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Case Study Open Access

Paroxysmal Spasmodic Dysphonia

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Abstract

Spasmodic Dysphonia (laryngeal dystonia) is a lifelong condition that causes the muscles of the voice to spasm. This creates breaks and pauses within sentences or between words, making it difficult to speak and be understood. The pauses or breaks can be as bad as every word, often increasing in intensity when frustration or anxiety occurs. This case study is focused on a 51-year-old male who had been experiencing changes in voice after specific triggers such as a poor nights rest, an emotional event or a respiratory illness. The events last for 1-3 days, where after he regains full function and ability to speak. The patients inciting event occurred years earlier just prior to a bloodletting procedure in treatment of polycythemia vera.

Introduction

Spasmodic Dysphonia (SD) is a rare speech disorder that is hypothesized to be neurologic in origin, specifically the basal ganglia (Cleveland Clinic). The exact cause of SD is unknown, however, most cases result from a trigger in the brain and nervous system, sometimes from psychological stress (Penn Medicine). It is characterized by task-specific voice dysfluency resulting from selective intrinsic laryngeal musculature hyperfunction and is typically a sporadic phenomenon [1-8]. Usually, the voice disruptions gradually increase over several months then become consistent and remain chronic without further progression [4]. The vast majority of those affected are female, with some estimates as high as 80% [1]. SD is rare; it affects roughly 50,000 people in North America and usually starts during middle age (30-50) (Mount Sinai). It is task specific, meaning it only occurs during speaking and does not affect emotional expression such as laughter, crying and shouting [3].

Case Study

The patient is a 51-year-old male who presented with broken and strained speech. The breaks in speech occurred between every word, increasing in intensity when the patient became more anxious or frustrated. He had been experiencing these episodes a few times per year and speculated they were caused by a lack of sleep or an inciting emotional event. The patient had an extensive medical history including over 20 surgeries involving his neck, spine, and knees. He had chronically high levels of white blood cells (WBCs), RDW, creatinine, immature granulocytes, neutrophil abs, monocyte abs, neutrophils, lymphocytes, and C-Reactive Protein (CRP) (Figure 1).

The primary event occurred years early when the patient was entering the hospital for a bloodletting procedure in treatment of Polycythemia Vera (PV). PV is a rare blood cancer that causes the bone marrow to produce an excess of red blood cells (RBCs) resulting in a thickening of the blood (Mayo Clinic). He had been bloodletting for the previous year and was becoming low on iron as documented by his hematologist. Before beginning the treatment, he began having trouble speaking and felt weak. The patient was then admitted, following a list of neurological tests which all returned normal. The significant dysphonia lasted for a few hours and subsided, leaving behind a few days of slightly disfluent speech. The patient stated that after a few days, he had completely returned to his regular fluency.

The next few months the patient experienced these attacks at a higher rate, some being brought on by slight triggers. These attacks then tapered off and he experienced them less often and less severe. He attempted to avoid his known triggers which include respiratory illness, poor sleep, fatigue, stress, anxiety, and other strong emotions. During an episode when he would take long breaks from talking, such as a day, he would recover faster (the next day).

On exam the patient was in no acute distress and was alert and oriented to person/place/time. He showed no loss of deep tendon reflexes (DTR) in all extremities and no signs of neurological deficits. The patient was able to speak in sentences when he used a higher voice or if he tried to sing. His normal voice resulted in the inability to say more than one word, often having to repeat the word. The patient would frequently get frustrated, and the dysphonia would increase in intensity, inhibiting the patient from starting words—often looking out of breath with a spastic appearance to his diaphragm. When the patient was not attempting to speak, he involuntarily made small grunts or loud short breaths. All imaging was found to be normal including a head CT and ECG.

Discussion

For muscle tension dysphonia, there is usually an inciting event that leads it to develop. These events may include surgery, virus, inflammatory illness, lesions and neurological conditions such as multiple sclerosis and Parkinsons disease (Penn Medicine). Although it has been described in the literature, the symptoms have not been well defined and may appear similar to those of vocal tremor or muscle tension dysphonia (MTD). Thus patients with SD might not be easily identified by local clinicians for treatment [2].

Christy Ludlow 2019 states the level of knowledge of the pathological mechanisms and the pathways involved in this and other focal dystonias is limited compared to progressive neurodegenerative disorders. As the disorder is not progressive, yet results in a chronic disability, a different type of molecular mechanism is likely involved and needs to be determined.

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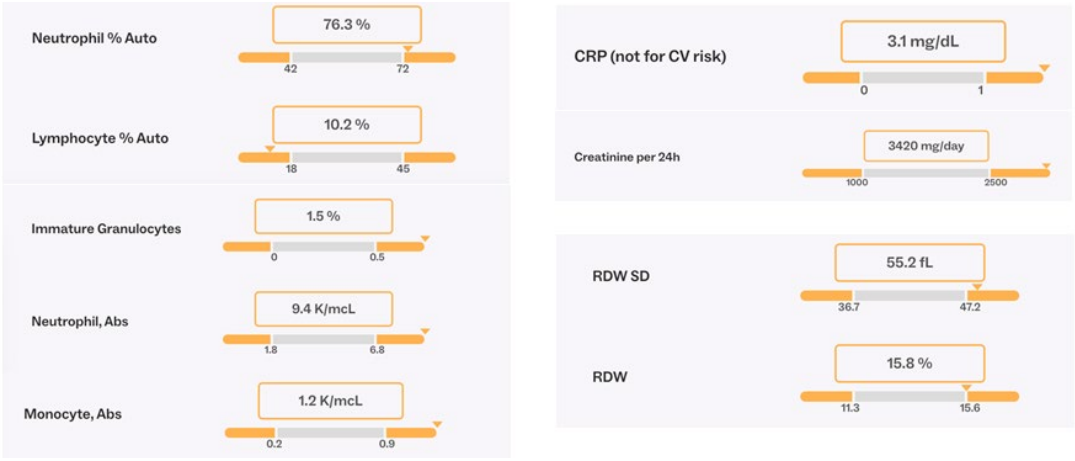

Figure 1: Patients labs were chronically high in inflammatory markers.

While there is currently no cure for SD, voice therapy and chemo denervation with botulinum toxin injections remain he mainstay of treatment [7-11]. No medications have been proven to provide constant relief from SD but a number of products are used to settle muscles or nerves that are spasmodic such as lorazepam, clonazepam, gabapentin, diazepam and other benzodiazepines (Dysphonia International). The patient has treated his attacks by using benzodiazepines to reduce the anxiety and muscle/nerve activity he experiences.

Conclusion

We believe that the patient was in a low iron state with high inflammation markers (a known cause for SD) which caused a neurological change, leading to an alteration in voice and the events he now experiences. These neurological changes are more profound and reemerge when the patient is in a weakened state such as sleep deprivation, high emotions or physically ill. The patient has been treated with benzodiazepines which do not cure the speech impairment but alleviate a significant burden of it. He takes long breaks from talking, such as a day, which hastens his recovery.

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