

Selective Mutism: A Review of the Concept and Treatment

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Selective mutism is the persistent failure of a child to speak in a specific social situation where speaking is expected, despite normal or near-normal speech in other situations [1,2]. Selective mutism is not a rare disorder, with an estimated prevalence of between 2/10,000 and 70/10,000 school-age children [3]. Until recently, selective mutism was considered difficult to treat and even "intractable" [4], requiring a variety of treatments such as behavioral techniques, psychodynamic approaches and combinations of family, art and speech therapies [4–8]. However, the conceptual basis of selective mutism has changed, the consensus being that social phobia and anxiety components are the major, or at least important, etiologic factors [2,9]. This concept bears important pharmacotherapy implications [2,9,10–12]. Since only one article has been published in the general English-language literature on the etiologic and therapeutic issues of selective mutism [13], we would like to draw particular attention to the pharmacotherapy – of major importance in the management of selective mutism.

Diagnostic features and assessment

Selective mutism is a childhood disorder of at least 1 month duration where the failure to speak is both persistent and specific to certain social situations (e.g., school), despite adequate speech in other situations [1]. The disturbance interferes with educational achievements and social communication. Furthermore, the failure to speak is not due to the child's lack of knowledge of, or comfort with the spoken language required for the social situation. Other disorders that must be excluded include communication disorders (e.g., phonologic disorder, expressive language disorder, etc.), lack of knowledge of the language, pervasive developmental disorder, schizophrenia, other psychotic disorders, and severe mental retardation [1].

Until 1994, elective mutism was the term used to describe this entity. The current term, selective mutism, is consistent with the consensus that anxiety issues [1,4] are the underlying reason for the child to "select" not to speak.

Assessment of a child with selective mutism is challenging since avoidance anxiety and lack of speech are part of the characteristics of the disorder [2]. The most thorough assessment method was suggested by Dow et al. [4] and requires assessments of symptoms, including medical and psychiatric examination, speech, language, communication skills and audiology, as well as academic and cognitive functions.

Epidemiology

About 1% of children seen in mental health settings have this disorder [1], and the numbers cited in the literature for school-age children range from 0.02% to 2% [3,14]. A systematic, community-based study [15] identified 18 children with selective mutism among a population of 10,000 children aged 7–15 (0.18%). The authors concluded that "the disorder is not extremely rare," and, therefore, more common than well-known disorders such as William's syndrome [15]. Selective mutism is more prevalent in girls and is observed in all social strata [16,17].

Several studies confirmed the finding of developmental language delay or specific language disorders in children with selective mutism [16,18,19]. In an analysis of 100 cases, 38% had pre-morbid speech and language disorders such as stuttering, receptive or expressive language disorders [16]. Co-morbid psychiatric manifestations that are diagnosed in children with selective mutism include separation anxiety, school phobia, elimination problems, obsessive-compulsive features, and even mental retardation [20,21]. Somatic symptoms such as sleeping and eating problems are common, while disruptive disorders marked by hyperactivity or aggression are relatively rare [3,9,16,21]. Selective mutism has also been described in a patient with Tourette's syndrome [22]. Thus, a comprehensive assessment of cognitive and emotional parameters is essential in the evaluation of any child with selective mutism [20,23].

Etiology

Of the many etiologies suggested, one is pregnancy or birth-related complications, which is more prevalent in children with selective mutism than expected for the normal population [3]. Another etiologic consideration is oriented towards psychodynamic problems, related to family neurosis, oppositional behavior, post-traumatic reaction, dissociative identity disorder, and unresolved psychodynamic conflicts [24,25]. Since selective mutism has been described in twins [26], in a child with deletion of the short arm of chromosome 18 [27] and in association with the fragile X syndrome [28], the genetic component has also received considerable attention.

However, the innate personality characteristics of patients with selective mutism, i.e., shyness, inhibition, social phobia and anxiety, have merited comment in many reports of children with this disorder [9,29–31]. Dummit and colleagues [17] noted that children with selective mutism demonstrated features associated

with social phobia or alternatively, avoidant disorder traits, termed "behavioral inhibition" by the Harvard infant program [32]. Anstendig [9] concluded that "the etiology and symptom overlap demonstrates selective mutism as being an anxiety disorder...", indicating specific pharmacologic and psychological therapies.

Treatment

The treatment of selective mutism has traditionally been behavioral and employs methods such as family therapy, speech therapy, a school-based multidisciplinary plan, and combinations of these methods [5–8]. Since anxiety disorder and social phobia were found to be etiologically relevant to selective mutism, pharmacologic treatment specific for anxiety and phobia became therapeutic options. Several investigators [5,11,12,29,33,34] suggested treatment with monoamine oxidase inhibitors and selective serotonin re-uptake inhibitors.

