Conservative Approach to Tardive Dyskinesia–Induced Neck and Upper Back Pain

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The management of schizophrenia typically involves the use of antipsychotics (neuroleptics). Use of such medications, however, can result in tardive dyskinesia, or the involuntary contracture of muscles, and associated symptomatic somatic dysfunction. The authors present a case of a 29-year-old woman who presented to a family medicine clinic for ongoing management of schizophrenia with noticeable tardive dyskinesia and complaints of neck and upper back pain. Conventional management of tardive dyskinesia involves either a change in or reduction of the offending antipsychotic. In the present case, the patient received osteopathic manipulative treatment in addition to conventional care for the management of her neck and upper back pain. Although not curative, osteopathic manipulative treatment can provide palliative relief for patients with tardive dyskinesia.

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Schizophrenia affects approximately 1% of the population, with presentation occurring most commonly in individuals aged 15 to 35 years.[1,2] This mental disease appears to affect both male and female individuals equally.[1] There is a genetic correlation, but no single gene has been identified as a cause of the disease.[3] Rather, current theories propose that schizophrenia is caused by multiple genetic mutations that result in a multifaceted clinical presentation and course.[3] Approximately 10% of individuals with schizophrenia commit suicide, and substance abuse is common as these individuals attempt to self-medicate.[2] It is common practice for schizophrenia to be managed with antipsychotic (neuroleptic) medications to control highly frequent auditory hallucinations. Antipsychotic medications, however, are associated with tardive dyskinesia, or the involuntary contracture of muscles. This adverse reaction is more likely to occur in patients who receive first generation, or "typical," antipsychotics such as haloperidol.[3]

Tardive dyskinesia is purportedly a result of dopaminergic hypersensitivity[1,3] secondarily causing a cholinergic deficiency.[3] The orofacial muscles are most commonly affected, resulting in repetitive chewing or tongue-smacking movements.[4] Unwanted movements are not limited to the face, however; they can occur in the cranium, spine, thorax, and upper and lower limbs. Individuals with the classic orofacial involvement may be unaware of these movements, but others often find them to be embarrassing and disabling.[4] Time from the start of antipsy-

chotic medication to the onset of tardive dyskinesia is generally greater than 6 weeks.[4]

When administering antipsychotic medications, physicians should monitor patients for early signs of tardive dyskinesia because advanced cases of the disease are difficult to reverse.[3] It is estimated that even after the offending antipsychotic medication is discontinued, approximately 50% of patients will continue to experience chronic symptoms of tardive dyskinesia.[4] At the first sign of tardive dyskinesia, physicians should switch to an atypical antipsychotic with a lower risk profile such as quetiapine fumarate or clozapine.[3] Other pharmacologic management options include avoidance of centrally acting anticholinergics (particularly antiparkinsonisms and tricyclic antidepressants),[3] addition of benzodiazapines,[3] and the dopamine-depleting agent tetrabenzanine.[4,5] In addition to these pharmacologic treatment strategies, the Dystonia Medical Research Foundation also lists the following: botulinum toxin injections; physical, occupational, and speech therapies; surgical intervention; and complementary therapies (eg, biofeedback, yoga).[5] The Foundation did not list manipulative treatment as a therapy option for patients with dystonia.[5]

A literature review for manipulative management of tardive dyskinesia yielded a single case report in which chiropractic manipulation was used to manage pain associated with tardive dyskinesia.[6] It is understood that these treatments are not used to cure tardive dyskinesia but instead are used palliatively to improve the patient's pain and overall quality of life. The following case is an example of how osteopathic manipulative treatment (OMT) can be used to reduce pain and improve quality of life in a patient with tardive dyskinesia.

Report of Case

In July 2012, a 29-year-old woman presented to her family physician for ongoing management of schizophrenia; she had been receiving care at the same family medicine clinic since December 2010. The patient described "a lot" of pain throughout her neck and upper back. The patient characterized her pain as dull, aching, and constant. She reported that the pain began shortly after the onset of an "occasional, involuntary muscle twitch," which started in November 2011. The patient stated that the pain had progressively worsened and had become more constant, and that she had been treated on an as-needed basis with diclofenac. The patient reported that since she had developed the involuntary twitches, she had tried to self-manipulate her neck but had been unsuccessful. She scored her neck pain as a 7 on a 10-point scale (with 10 indicating severe pain). She denied pain referral, weakness, and paresthesia in the extremities, as well as bowel and bladder incontinence.

The patient's past medical history included 2 admissions to mental health facilities for psychosis in 2008 and 2010. The patient had been experiencing mild auditory hallucinations since her late teenage years. She did not recall a major effect to her activities of daily living, however, until she was hospitalized for 3 months in 2008 for her psychosis. In June 2011, the patient was started on haloperidol (15 mg orally at night) by her family physician. The patient did not recall what medication she received before she established care at the clinic, but she did admit to noncompliance with prior medications. The patient underwent a Loop Electrical Excision Procedure for cervical dysplasia in 2003 and radiation therapy for hyperthyroidism (the exact date of therapy was unavailable).

