# Astasia–Abasia and Ganser Syndrome in a Preadolescent Girl: A Case Report

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#### SUMMARY

**Introduction** Astasia is the inability to maintain an upright body position without assistance in the absence of motor weakness or sensory loss. Abasia is described as the inability to walk or as uncoordinated walking, while preserving mobility of the lower limbs. Ganser syndrome is described as a dissociative disorder characterized by approximate answers, somatic conversion symptoms, clouding of consciousness, as well as visual and auditory pseudohallucinations. The aim of this study is to present a case that seemed like a combination of neurological and internal disturbances, but actually represented a psychogenic disorder.

**Case Outline** This paper presents the case of a 13-year-old patient with the first manifestation of the inability to walk and stand. Medical history, diagnostic instruments and differential diagnostic methods have been presented in detail. The clinical manifestation was initially interpreted as a neurological disorder. However, after the application of diagnostic procedures and a change in family circumstances, the patient was diagnosed with a psychogenic movement disorder, astasia– abasia, with progressive clinical presentation that included dissociative psychotic reactions (Ganser syndrome). Differential diagnosis as well as the elements of the therapeutic approach have been discussed.

**Conclusion** Presenting a case of psychogenic astasia–abasia in children contributes to a better understanding and differentiating between conditions with a clinical presentation of signs and symptoms dealt with by other branches of medicine.

Keywords: astasia-abasia; Ganser syndrome; preadolescent

#### INTRODUCTION

Astasia is the inability to maintain an upright body position without assistance in the absence of motor weakness or sensory loss [1]. The syndrome is also called Blocq's disease, named after JM Charcot's student Paul Oscar Blocq (1860–1896), who first described astasia as an inability to maintain an upright position despite normal function of the legs when seated [2, 3]. Abasia is described as the inability to walk, or uncoordinated walking, while preserving mobility of the lower limbs. When seen together, the condition is referred to as astasia–abasia [4].

Patients described in the studies of "psychogenic" astasia–abasia, exhibited an unusually dramatic gait disturbance, lurching in various directions, stumbling against surrounding objects (which are usually soft) and falling only when a physician and/or family member is nearby [5].

Since the inability to stand or walk can also be seen in some neurological and metabolic disorders [6] and neurological symptoms in children are clinically not as distinct as in adults, there is a constant need for a differential diagnosis for "psychogenic" astasia–abasia [7].

Psychogenic movement disorders in preadolescence and early adolescence are rarely described in literature. The average age of onset for movement disorders is 12.3, with a female predominance (ratio of 4:1). Astasia–abasia is observed in only 11%. The most common movement disorders are hyperkinetic disorders. The average duration of the disorder varies from two weeks to five years, and the prognosis is generally good. Treatment methods described in literature range from individual psychotherapeutic methods (primarily cognitive behavioral therapy), pharmacotherapy and rehabilitation to multidisciplinary therapeutic approaches, but there is no reliable evidence of effectiveness [8].

Comorbidity between astasia-abasia and Ganser syndrome is poorly described in current literature [9]. There are four essential signs of Ganser syndrome, which are: 1) approximate answers, 2) somatic conversion symptoms, 3) clouding of consciousness, and 4) visual and auditory pseudohallucinations [10]. There has been disagreement about exact etiology of Ganser syndrome (hysterical [11], psychotic after acute cerebral trauma or during acute psychotic illness [12], depression [13] or stress induced disorder which lies between malingering and hysteria [14, 15]). Sigbert Ganser, who described the syndrome, thought that symptoms occur in individuals who are faced with stressful and intolerable situations and who are poorly prepared to cope with stress [9, 14].

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### **CASE REPORT**

The 13-year-old patient "S", a girl from southern Serbia, was admitted to the Department of Child Psychiatry four days after the New Year's Eve. It was her first psychiatric hospitalization. On admission, the dominant symptoms were daily nausea, vomiting after eating, physical weakness and inability to walk without assistance, dizziness, tearfulness.

