Psychosocial short stature with psychosis: a case report

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Abstract

Objective: Our objective was to report and describe a case of psychosocial short stature in an adolescent girl with psychotic features. Psychosocial short stature is a rare condition in which emotional stress or deprivation in childhood profoundly reduces growth, leading to persistent short stature. This disorder is variably known as psychosocial dwarfism, hyperphagic short stature or maternal deprivation dwarfism. In the literature, psychosocial short stature has not been associated previously with psychosis.

Conclusions: We formulate that our patient’s short stature, developmental regression and psychotic features were culminations of insecure mother–child attachment, personal traumatic experiences, immigrant status, high family expressed emotions and social isolation. Neuropsychiatric influences were critically regarded due to our patient’s fluctuations in behaviour and affect, in the setting of cortical volume loss on brain MRI. Diagnostic hypotheses included childhood disintegrative disorder or childhood-onset schizophrenia. The management plan involved inpatient family psychoeducation, a pharmacological trial with an atypical antipsychotic and community mental health service follow-up for family therapy and psychotherapy.

Keywords: psychosocial short stature, psychosis, childhood-onset schizophrenia, childhood disintegrative disorder, post-traumatic stress disorder

Psychosocial short stature is a rare condition in which emotional stress or deprivation in childhood profoundly reduces growth, leading to persistent short stature. This disorder is variably known as psychosocial dwarfism, hyperphagic short stature or maternal deprivation dwarfism. We report a case in an adolescent girl with psychotic features. In the literature, psychosocial short stature has not been associated previously with psychosis.

Anna is a 16-year-old girl of Filipino origin, the third of her parents’ four daughters. From age 12 she experienced a progressive decline in multiple functional domains. In the Philippines, her mother’s pregnancy was complicated by varicella infection and lobar pneumonia in the third trimester. Anna’s birth was at term via normal vaginal delivery. Due to her mother’s ongoing physical illness post-natally, cultural custom saw Anna receive primary care from her paternal grandmother until early childhood. Similarly, her mother had been cared for as an infant by a paternal aunt. In hindsight, Anna’s mother described her early mother–child attachment with Anna as superficial; they “hadn’t been close”, compared with her other children. Anna reached appropriate developmental milestones globally and was a healthy child, except for measles aged six with typical recovery. The immediate family immigrated to a regional centre in Australia for work opportunities, but experienced marginalisation with school bullying of Anna and her sisters, social isolation of their mother and unstable employment for their father. Together, they moved to a capital city with more successful assimilation evident in their school inclusion, Christian church participation and permanent employment. Anna aged six developed as an average student. Her menarche was aged nine, with

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expected traumatic incidents in late childhood. At age 11 she witnessed the invasion of the family home, in which the eldest sister had a sexual encounter, and, at age 13 Anna said she had been raped at her youth group. Anna became labile emotionally and relationships with her family members became progressively intolerant and volatile. Her parents’ marriage was strained with her father taking a disciplinary role while her mother was increasingly anxious about caring for her children while absent from the home during weekdays, living near her workplace. Anna completed year eight of secondary school aged 14, but refused to attend further. Currently, she is housebound with her mother as her full-time carer.

Community mental health services referred Anna for diagnostic clarification. When asked why she had come to hospital Anna did not verbalise an answer; she smiled and laughed inappropriately. Her mother commented, “This is not the same Anna”, reporting deterioration since age 12. Anna’s presentation on the ward showed primitive speech (unintelligible muttering to herself and poverty of speech in conversation); a labile affect with outbursts of anger and laughing; catatonic episodes of stupor and trance-like states; difficulty sleeping due to “hearing voices” and “seeing scary things”; disturbed behaviour such as nocturnal enuresis, hyperphagia, and masturbating in public; plus social problems with rising isolation and truancy for two years. Her parents stated no family history of psychiatric or neurological illness.

