single-patient outcome.

Author(s), Year	Age/Sex	Core Somatic Delusion / Theme	Setting & Key Work-up	Treatment	Outcome / Follow-up
Medicine) - olfactory reference syndrome diagnostic report	tic report)	delusional intensity may vary.	anxiety disorder, and medical causes of malodor.	and psychotherapy may be considered.	
Comardelle *et al.*, 2022 (Health Psychology Research) – foreign body	67 F	"Glass under the skin," nail removal, excoriations	Organics ruled out; BMI low; UDS only THC	Olanzapine 5 mg qHS + psychotherapy	Rapid relief within 2 days; discharged stable; argues SGAs over pimozide when pruritus absent.

The patient's withdrawal from academic and social roles, deterioration in hygiene, intermittent refusal of food and water, and periods of psychomotor inactivity represent impairment that is not readily attributable to a circumscribed delusion alone (12). Across many published reports, functional disruption appears to be more directly linked to the delusional theme—such as repetitive medical consultations, checking or avoidance behaviors, and activity restriction—while basic self-care and broader functioning are less severely affected (Table 3). In this context, three clinical interpretations merit consideration. First, severe disability may reflect an affective syndrome (for example, depression following psychosocial stress) superimposed on a longstanding somatic delusion, thereby contributing to reduced intake, psychomotor slowing, and self-neglect. Second, the presentation may represent an early stage of a broader psychotic illness in which functional decline can precede or occur without prominent hallucinations or disorganization during the initial course. Third, profound impairment may occur within somatic-type delusional disorder in a minority of patients, but such presentations may be less likely to be captured in published case reports and therefore remain underestimated (Table 3). Because these possibilities imply different prognoses and follow-up needs, the functional profile is best treated as a central interpretive feature rather than a peripheral clinical detail. The age of onset further contributes to diagnostic uncertainty. Persistent delusional conviction emerging around 18 years of age is uncommon in classical descriptions of delusional disorder, whereas earlier onset is more frequently associated with schizophrenia-spectrum illnesses. Although the age of onset is not diagnostic in isolation, it meaningfully shifts diagnostic probability when accompanied by marked functional impairment. In Table 3, most reported cases involve an adult onset, and adolescent onset is rare or absent, again indicating that the present case does not match the modal pattern in published reports (Table 3). On this basis, the most defensible formulation is a working diagnosis coupled with careful follow-up to detect the potential emergence of broader psychotic symptoms, sustained negative symptoms, or recurrent mood episodes over time (13, 14).

With respect to the differential diagnosis, schizophrenia-spectrum disorders were considered because an early onset and severe functional decline can be characteristic of these conditions. At the same time, several aspects supported a working diagnosis within the somatic-type delusional framework: the delusional content remained narrow and systematized, persistent hallucinations were not evident, formal thought disorder was not a prominent feature, and substantial functional recovery occurred with treatment. Nevertheless, the absence of these features at a single time point does not exclude the evolution of a schizophrenia-spectrum illness, particularly early in the course; therefore, longitudinal reassessment remains essential.

Psychotic mood disorders (major depressive disorder with psychotic features or bipolar disorder) were also considered given depressive symptoms and psychomotor changes. The key clinical question is whether psychotic content is mood-congruent and confined to a mood episode, and whether the mood syndrome precedes and explains the psychosis. In this case, somatic conviction appeared longstanding and not limited to an affective episode, suggesting that mood symptoms were more plausibly secondary to distress and psychosocial stress than the primary generator of psychosis. However, the severity of vegetative and psychomotor symptoms supports continued monitoring for recurrent or distinct mood episodes (15).

Body dysmorphic disorder and somatic symptom disorder can present with prominent preoccupation and repetitive behaviors, yet the belief often retains some capacity for doubt and may fluctuate with reassurance or psychological interventions. Here, the conviction was fixed and persisted despite repeated normal evaluations, supporting a delusional level of belief. Even so, the boundaries between delusional conviction and overvalued ideas may be clinically difficult to define, and documentation should make explicit the features that supported a delusional judgement in this case (16,17).

Psychosis due to medical or substance causes was considered less likely given the absence of supportive medical findings, normal neuroimaging, and a lack of substance use. Nutritional abnormalities (for example, anemia or vitamin D insufficiency) are more plausibly interpreted as contributors to general well-being and mood vulnerability than as primary causes of a well-structured somatic delusion (18).

Treatment response and interpretation in light of the literature (Table 3).

The patient improved with olanzapine combined with psychoeducation, CBT-informed strategies, and family counselling. Table 3 indicates that antipsychotic treatment is commonly reported across published cases, with many recent reports utilizing second-generation antipsychotics and, in some instances, combining pharmacotherapy with psychotherapy and family-based interventions (Table 3). The present case is consistent with this pattern and supports a multimodal approach, particularly when psychosocial stressors and family interactions contribute to distress and the reinforcement of maladaptive behaviors. At the same time, improvement with antipsychotic treatment should be interpreted as a therapeutic outcome rather than as confirmatory evidence of a single diagnostic category, because a response can also be observed in schizophrenia-spectrum and psychotic mood disorders (19).

Diagnostic implications and clinical approach. When somatic delusions occur with an early onset or marked global impairment, the most clinically appropriate stance is to document diagnostic uncertainty, maintain longitudinal follow-up, and avoid overstating categorical certainty. In practical terms, this involves repeated monitoring for hallucinations, thought-form disturbances, negative symptoms, and mood episodes; a structured review of functioning (education/occupation, self-care, social engagement); and family-focused interventions to reduce confrontation, support adherence, and reinforce adaptive behaviors (20). The comparative synthesis (Table 3) suggests that the severity of impairment observed here is not typical of the published case-report phenotype, thereby supporting the interpretation of this presentation as diagnostically boundary-testing rather than representative of "classical" MHP (Table 3).

Limitation

This report is limited by its single-case design, the absence of standardized symptom and functioning scales, and a follow-up period that remains short relative to the longitudinal evolution of psychotic disorders. These limitations are particularly relevant because diagnostic uncertainty is central to the clinical interpretation, and a longer follow-up period with structured assessments would strengthen conclusions regarding diagnostic stability.

Conclusion

In summary, this case broadens the clinical picture of somatic delusional presentations by showing that an early onset and severe functional deterioration may occur and may challenge the assumption of preserved functioning often associated with monosymptomatic formulations. Comparison with published cases (Table 3) suggests that this phenotype is uncommon in the case-report literature, reinforcing the need for careful differential diagnosis, longitudinal monitoring, and integrated pharmacological and psychosocial management.

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Conflict of Interest

The authors declare that there are no conflicts of interest regarding the publication of this case report.

Author's Contributions

Lakshmi Dorai B contributed to conceptualization, clinical supervision, diagnostic formulation, and critical manuscript revision. Alluri Swetha Reddy contributed to clinical data collection, literature search, follow-up documentation, and drafting of the case report. Mukesh B M contributed to clinical assessment, treatment planning, follow-up monitoring, and critical revision. Arbind Kumar Choudhary contributed to literature synthesis, methodological structuring, reference checking, and critical editing. All authors reviewed and approved the final manuscript.

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