#### **Current medical history**

The first symptoms, predominantly nausea, appeared a year earlier, a few months after the death of the patient's paternal grandfather (in March 2011) and soon after the death of her maternal grandfather (in November 2011). The patient's relatives insisted that the patient participate in the burial and mourning rituals, against her will, despite the fact that she expressed rejection and fear of such activities. In March 2012, her great-aunt died as well, and since then the girl had reported to have occasional dreams of the great-aunt and mentioned that she has a fear of her appearance in dreams. She wanted the lights to be turned on during the night and her mother to put her to sleep. One month before the admission to the Department, nausea suddenly became worse, with multiple daily vomiting after and between meals, followed by a 5 kg weight loss. In mid-December, she was hospitalized at the Pediatric Internal Medicine Clinic for seven days. Gastroenterology examination gave no explanation for the nausea and daily vomiting, which had been complicated by exhaustion and seeking assistance when walking. She was discharged and referred to the Department of Child Psychiatry. Upon discharge, the patient was not taken to a child's psychiatrist. She was treated by a private practitioner and treated discontinuously (with escitalopram, sulpiride).

#### **Past medical history**

The patient and her parents denied the possibility of psychoactive substance abuse (intentional or accidental), as well as the girl's involvement in a delinquent peer group. They also denied the possibility of deliberately induced vomiting or keeping rigorous diets and fear of gaining weight. She had been an excellent student. She did not have a lot of peer friendships, because she had been hurt by her friends' comments.

#### **Family history**

There were no records of the existence of a psychiatric or neurological heredity. Both the father and the mother were unemployed. Family functioning was burdened by indirect communication, conflict avoidance, enmeshed family relationships, rigid family adaptability and passive-dependent behavior patterns.

#### Initial somatic, neurological and psychiatry status

"S" was brought to the department accompanied by a parent. She was conscious and oriented in all modalities. Skin and visible mucous membranes were pale, decreased turgor pressure. The gait was broad-based, ataxic with "la belle indifférence" facial expression. Romberg was positive. During the interview, she vomited twice, but there were no difficulties in establishing verbal and eye contact. The speech was slow of uneven pace, described as speech mannerism although verbal contact could be easily established, as well as eye contact. Hypovigilance and hypertenacity of attention directed to physical symptoms. Thought processes was slow without abnormality in thought content, but with emphasized alexithymia. Intellectual functioning was estimated as average, but uneven. Mnestic features were preserved. On admission and within the period of seven days prior to admission, the patient exhibited signs of subdepressive mood, apathy, social withdrawal, appetite reduction (primarily due to the fear of nausea and vomiting) and increased suggestibility. There were signs of dissociation from voluntary control of limb movement. Mild separation anxiety was noted upon admittance to the Department. The girl gave the impression of a severely ill patient, dependent on the help of others.

#### Hospitalization

Rehydration therapy and vitamin supplements were included on the first day at the Department. On the second day, there were already fewer episodes of nausea and vomiting. On the third day, after having administered sulpiride (50 mg, twice daily), there were no dyspeptic symptoms. Uncoordinated gait was maintained, while nystagmus, dysarthria and hyperesthesia were variably present, especially during her parents' visits, when the patient demonstrated heightened passivity, dependence, reduced ability to execute movements, as well as difficulties in self-feeding and independent standing and walking.

The patient was unable to perform coordinated movements of the lower limbs when walking forward. The gait was ataxic and she supported herself on the surrounding objects and walls without falling, but occasionally hit her knees against nearby objects, which caused hematomas in the knees. Her gait backward was not ataxic. She could get up from a chair on her own and without arm support, climb up and down the stairs and run. The "chair test" was also used [16]. While sitting in a rotary chair, the patient was able to move the chair in all directions, as well as lift her legs to a horizontal position.

Additional differential diagnostic procedures were aimed at excluding a physical cause and organic disease. The results of the blood and urine analyses indicated clinically insignificant dehydration and starvation. Toxicological analysis and screening for drugs were negative. Repeated test results were within normal values, just like the results of gastroenterology examinations. Ophthalmologic and neurology findings were normal. Preserved gross muscle strength of the lower limbs was normal (grade 4+). An ultrasound of the abdomen, magnetic resonance of the endocranium, and EEG and EMG recordings for measuring tremor in the limbs showed no abnormalities. Wechsler Intelligence Scale for Children (WISC) showed the following: verbal IQ 99, manipulative IQ 57, total IQ 77. There was no significant cognitive dysfunction, but there was a drop in intellectual efficiency due to attention deficit, which caused poor organization of thought and general psychomotor slowness. It was concluded that there was a dissociative reaction with secondary instrumentalization which compromised the patient's recovery.