On mental state examination, Anna was a girl of Filipino ethnicity who appeared physically 10 years old with a short, petite stature and no syndromic features. She hid her face beneath a mop of hair, her eye contact was fleeting and facial expressions were vacant intermittently. Anna was restless but displayed no repetitive movements. Her speech was barely audible; she quietly whispered and sang to herself, occasionally answering questions with single words. When given a pen and paper, Anna neatly wrote her full name and then covered it with scribble. Anna’s affect fluctuated between incongruently elevated and emotionally numbed. Consultation by a child psychologist suggested that intelligence was intact. Throughout her admission, rapport was minimal and Anna did not interact with other children on the ward. Her speech comprised short sentences that lacked prosody but sometimes included sophisticated vocabulary, and her personal skills were observed to fluctuate from incapacitation to being able to feed, shower and dress independently. Although it was considered, the treating team came to the concluding belief from several family meetings that there was no evidence of child sexual abuse.

Physical examination found proportionate short stature with height 139 cm (<1st percentile), weight 36.5 kg (<3rd percentile) and normal body mass index 18.9 kg/m2 (25th percentile) on World Health Organization charts. Family members exhibited normal stature and Anna’s sisters appeared healthy. The full medical assessment by a consultant paediatrician was normal, including neurological examination. Normal blood tests comprised the complete blood count, urea and electrolytes, liver and thyroid function, glucose and prolactin. The only anomaly was mild vitamin D deficiency. Electroencephalography, brain computed tomography, and cerebrospinal fluid analysis were normal. Brain magnetic resonance imaging (MRI) revealed, however, craniofacial disproportion with volume loss in the posterior parietal and occipital regions bilaterally with slender gyri and prominent sulcal spaces, the rest of the brain parenchyma showing normal grey–white differentiation. In psychotic disorders, parietal lobe volume loss has been associated with passivity phenomena, any experience of which Anna did not verbalise. These findings were not felt to completely explain the spectrum of Anna’s disturbance in organic terms, namely schizophrenia with its classical cortical thinning in the prefrontal and temporal areas.

We formulate that Anna’s short stature and developmental regression were psychosocial culminations of insecure mother–child attachment, personal traumatic experiences, immigrant status, high family expressed emotions and social isolation. The disorganised mother–child relationship developed from separation through infancy and early childhood. With subsequent reunion, their relationship evolved into one of emotional enmeshment as they attempted to establish delayed attachment; Anna’s mother expressed her anxiety, as “I’ll only be ok if she is ok”. Neuropsychiatric influences on Anna’s presentation were critically regarded due to her fluctuations in behaviour and affect in the setting of cortical volume loss on brain MRI. Anna’s psychotic features (speech suggesting auditory hallucinations with internal dialogue, inappropriate affect, catatonic episodes and lack of social skills) led to the neurodevelopmental diagnostic hypothesis of childhood disintegrative disorder or childhood-onset schizophrenia. Post-traumatic stress disorder was considered with the constellation of several traumas, isolative avoidance behaviour, visions and trance-like states implying reliving and dissociative experiences respectively, restlessness from hyper-arousal and fluctuating affect.

Anna’s management plan concentrated on family dynamics and a pharmacological trial with an atypical antipsychotic (quetiapine 50 mg bi-daily). The treatment of choice in the literature of removing the child from the family environment was actively discussed by the treating team. In this case, however, it was considered clinically inappropriate due to her parents’ demonstrable efforts to connect with Anna and their dual engagement in inpatient psychoeducation. Vitamin D was supplemented, but growth hormone was not trialled due to minimal responsiveness in psychosocial short stature, plus age seven years post-menarche. The recoverability of organic psychosis and psychosocial elements of Anna’s condition will influence her uncertain prognosis. For Anna, the ongoing focus is on family
therapy and individual psychotherapy to facilitate global functional support, through the Child and Adolescent Mental Health service.

Disclosure

The authors report no conflict of interest. The authors alone are responsible for the content and writing of the paper.

References


