research paper

A case of remission of childhood onset fluency disorder induced by SSRIs (selective serotonin reuptake inhibitors) in response to benzodiazepines

Toshiro Takami

Abstract: We experienced a case in which selective serotonin reuptake inhibitors (SSRIs) remitted a childhood onset fluency disorder. The patient had a mild dysarthria since childhood. Just before the end of her high school years, she developed interpersonal tension. She was suffering from interpersonal tension and had been prescribed relatively high doses of benzodiazepines since she came to our hospital. And incidentally, the benzodiazepines had responded to the benzodiazepines for childhood-onset fluency disorder. Because the patient had partial epilepsy, it was thought that she might respond to benzodiazepines.

Although her childhood-onset fluency disorder was eventually remitted by selective serotonin reuptake inhibitors (SSRIs), there are many cases of childhood-onset fluency disorder remitted by SSRIs in this way.

Keywords: childhood-onset fluency disorder; selective serotonin reuptake inhibitors; benzodiazepines; Paxil; partial epilepsy

Introduction.

Childhood-onset fluency disorders have long been studied as a serious disorder in the West. Because of its high familial accumulation, it has been regarded as an inherited disorder. Childhood-onset fluency disorders occur between the ages of 3 and 6 years, occurring in 5% of children and 1% of adults. The fact that many cases of childhood-onset dysfluency are in remission by adulthood suggests that it may be a form of partial epilepsy. The prevalence of epilepsy in males is four times greater than in females during and after childhood. Compared to the normal average, children with childhood-onset fluency disorders are three times more likely to have childhood-onset fluency disorders if their parents have them.

Cerebral infarction (Sahin, Krespi & Yilma, 2005) or very severe head bruising can cause adult-onset dysfluency.

Currently, the search for loci for childhood-onset fluency disorders is proceeding rapidly, based on studies of multiple cases in families. Many loci for childhood-onset fluency disorders have been discovered, and childhood-onset fluency disorders are considered to be one syndrome that is caused by a number of factors (Raza, Gertz & Mundorff, 2013).

The case was successful with acupuncture and stellate ganglion block therapy for childhood-onset fluency disorder. This suggests that the childhood-onset fluency disorder in the

case was sympathetic overstrain. There is a significant probability that this type of case will be included.

Case.

(Case) 42 years old, male, right-handed (father, mother and sister are also right-handed)
No childhood-onset fluency disorder or epilepsy in his parents, siblings or other close relatives.

His grandfather, who was very strict, took him to the psychiatric department of a university hospital and diagnosed him with a personality disorder (no medication) because he did not go to school and played around.

However, my father was also extremely stubborn, and there is a strong possibility that he had an autism spectrum disorder. The father was strict with the mother, but was very kind to the child, and the case does not recall ever being scolded by the father.

The father also had a pathological hotness, as in the case.

(Character) Strongly stiff, nervous, gentle, honest, good-natured, hard-working. (History) None to be noted

(Current medical and life history) Father was 25 years old at the age of 25 and mother was 24 years old at the age of 24, with no perinatal issues of note. No parents or relatives have ever told him about febrile convulsions or other epilepsy in infancy or early childhood.

It is possible that the stress caused by this family discord may have hung over the case, as the father was too absorbed in gambling and other activities to work, while the mother worked alone to make the family budget.

Until kindergarten, she was an extremely crier, and every day, especially in the evening, she would cry for several hours for no reason. Also, when she was in kindergarten, she was dropped off from the kindergarten bus and could not walk the very short distance from the bus stop to the house, so she was always crying at the bus stop. He had only attended kindergarten for one year, but he had only been there about a dozen times because his case was very reluctant to come to school and because of a very prolonged case of rubella and other symptoms. When he was in kindergarten, he was spoken to by a girl but could not speak, and was apparently ridiculed for his fluency disorder.

He grew up quickly, and when he entered elementary school, he was the second tallest child in his class.

In elementary school, he stopped crying at all and began running from home to school (about 15 minutes in first grade). Interpersonal relationships were not my strong suit. I didn't have any close friends until the first grade. In the second grade, through an intermediary between my parents, I made a classmate friend, and we began to hang out together almost every day after school. The friend was extremely calm.