During the hospitalization, we observed the girl's behavior while she was separated from her parents and upon reunion with her mother. While they were separated, she stopped complaining of nausea and vomiting. There were only verbal expressions of emotional and physical experiences ("I am sick from missing my mom"). Separation anxiety was significantly intensified by the mother's instrumental behavior (suggesting over the phone that they should be together). Five days after admittance, she phoned her mother, started whining, crying, and expressing dysphoria. The gait was still characteristic, unusual and bizarre. She walked to the bathroom without her slippers on, lay in bed with the slippers on, didn't know where her bed was and didn't know how to cover herself with a blanket. She was in the supine position most of the time, neglecting personal hygiene and appearance. Gestures become poor as well as her general motor movement. Facial expressions were exaggerated with evident affective blunting and there was an "empty" look in her eyes. She was unsure about orientation to time, her attention easily distracted. Verbal contact was difficult to establish and communication was poor. Speech was dysarthric and the answers were only approximate or contrary to the expected. Intellectual functioning was extremely uneven, with difficulty in abstract thinking. The patient did not understand proverbs or interpreted them in a bizarre way, did not understand common metaphors or comparisons and was either focused on the functional aspects or not focused at all. Fluency and spontaneity of speech were reduced to a few words, no conversational quality ("yes-no" answers, "I know - I do not know", or the opposite to the expected). Her thought process was poor, seemed devastated. No interest in what was happening at the ward. No manifested delusions, hallucinatory behavior, conceptual disintegration, grandiosity, suspiciousness and hostility. PANSS score was 82. Risperidone was included (1 mg once daily) and sulpiride discontinued. The mother was invited so that reunion behavior could be observed. On the same day, the patient's behavior and speech became significantly more coherent, but the need for assistance while performing other activities persisted. The mother helped her walk, eat, change clothes, which the patient eagerly accepted. Over the following seven days, the intensive individual cognitive behavioral therapy was continued, and so was family counseling and psychotherapy. As a result, the regressive course was stopped. Having gradually recovered physical and mental functions, the patient was released 21

days after her admission. The patient was discharged with the following diagnoses: psychosomatic disorder (psychological factors affecting other medical conditions) – F54; conversion disorder – F44 (F44.4, F44.8); family dysfunction Z63.8.

#### Follow-up

Follow-up examination showed evidence of recovery, as well as compliance with the pharmacotherapy. Renewed social peer contacts and gradual adaptation to school environment were reported. The patient had a partial amnesia of the time spent in the hospital while being separated from her mother. Over the next three months there was a complete recovery and the pharmacotherapy was discontinued.

#### DISCUSSION

There is currently no "gold standard" for diagnosing astasia-abasia [17] and as far as our knowledge goes, Ganser syndrome in children and adolescents is rare. In total of 94 reported cases in literature, only 14 individuals were aged 16 or younger [18].

Our differential diagnostic procedure was carried out in two directions: exclusion of organic disorders and meeting the criteria for a psychological disorder.

While trying to find the causes of astasia-abasia, we first excluded the "organic" disorders. Astasia-abasia as a result of exhaustion from vomiting and electrolyte imbalance, as well as other gastrointestinal disorders, was excluded after abdominal examination. Neurological examination and laboratory analyses helped us exclude inflammatory brain disease (cerebellitis). Imaging techniques (magnetic resonance of endocranium), ophthalmic examinations and determining the levels of copper and ceruloplasmin were used to exclude multiple sclerosis, brain tumors, brain stroke, Wilson's disease, and chorea minor. Creutzfeldt-Jakob disease was excluded because there were no specific electroencephalography findings. The possibility of a neuroleptic syndrome was excluded based on the patient's medical history and neurological examination. "Hydrocephalus astasia-abasia" in normotensive hydrocephalus was excluded due to the results of the neurological examination and the absence of the characteristic clinical features (patient had normal movements when lying in bed). Early-onset chronic organic psychosyndrome with global mental dysfunctioning was excluded because there was reverse dissociation between explicit and implicit memory compared to the neuropsychological characteristics of amentia syndrome. Parkinsonism was excluded as a result of neurological examination and tremor evaluation.