Phenelzine, a non-selective monoamine oxidase inhibitor, was the first drug to be used successfully in the treatment of selective mutism [12]. Phenelzine was chosen because of its therapeutic efficacy in social phobia and the fact that it is, for the most part, well tolerated by children. However, necessary dietary restrictions may render its use cumbersome. The anti-anxiety effect of phenelzine is attributed to its dopaminergic and serotonergic action as well as the ability to increase brain levels of gamma-aminobutyric-acid (GABA) [12,31].

Fluoxetine was successfully used in 21 children with selective mutism co-morbid for overanxious disorder or social phobia [35]. It was well tolerated with minimal side effects. Black and Uhde [10] reported an additional 16 subjects with selective mutism treated with placebo for 2 weeks. The 15 placebo non-responders were randomly assigned for 12 weeks of treatment with either fluoxetine or placebo. The condition in subjects treated with fluoxetine was significantly improved on parents' ratings compared to the placebo group. Similar results were reported for other patients and other SSRI drugs such as sertraline [33,34,36].

We used fluoxetine in four girls, age 5–17 years, with selective mutism. All four showed significant improvement in speech communication after 8–10 weeks. Two of them had been treated by psychiatrists or psychologists for 1–2 years with no substantive change in their clinical condition. Pharmacologic treatment had not been suggested as an option.

We now describe one of these children in more detail. This otherwise healthy girl was referred to us at the age of 11 because of progressive learning and social problems. Her language development was initially normal although in first grade she received speech therapy to improve articulation and private tutoring for reading. At the beginning of the fourth grade she gradually refused to speak at school, both to adults and children, and withdrew from most social interactions including passive participation in the school room. Her school work rapidly declined, accompanied by social rejection by her classmates, and she was transferred to a special education class within the school. After 2 years of intensive

psychological treatment and art therapy that proved ineffective in significantly altering her status, fluoxetine was begun at a dose of 0.3 mg/kg and gradually increased to a maximum of 20 mg/day (0.6 mg/kg/day). Within 6 weeks, her condition improved and she began speaking in a more appropriate manner. At that time she was able to talk on the phone with other children, and within 3 months had improved to such an extent she was able to speak in front of the entire class. Her school achievements and social interactions normalized and she was eventually transferred back to a regular class. After 18 months of fluoxetine, the medicine was discontinued at the request of the parents due to recurrence of the mutism, which subsided after re-institution of fluoxetine.

Conclusion

Selective mutism can be a disabling condition for the affected child, parent and school staff. This disorder requires a comprehensive bio-psycho-social assessment as well as a bio-psycho-social approach to treatment. Since social phobia and anxiety components are major etiologic considerations, and in view of some promising symptomatic improvement with SSRI medications, this form of treatment should be routinely considered together with the other known modalities.

Based on the literature and our own experience, every child for whom the diagnosis of selective mutism is considered should undergo a complete neurologic, psychiatric, speech and language, audiology, social and academic evaluation. Treatment options should also include a variety of pharmacologic options, mainly SSRI medications.

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SSRI = selective serotonin re-uptake inhibitor

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Vote for the man who promises least; he'll be the least disappointing

Anonymous

When truth is buried, it grows, it chokes, it gathers such explosive force that on the day it breaks out, it blows everything up with it.

Emile Zola (1840-1902), French novelist and social reformer, most famous for his support of Dreyfus, expressed in "J'accuse."

Capsule



Possible genetic basis to retinopathy in type 2 diabetes

Diabetic retinopathy in some patients with type 2 diabetes mellitus and good glycemic control suggests a possible genetic factor. Rema et al. estimated the prevalence of retinopathy in 322 families with type 2 diabetes having at least two siblings. In each family, the sibling with the longest standing diabetes was identified as the proband.

The prevalence of retinopathy was more than threefold higher among siblings of patients with diabetic retinopathy, and diabetic retinopathy was present in 136 probands and in 53 of their

siblings (35.3%). In contrast, the prevalence of retinopathy among siblings of probands without retinopathy was only 11.2%. Siblings of patients with diabetic retinopathy had a 3.37 higher odds ratio of developing retinopathy than did siblings of diabetics without retinopathy. The higher risk of retinopathy was not influenced by duration of diabetes, glycemic control, or the presence of hypertension. The authors conclude that the familial clustering of retinopathy could have a genetic basis.

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