Since elementary school, he had some dysarthria, such as not being able to distinguish between "ki" and "chi". He says that this continues to this day, but for example, he did not know how to pronounce the word "earth". She is still slurring her pronunciation and is very bad at telephone calls.

When he was in the lower grades of elementary school, he had a habit of saying "anone" first in class, which was often pointed out by the teacher. It was easier to speak up if you put "you know what" at the beginning.

Maybe it was because he was in the country, or maybe it was because he was extremely good at math (mathematics), but he was never tormented.

In the early grades of elementary school, he often had episodes of "feeling lightheaded and unrealistic in the evening" and "stopping chopsticks at dinner for a few minutes to ten minutes". These attacks ceased to occur after the fourth or fifth grade.

When exercising, he was unable to relax his strength, which made exercise difficult for him. In the third grade, he was ridiculed for his awkward running style, and was nicknamed "Dottingbuttan" because of the way he ran.

He didn't train at all, but in the third grade, he learned that he could get a bicep muscle bump. It could be assumed that the force bumps were noticeable because he had very little body fat.

He had been told since elementary school that his reaction to surprise was very stronger than normal. His head circumference was large. He was also fearful and was very afraid to go to the bathroom at night in elementary school. My fear of blasphemy was stronger than in elementary school.

In the fourth grade, he realized that his abdominal muscles were so tight that he could withstand being hit in the abdomen hard. He also realized that he could hardly do abdominal exercises, which anyone could do.

In the fourth and fifth grades, he talked about how funny it was to contort his face so badly when he was running.

His development was rapid, and his height growth almost ended in early seventh grade, and there was a time in his first year of middle school when his 50-meter run was the fastest in his grade. He was a fast short-distance runner, but not a good long-distance runner.

A few times in junior high school, his close friends ridiculed him for his serial fluency disorder, but he was a close friend and didn't care much about it. In the second year of junior high school, he had occasional episodes of sleep paralysis.

In his first year of high school, he noticed that the first word was not coming out for the first time. It is presumed that the serialization changed to refractory. From this point onwards, he would suffer greatly from fluency disorders: in September, he would frequently leave early before his Japanese language class because he could not get the first word out of his Japanese language class, and he also refused to go to school on numerous occasions.

In high school, he learned that his fluency disorder became more severe when he was stressed.

He was extremely bright in science and math, but performed very poorly in English. He did not study science and math at all, only English, but English was always the worst scoring subject. My grades were also poor in Japanese. Case thought that he had an innate weakness in language courses. His English pronunciation had been a topic of conversation since middle school, as he found it hilarious. In high school, he was often told that he couldn't understand sarcasm and sarcasm.

I heard that I had this problem in the first half of my college and junior high school days, but there were many times when I realized or was pointed out that my mouth was

distorted for some reason. In addition, I was aware of the fact that my face was strained, but I was often pointed out that I had a funny expression when I was a high school student.

Towards the end of his senior year in high school, he developed interpersonal tension. When he took the second examination, he was so nervous that he failed the University of T, which was his school of choice. I quit the prep school and came back to my hometown after 2 months due to the interpersonal tension. And I did a home-nominated test. After failing one year, he dropped his application and entered the former Imperial University in his hometown.

When he was in college, he was asked to read English in English class, which was embarrassing for him because he could only read English tritely due to his fluency disorder.

In addition, when I was in college, it was very difficult for me to attend a crowded class due to interpersonal tension, and I had to stay in school for many years.

Due to interpersonal tension, he had few friends in college.

In the latter half of his college days, he learned from the Internet that acupuncture and stellate ganglion blocks were effective for fluency disorders, and he received them. Especially the stellate ganglion block had a very strong effect on both fluency disorders and interpersonal tension.

In his fourth year at university, when he was convinced that he would be expelled if he stayed in school any longer, he suffered a skull fracture in a head-on collision with an oncoming car and was hospitalized for two months (with post-traumatic amnesia of four and a half days). He was hospitalized for two months (post traumatic amnesia was four and a half days).

