Tic Disorders & the Psychomotor Domain in Schizophrenia Spectrum Disorders

A case report and literature review

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Background

We present a **16-year-old female** with a history of global developmental delay (GDD) and mild intellectual disability (ID), initially assessed and treated by paediatric neurology for a provisional tic disorder. Neuroimaging, autoimmune and metabolic investigations were unremarkable.

She did not respond to **Clonidine**. Due to reduced oral intake, weight loss and associated nutritional concerns, treatment was switched to **Aripiprazole**. She was referred to paediatric neuropsychiatry for evaluation of possible co-existing conditions, including catatonia.

Psychiatric assessment — semi-structured clinical history, behavioural observation and mental/physical examination — was suggestive of a **first episode of psychosis**, characterised by auditory hallucinations and associated behavioural change. **CAARMS** (Comprehensive Assessment of At-Risk Mental States) was attempted.

The patient also showed features consistent with catatonia on **BFCRS** (Bush–Francis Catatonia Rating Scale) — psychomotor slowing, social withdrawal, perseveration, negativism, verbigeration and ambitendency. **Facial motor tics** were observed concurrently, with onset temporally associated with the emergence of catatonia and psychotic symptoms.

Optimisation of Aripiprazole produced improvements in oral intake, weight and school attendance, with residual psychotic symptoms, facial tics and catatonia. **Lorazepam** was introduced for catatonia, with ongoing follow-up planned.

Methods

Literature review of publications to **10 March 2026** across **PubMed, Embase, Medline, PsycInfo and EMCare**. After removal of duplicates and irrelevant studies, **9 studies** were included — comprising systematic reviews, conceptual analyses, narrative reviews, cross-sectional and prospective cohort designs.

Results — Key Findings

1 **Neurodevelopmental origin of SSD** — Ref 1

Growing evidence places the origin of schizophrenia spectrum disorders (SSD) early in life. Motor abnormalities are proposed as an important early indicator of vulnerability to SSD.

2 **Sensori-/psychomotor domain renewed** — Ref 2

Over the past decade, the sensory and psychomotor domain has received renewed attention — relevant not only for describing symptoms but for early identification, predicting outcomes and supporting personalised treatment across the lifespan.

3 **Prodromal hyperkinetic signs** — Ref 3

Mittal et al. (2008) found that adolescents with prodromal psychotic symptoms displayed subtle hyperkinetic movement abnormalities, particularly involving the facial area — directly relevant to our case.

4 **Transdiagnostic motor dysfunction** — Ref 4, 5

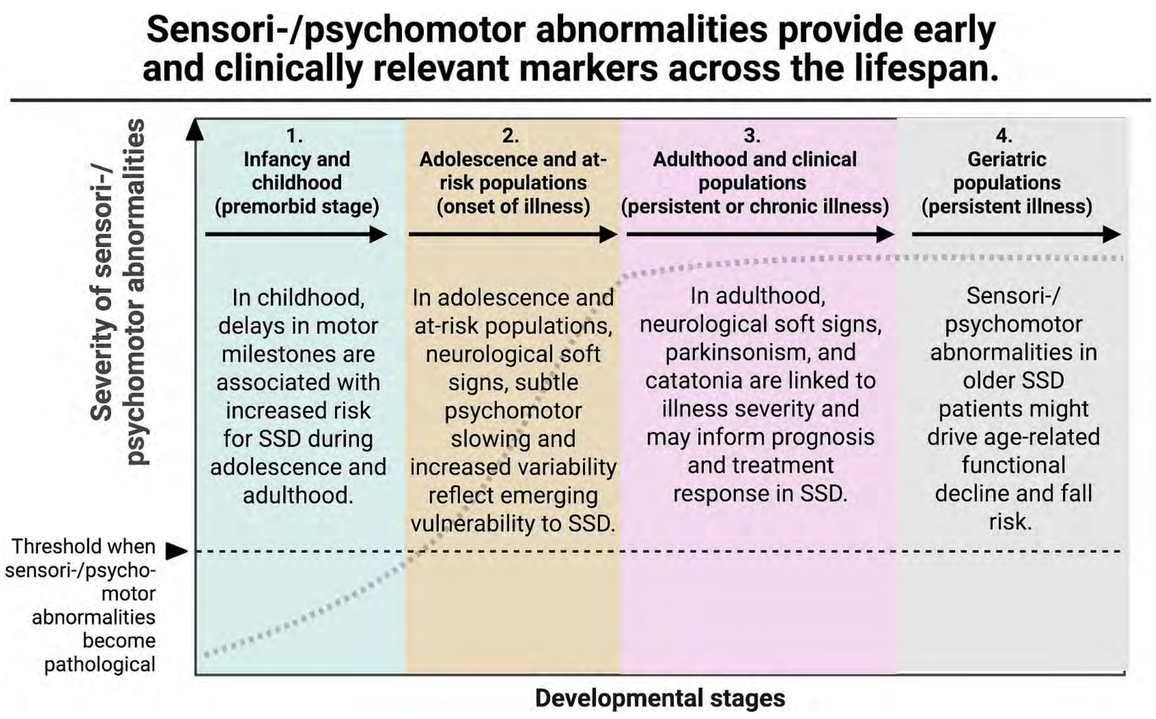
Motor disturbance is common across neuropsychiatric disorders — schizophrenia, OCD, autism, mood disorders — and represents a key transdiagnostic indicator of illness severity. In a study of 55 non-psychotic adults with Tourette syndrome, 87% expressed catatonic signs including echophenomena, perseveration and excitement.

