Trigeminal neuralgia associated with seizure and syncope

Case report

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A patient with trigeminal neuralgia experienced a generalized seizure and a prolonged syncopal episode. He was found to be asystolic during the syncopal episode. There was no recurrence of loss of consciousness after implantation of a pacemaker. Mechanical stimulation of the trigeminal nerve during craniotomy for microvascular decompression of the trigeminal nerve resulted in bradycardia. Since vascular decompression of the trigeminal nerve, there has been no recurrent facial pain, and no further syncope, seizures, or bradycardia. Syncope and seizures have not been previously reported in association with trigeminal neuralgia, although they are well described with glossopharyngeal neuralgia.

KEY WORDS □9 trigeminal neuralgia □9 syncope □9 trigeminal nerve □9 seizure

SYNCOPE, seizures, and cardiac arrest are well described in association with glossopharyngeal neuralgia, but have not previously been reported with neuralgia affecting other cranial or peripheral nerves. We report a patient who had trigeminal neuralgia associated with syncope and seizures due to bradycardia and asystole. The episodes of loss of consciousness did not recur after a pacemaker was inserted. The neuralgia and bradycardia later resolved with microvascular decompression of the fifth cranial nerve.

Case Report

This 60-year-old man was in excellent health until May, 1981, when he developed the abrupt onset of excruciating pain over the upper part of his left cheek, his left eye, and left temporal area, radiating toward the left ear. This brief episode of pain resolved spontaneously, but subsequently he had two other episodes during a 1-month period. The third episode was accompanied by a generalized seizure. No neurological deficit was found after the ictus. Approximately 2 months later, the patient experienced similar pain associated with loss of consciousness but unaccompanied by tonic-clonic movements. The patient was unresponsive for approximately 15 minutes, and paramedics upon arrival could not obtain a pulse or blood pressure. The patient was considered to have had a cardiac arrest, but, while en route to a local hospital, he regained consciousness and was free of pain. At this time his pulse rate had increased to 40 beats per minute and blood pressure to 90/50 mm Hg. The patient had no prior illnesses or syncopal episodes and had not been taking medications. His general physical examination and detailed examination of the cranial nerves were normal. No trigger points were demonstrated on the face. Repeated electrocardiograms were normal and he had no evidence of a myocardial infarction.

A permanent demand pacemaker was inserted after the episode of loss of consciousness; the pacemaker was set at 50 beats per minute. After insertion of the pacemaker, the patient continued to have recurrent facial pain which became more frequent and required institution of carbamazepine (800 mg a day). However, carbamazepine had to be discontinued because of development of side effects. He experienced no further syncopal episodes or convulsions after pacemaker insertion; however, his pulse rate slowed to 50 beats per minute (counted on numerous occasions by his wife) during the pain, and on those occasions he felt activation of the pacemaker. Because of persistent pain, the patient was referred to us for microvascular decompression of the trigeminal nerve.

Examination. Further evaluation showed normal baseline laboratory tests, skull x-ray films, electroencephalography, computerized tomography of the head, and Holter monitor. Cerebral angiography showed a
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markedly long and tortuous basilar artery. The patient underwent left retromastoid craniotomy.

Operation. With microsurgical exposure of the cerebellopontine angle, vascular compression of the root entry zone of the trigeminal nerve was demonstrated. The portio minor was markedly stretched and compressed by the superior cerebellar artery on the rostral side with the lateral branch of the superior cerebellar artery compressing the root entry zone of the portio minor. These arteries were mobilized and were held away with several small implants of shredded Teflon felt. The remainder of the trigeminal nerve looked normal. Mechanical stimulation of the trigeminal nerve intraoperatively resulted in slowing of his heart rate from 76 to 50 beats per minute, at which point the pacemaker impulses appeared on the electrocardiographic monitor. The patient’s blood pressure fell from 140/70 to 107/60 mm Hg during stimulation. The pulse rate and blood pressure returned to normal upon discontinuation of fifth nerve stimulation. Evaluation of the ninth and 10th cranial nerves disclosed slight stretching of the ninth cranial nerve by the posterior inferior cerebellar artery which was not considered significant. Compression of the trigeminal nerve was considered the most impressive and important finding during surgery.

Postoperative Course. Over 22 months of follow-up review there has been no recurrent facial pain or further syncope, seizures, or bradycardia.

Discussion

Our patient had clinical manifestations of trigeminal neuralgia. After one episode, he presented with loss of consciousness and was found to be without a pulse. Implantation of the pacemaker resulted in abolition of further episodes of loss of consciousness associated with neuralgia by maintaining the heart rate at a rate of 50 beats per minute. Activation of the pacemaker with stimulation of the trigeminal nerve during operation lends further support for the bradycardia being initiated by the trigeminal neuralgia. We believe that the initial episode of seizure with pain also resulted from bradycardia and hypotension.

Syncope, seizures, and cardiac arrest have not previously been reported in association with trigeminal neuralgia, although more than 30 cases have been described in association with bradycardia due to glossopharyngeal neuralgia. The clinical presentation of the pain in this patient was not suggestive of glossopharyngeal neuralgia and, although minor compression of the ninth cranial nerve was found on surgical exploration, this was not thought to be important. A vasodepressor (or vasovagal) reaction to pain is also unlikely because this patient did not experience recurrent syncope after pacemaker implantation.

The physiological mechanism of bradycardia associated with trigeminal neuralgia is not clear. This response may have a mechanism similar to that postulated for photic sneezing, which is termed “parasympathetic generalization.” Parasympathetic generalization refers to stimulation of one branch of the parasympathetic nervous system which leads to spread of excitation to other branches. The rare sensory reflex of sneezing induced by exposure to very bright light is thought to be evoked by the fifth cranial nerve. A similar mechanism may be operative in our patient.

Although balloon compression or direct mechanical stimulation of the trigeminal nerve is associated with bradycardia and hypotension, to our knowledge this patient represents the first reported case of trigeminal neuralgia resulting in bradycardia and loss of consciousness. Treatment of syncope should be directed initially toward the control of neuralgia with diphenylhydantoin and/or carbamazepine. In refractory cases, vascular decompression as performed in this case may result in abolition of neuralgia and syncope. If the neuralgia and syncope are uncontrolled or the patient is not a surgical candidate, a permanent demand pacemaker may abolish the recurrences of syncope.

References


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