After the accident, he suffered from interpersonal tension and came to our hospital. The author became the attending physician and started the administration of benzodiazepines: cloxazolam, flurazepam, lorazepam, clorazepate, flunitrazepam, flutoprazepam, ethyl lofrazepate, clonazepam, The effects of alprazolam, etc. on fluency disorders were good except for ethyl lofrazepate, clonazepam, and alprazolam. alprazolam was presumed to have an anticholinergic effect, but the fluency disorder worsened severely when the patient took it. The lofrazepate and clonazepam were weak in action or had little effect.

Amitriptyline was prescribed, but after taking one tablet, the patient slept for 24 hours, and for three days, the fluency disorder became severe and severe, and the dose was taken only once.

After various explorations, he settled on bromazepam 20 mg/day, diazepam 15 mg/day, etizolam 3 mg/day, and flunitrazepam 4 mg/day (a sleeper but taken during the day to relieve interpersonal tension).

Interpersonal tension became a problem because benzodiazepines were dramatically effective for fluency disorders, but were effective but not sufficient for interpersonal tension.

After graduating from college, he got a job as a programmer, which he had studied on his own during college. She thought she could do the programmer's work at home by herself, but the fact that she had to do it in the office was discouraging to her. As a programmer there was little to talk about, but there was interpersonal tension. He was forced to take a large dose of benzodiazepines to relieve his interpersonal tension. On his days off, he stopped taking benzodiazepines to avoid wasting them. In addition, therefore, he had to go back and forth between the office and his apartment. Another reason for his autistic lifestyle was that on his

days off, the case was absorbed in creating software that he was building himself, regardless of his work at the company.

The case used benzodiazepines by dissolving them in the oropharynx just before talking on the phone or other occasions. The patient claimed that if absorbed through the oropharyngeal mucosa, the drug would go directly to the brain without passing through the liver, which was faster and more effective.

When the sympathetic nervous system was stimulated, the patient became very hot for about 40 minutes, i.e., when he arrived at work, when he ate lunch, when he took a bath, etc. He suspects pheochromocytoma and goes to the hospital for a blood test, but is told that "dopamine and other substances are not elevated in the slightest, the sympathetic nervous system is thought to be very sensitive, not pheochromocytoma. I don't remember this pathological hotness so much when I was in elementary school, but it became more pronounced from junior high school or high school.

When I was 30 years old, I took about six bags of cold medicine (probably PL granules) in the morning before going to work, instead of one bag of cold medicine (probably PL granules), thinking that today was very important and I would never miss a day of work. While having lunch at work that day, he causes an episode in which he held a plate of food in front of his head and left it there for about 10 minutes. The case was hard to believe when he was told about it later, and he asked several people about it, but they agreed that it had indeed happened. The president of the company made him undergo a medical examination, which was probably an after-effect of the car accident he had had in college, but there was nothing of note in the CT scan of his head, EEG, or intelligence, and he had only a strong impairment in the ability to write. The patient's ability as a programmer was extremely high, especially the president of the company, who valued him very much.

At the age of 32 years old, the patient was suffering from influenza and took 10 tablets of loxoprofen sodium, but the fever did not go down at all. However, after two hours, his fever rose again and he became painful. He took four tablets of diclofenac sodium again, his fever was relieved and he felt better, but not enough, and he took one more tablet, and his fever fell sufficiently and he felt better. Thus, the case was proud of being very strong with drugs. (That year's flu was a very symptomatic flu.)

I prescribed Risperidone, olanzapine because it was claimed online to help with interpersonal tension, but it had no effect on interpersonal tension or fluency disorders with only side effects. On a day at work, he once took risperidone 2 mg at noon, felt severe general malaise, laid down on the couch for two hours, and went home.

He had taken risperidone 2mg at noon on a day at work. Case was an avid fan of Formula One, so much so that he had been playing F1 race videos as background music at work. A period of more than a decade had gone by when he watched nothing but F1 on TV. This is why he very much preferred manual transmissions, and it was a manual transmission car, and he may have unconsciously put it into neutral as soon as the seizure occurred. However, there was an omen, and the car was parked at the end of the roadway, most likely due to amnesia caused by the seizure and no memory of the omen.

The car was apparently not damaged or scratched. There were no traffic citations at all. It was later thought that a police officer apparently came to the scene where the child was crying in a baby chair.