Second directions were meeting the criteria for a psychological disorder of astasia-abasia. Blocq pointed out that in functional disorders, if there is paralysis of the walk, it does not correspond to the patient's gross muscle strength in the lower extremities seen upon neurological examination, sitting and lying. Other actions that do not involve the usual walking are performed with high accuracy (walking up the stairs, walking "on all fours", even jumping). The patient appears to have "lost memory" of the movements necessary to maintain upright position (astasia) and walk (abasia) which is why astasia-abasia is called "ataxia with a defect in automatic coordination" [19]. The difference between that and complete ataxia is in conversion of voluntary coordination. The patient also satisfied the criteria for conversion disorder because the onset was associated with insoluble and intolerable life events and relationships and the parents' reactions in critical situations. The astasia-abasia varied depending on the context or suggestions. There was a significant degree of voluntary control over the symptoms including calm acceptance ("la belle indifférence") with the absence of fear of illness and physical disorders. The symptoms could be explained as a "neurological" instrument for passive avoidance of aversive consequences. Patient showed tendency towards denial of obvious problems such as family dysfunction, but symptoms concomitantly regulated it.

During the hospitalization she exhibited all four essential clinical features of the Ganser syndrome, with acute onset and sudden change in behavior after separation from the mother (within less than two weeks). The course was short-lived with tendency of recurrence. The patient did not exhibit psychotic symptoms along Ganser syndrome. Schizophrenia and affective disorders were excluded based on the absence of typical productive psychopathological phenomenology and exclusion criteria as well as posttrau-

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matic stress disorder. Underlying organic illnesses were excluded previously.

On admission, the patient satisfied the criteria for the existence of a "psychosomatic disorder" (psychological factors affecting other medical conditions), due to the psychological component in the disturbance of normal functioning, with an impairment of at least one organ system (gastrointestinal) and vegetative nervous system dysfunction. Very few physical complaints (denegation or minimization), alexithymia, as well as the typical "psychosomatic" family pattern (enmeshed family relationships, rigid family adaptability) [20], further supported the symptomatic behavior. Family dysfunction was the key factor for triggering and maintaining the psychopathological state, but family was also a mediator in the process of recovery.

We conclude and confirm that the patient in our case report of astasia–abasia satisfied the criteria for conversion disorder. The existence of a somatic disorder was excluded and the symptoms were resolved upon pharmacotherapy and psychotherapy, which confirmed the diagnosis of the psychogenic disorder. Appearance of Ganser syndrome also supported that opinion. The nature of the Ganser syndrome remains unclear, but this case study highlights astasia–abasia and Ganser syndrome as symptoms of reduced individual capability to cope with stress and also as family system regulator. Presenting a case of psychogenic astasia–abasia in a child contributes to a better understanding and differentiating between conditions with a clinical presentation of signs and symptoms dealt with by other branches of medicine.

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## Астазија–абазија и Гансеров синдром код девојчице у преадолесценцији – приказ болесника

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#### КРАТАК САДРЖАЈ

Увод Астазија (гр. astasia) је неспособност самосталног одржавања усправног положаја тела иако не постоји слабост мишића нити губитак сензибилитета. Абазија (гр. abasia) се описује као немогућност ходања или некоординисано ходање уз сачувану покретљивост доњих удова. Гансеров синдром је дисоцијативни поремећај који се одликује приближним одговорима, соматским конверзивним симптомима, помућењем свести и визуелним и слушним псеудохалуцинацијама. Циљ рада је био да се прикаже случај који указује на комбинацију неуролошко-интернистичких сметњи, а представља психогено измењено телесно стање.

**Приказ болесника** Приказана је тринаестогодишња девојчица код које је први пут уочен поремећај самосталног ходања. Дати су детаљан приказ историје болести и инструменти дијагностичке и диференцијалнодијагностичке процене. Клиничка слика је у почетку схваћена као неуролошки поремећај, а након примене дијагностичких поступака и промене породичних околности, дијагностикован је психогени поремећај покрета типа астазија–абазија са прогресијом клиничке слике до дисоцијативне психотичне реакције (Гансеров синдром). Дискутовано је диференцијалнодијагностичко сагледавање, као и елементи терапијског приступа и исхода.

Закључак Приказ психогене астазије-абазије код деце представља допринос бољем разумевању и разграничењу стања која у својој клиничкој слици имају симптоме и феномене којима се баве друге медицинске гране.

**Кључне речи:** астазија–абазија; Гансеров синдром; преадолесцент

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