When the patient first described involuntary twitches in November 2011, tardive dyskinesia due to haloperidol was suspected. The patient received benztropine mesylate, lorazepam, and methocarbamol for the management of the tardive dyskinesia. In April 2012, a short course of haloperidol replacement with quetiapine fumarate was tried, but the patient's psychosis worsened, necessitating a reinstitution of haloperidol in May 2012 with accompanying medications. In July 2012, the

patient started haloperidol (15 mg orally at night) with olanzapine (10 mg orally per day), which appeared to be an effective alternative management option for her psychosis at 1-week follow-up.

The patient's family history included a paternal cousin diagnosed with bipolar disorder and reports of alcoholism in both the maternal and the paternal sides of the family. In addition, the patient's maternal grandfather had lung and pancreatic cancer, and her maternal grandmother had diabetes.

The patient was unemployed and living with her parents. She admitted to heavy alcohol consumption to cope with her psychosis and was a prior smoker with a 16 pack-year history. She denied recreational drug use.

Physical examination revealed an alert but distressed woman measuring 5 ft 8 in in height and weighing 230 lb. Vital signs were normal, and head, ears, eyes, nose, and throat; cardiopulmonary; and abdominal examination findings were unremarkable. Deep tendon reflexes, sensation, and strength were normal.

Musculoskeletal examination revealed tenderness throughout the cervical and upper thoracic paraspinal musculature. In addition to the involuntary spasms from tardive dyskinesia, somatic dysfunctions were noted in the upper cervical spine (C0 through C1 extended, sidebent right, and rotated left), the lower cervical spine (C5 through C7 flexed, sidebent right, and rotated right), and the upper to mid thoracic spine (T3 through T8 flexed). Active ranges of motion for both the cervical and the thoracic spine appeared moderately reduced but were difficult to ascertain because of the existing tardive dyskinesia. The patient's tardive dyskinesia contained mild involvement of the mouth, mostly involving the orbicularis oris muscle and the lip elevator muscles (levator labii superioris and zygomaticus major), with moderate involvement of the right sternocleidomastoid muscle that resulted in slow, repetitive involuntary motions of the head into right lateral flexion with left rotation. Involvement of the sternocleidomastoid muscle could have been the cause of the patient's cervical somatic dysfunctions and associated neck pain; a somatosomatic reflex was observed in the sternocleidomastoid muscle (innervated by the spinal accessory nerve, which is composed of fibers from C1 through C6), where it overlapped with areas of her palpated cervical somatic dysfunctions.

A third-year osteopathic medical student who was completing a rotation at the clinic (J.W.R.) treated the patient's somatic dysfunction first with soft tissue techniques to provide a general sense of relaxation and then with high-velocity, low-amplitude (HVLA) thrusts to both the cervical and the thoracic regions. After treatment, immediate improvement of the somatic dysfunctions was noted. The patient reported an immediate reduction of her pain level to 1 out of 10. At 1-week follow-up, the patient continued to report improvements to her symptoms and also reported improvements to her quality of life (decreased pain with improved mood, concentration, and sleep) and a substantial reduction in alcohol use. However, involuntary spasms from tardive dyskinesia continued without noticeable change. The student was unable to continue monitoring the patient's condition because of a change in rotation assignment, but the patient was encouraged to pursue palliative manipulative therapy for associated future pain.

Comment

Fortunately, tardive dyskinesia is not as common today in patients with schizophrenia because of the advent of atypical antipsychotics. As seen in the present case, however, the typical antipsychotics (ie, haloperidol and chlorpromazine) are still used, and thus physicians should be alert to the potential development and recognition of tardive dyskinesia. Although tardive dyskinesia may be irreversible, particularly if the symptoms are not caught early, the physician can attempt to provide palliative relief through various avenues.

Spinal manipulation has been found to be relatively safe and effective for the management of neck pain. In 2009, the American Osteopathic Association House of Delegates reaffirmed its 2004 position paper supporting the use of cervical HVLA by osteopathic physicians on the grounds that the benefits outweighed the risks.[7] If performed appropriately, HVLA treatment of the spine

could be beneficial for a patient experiencing pain from tardive dyskinesia.

We used the HVLA technique in the present case because of its rapid administration, which was used to intercede between involuntary spasms. Other techniques such as counterstrain, muscle energy, and balanced ligamentous tension, although possibly efficacious, would have required a longer treatment interval that would have been interrupted by the involuntary spasms. In addition, we believed that HVLA would result in a faster resolution of the patient's neck and upper back pain.

As previously mentioned, to our knowledge only 1 other case of manipulative treatment for tardive dyskinesia has been published. The chiropractic technique described in the previously published case seemed similar to the HVLA technique used on our patient. The chiropractic manipulation resulted in a similar positive response; according to the case report, the patient continued to follow up with palliative care for her chronic tardive dyskinesia.[6] Although we were unable to follow up with the patient in the present case, we believe it is likely she would have continued to benefit from ongoing OMT sessions. However, it would be imprudent to assume that 1 treatment would be sufficient for a chronic neuromusculoskeletal condition such as tardive dyskinesia, which is likely to result in repetitive somatosomatic reflexive spinal dysfunction.

Conclusion

Although it is unlikely that spinal manipulation would result in a reversal of tardive dyskinesia, osteopathic physicians should consider the use of OMT to address pain associated with tardive dyskinesia. With relief of pain, patients can have an improved quality of life, with positive changes to their overall mental and physical health. A controlled clinical trial assessing the safety and effectiveness of spinal manipulation over a longer time frame would be helpful to establish further use of OMT in the management of tardive dyskinesia–induced neck and back pain.

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