The police officer apparently transported the child to the neurosurgical hospital. The case has no memory of what happened before the child was transported to the neurosurgical hospital. When he arrived at the hospital, he regained consciousness, but he was in a dazed state. He has partial memory of the events in the hospital.

The lack of memory of the aura is identical to the episode during lunch.

The case was so eager to heal his interpersonal tension that he researched in detail on the Internet and learned that SSRIs are effective for interpersonal tension and strongly desired to be prescribed SSRIs. However, the patient learns that her fluency disorder is in remission during this period. The case says, "I don't know for sure which drug was responsible for the response, but I think it was probably Paxil.

The patient had a strong interpersonal tension problem and was hardly troubled by the response to benzodiazepines, so she was hardly pleased with the remission of her fluency disorder.

She had "an inability to read the atmosphere and understand people's minds, pathological stiffness, pathological clumsiness in manual and physical movements, pathological clumsiness in interpersonal relations, and an inability to look people in the eye", which was a typical Autism Spectrum Disorder (yes, definitely Autism Spectrum Disorder).

Consider

Because of her Autism Spectrum Disorder, she had a low tolerance for stress and an anxiety disorder since childhood, which may have resulted in dysarthria, inability to perform abdominal exercises, and awkwardness in running. His family was extremely poor in his childhood and the family discord was so severe that his mother and father quarreled with each other almost every day.

Even now, the case is unable to distinguish between "chi" and "chi" and does not know the word "earth" when typing on the keyboard.

Since the patient has some forms of epilepsy, particularly partial epilepsy, it is possible that benzodiazepines may be helpful in controlling the fluency disorder. However, there is also a possible mechanism by which benzodiazepines help to relieve the strong muscle tone caused by the anxiety disorder.

An article suggesting an association between epilepsy and impaired fluency has been published (Sechi, Cocco & D'Onofrio, 2006) that describes the efficacy of the antiepileptic drug levetiracetam in the treatment of comorbid conditions with partial-onset epilepsy. It is possible that this may be the case.

Neurosurgery has been performed in the United States for childhood onset fluency disorders (Edgar, Alexander & Leveque, 2015). This is because organic functional deficits in the left basal ganglia are thought to be at the root of childhood-onset fluency disorders.

Benzodiazepines are generally close to quasi-narcotics in the West due to their dependence, and possession of them without a prescription can result in arrest. Although childhood-onset fluency disorders have long been the subject of extensive research in the West, this may be the reason why there have been no papers published on the efficacy of benzodiazepines for childhood-onset fluency disorders.

In Europe and the United States, alprazolam and clonazepam are relatively frequently prescribed among benzodiazepines. However, alprazolam has anticholinergic effects and causes temporary but severe fluency problems (Elliott & Thomas, 1985). Cases have also taken clonazepam, but have failed to feel any effect on fluency disorders as well as interpersonal tension. Clonazepam has not been reported to decrease the severity of fluency disorders.

Clobazam, another recently launched antiepileptic drug, is a relatively newer benzodiazepine that has also not been reported for the treatment of childhood-onset fluency disorders.

Whether benzodiazepines have a dramatic effect on childhood-onset fluency disorder in a small number or a large number of cases, as in this case, benzodiazepines are almost quasinarcotic in Europe and the U.S. In Europe and the U.S., benzodiazepines other than alprazolam and clonazepam, which are considered to be less dependent, are rarely prescribed. Also, benzodiazepines may be considered to worsen impaired fluency, especially because alprazolam, due to its anticholinergic effects, can severely, if only temporarily, worsen it.

The SSRIs paroxetine (Boldrini, Rossi & Placidi, 2003; Busan, Battaglini & Borelli, 2009; Costa & Kroll, 2000; Murray & Newman, 1997; Schreiber, Shaul & Pick, 1997), fluoxetine (Kumar & Balan, 2007), and sertraline (Brewerton, Markowitz & Keller, 1996; Costa & Kroll, 1995) have been reported to lessen the severity of childhood-onset fluency disorders. However, there have been reports of sertraline induced fluency disorders, even if only temporarily (Christensen, Byerly & McElroy, 1996; McCall, 1994). This large number of reports proves that childhood-onset dysfluency is considered to be an important disease in the West.