5 **Corollary discharge as mechanism** — Ref 6, 9

Corollary discharge (CD) — a sensorimotor physiology — and its impairment may underpin psychotic experiences and represent a specific pathophysiological link between motor impairment and longitudinal psychotic risk.

6 **Shared neural substrate** — Ref 7, 8

Functional neuroimaging shows that both movement disorders and psychiatric conditions involve shared circuits connecting the basal ganglia to the cerebral cortex. Neurological soft signs (NSS) — subtle deficits in coordination, sensory integration, sequencing and movement inhibition — are frequent in SSD and linked to frontoparietal and cerebellar dysfunction. These same regions generate, regulate and suppress tics, suggesting a possible shared neural substrate.

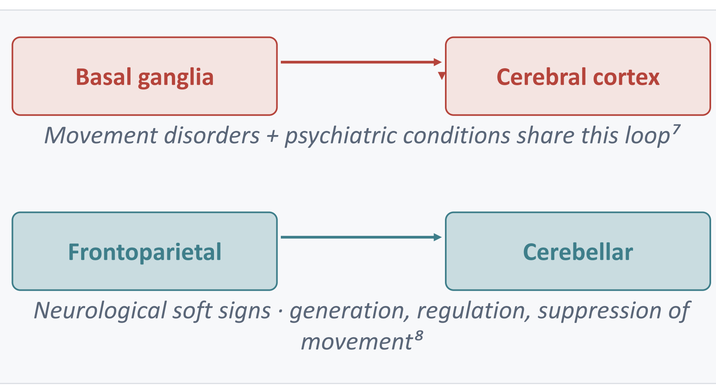


Sensory motor changes in Schizophrenia spectrum disorder across lifespan from Hirjak et. Al. 2025

A Shared Neural Substrate

Tics, catatonia and psychosis converge on overlapping circuits — the substrate for movement generation, regulation and suppression is also implicated in psychotic experience.

Shared Networks



Distinguishing the motor signs

Feature	Tics	Catatonia	NSS
Onset	Childhood, fluctuating	Acute, situational	Stable, lifelong
Voluntariness	Involuntary with suppressibility	Involuntary	Involuntary
Premonitory urge	Yes	No	No
Response to BZD	Partial	Marked (diagnostic)	None
Neuronal Circuitry	Basal ganglia–cortical	Cortico-basal-thalamic	Frontoparietal–cerebellar

Discussion

This case illustrates the diagnostic complexity of co-occurring tics, catatonia and emergent psychosis in an adolescent with developmental vulnerability. Sequential pharmacological treatment — Clonidine, Aripiprazole, Lorazepam — reflects an evolving formulation as new symptoms emerged, rather than treatment failure alone.

Integrating neurophysiological, developmental and phenomenological viewpoints suggests motor abnormalities may not merely reflect general neurodevelopmental disruption but could be **more directly linked to psychosis risk**. The temporal clustering of tics, catatonia and psychotic symptoms in this case is consistent with a shared substrate hypothesis.

Conclusions

Motor abnormalities may not just reflect general neurodevelopmental disruption
— they could be directly linked to the risk of psychosis.

Digital assessment, neuroimaging and AI-based analytics
offer new possibilities for measuring motor dysfunction with accuracy and real-world relevance.

Sensory and psychomotor evaluation should be embedded
in both clinical pathways and research — supported by longitudinal, multimodal designs, open-access data and interdisciplinary collaboration.

Integration into psychiatric care pathways
could enhance diagnosis, treatment strategies and understanding of brain–behaviour relationships in severe mental illness.

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