Conclusion

"As a potential treatment for childhood-onset fluency disorders, SSRIs

SSRIs (selective serotonin reuptake inhibitors) permanently degenerate the neural junctions, and the effects of SSRIs on childhood-onset fluency disorders have been published in a relatively large number of papers overseas. There are five papers on the effects of paroxetine on childhood-onset fluency disorders, and none of them showed severe effects.

There have been attempts to heal childhood-onset fluency disorders with drugs in the West for many years. Especially in Europe, there have been many papers on the use of SSRIs, which have been used for a long time for the treatment of childhood-onset fluency disorders, and there have been eight papers on the use of SSRIs that have been found to cure childhood-onset fluency disorders.

Attempts to cure childhood-onset fluency disorder with antipsychotic drugs used for schizophrenia in the United States (which can only have a temporary effect) are now prominent in the United States, and the treatment of childhood-onset fluency disorder with SSRIs in Europe has been less prominent online.

In summary, as

- 1) SSRIs can lead to remission of childhood-onset fluency disorders, especially paroxetine (Paxil), which is most effective.
- 2) SSRIs taken over several years can lead to almost complete remission of childhood onset fluency disorders.

3) Paroxetine (Paxil), among SSRIs, often starts to work within a few weeks.

Conflict of interest self-assessment: nothing to declare.

References

American Psychiatric Association (2013) Diagnostic and Statistical Manual of Mental Disorders, 5th edition (DSM-5), APP, Arlington VA. (2014) DSM-5 Diagnostic and Statistical Manual of Mental Disorders, edited by S. Takahashi and S. Ohno (2014), Igaku-shoin

Boldrini, M., Rossi, M., & Placidi, G.F. (2003) Paroxetine efficacy in stuttering treatment. Int J Neuropsychopharmacol 6(3): 311-312.

Brewerton, T.D., Markowitz, J.S., & Keller, S.G. (1996) Stuttering with sertraline. J Clin Psychiatry 57(2): 90-91.

Busan, P., Battaglini, P.P., & Borelli, M. (2009) Investigating the efficacy of paroxetine in developmental stuttering. Clin Neuropharmacol 32(4): 183-188.

Christensen, R.C., Byerly, M.J., & McElroy, R.A. (1996) A case of sertraline-induced stuttering. J Clin Psychopharmacol 16(1): 92-93.

Costa.D., & Kroll.R. (2000) Stuttering: an update for physicians. CMAJ June 27, 162 (13) 1849-1855.

Costa, D., & Kroll, R. (1995) Sertraline in stuttering. J Clin Psychopharmacol 15: 443-444. Edgar, D., Alexander, G.W., & Leveque, M. (2015) Psychosurgery for stuttering, Neuropsychiatr Dis Treat 11: 963-965.

Elliott,R.L., & Thomas,B.J. (1985) A case report of alprazolam-induced stuttering. Clin Psychopharmacol 5: 159-160.

Kumar, A., & Balan, S. (2007) Fluoxetine for persistent developmental stuttering. Clin Neuropharmacol 30: 58-59.

McCall, W.V. (1994) Sertraline-induced stuttering. J Clin Psychiatry 55(7):316.

Murray, M.G., & Newman, R.M. (1997) Paroxetine for the treatment of obssive-compulsive disorder and comorbid stuttering. Am J Psychiatry 7:1037.

Raza, M.H., Gertz, E.M., & Mundorff, J. (2013) Paroxetine for treatment of obssive-compulsive disorder and comorbid stuttering. (2013) Linkage analysis of a large African family segregating stuttering suggests polygenic inheritance.

Hum Genet 132(4):385-396.

Sahin, H.A., Krespi, Y., & Yilma, Z. (2005) Stuttering due to ischemic stroke. Behav Neurol 16: 37-39.

Schreiber, Shaul M.D., Pick, & Chaim G. (1997) Paroxetine for secondary stuttering: Further Interaction of Serotonin and Dopamine. The Journal of Nervous & Mental Disease: vol 185(7): 465-467.

Sechi,G., Cocco,G.A., & D'Onofrio,M. (2006) Disfluent speech in patients with partial epilepsy: beneficial effect of levetiracetam. Epilepsy Behav 9(3):521-523.