

Neck and brain transitory vascular compression causing neurological complications. Results of surgical treatment on 1,300 patients.

Review of 1,300 cases of patients operated in Hato Rey Community Hospital

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Transitory Neck and Brain Vascular Compression Causing Neurological Complications

SUMMARY: This article describes our experience with the surgical treatment of over 1.300 patients with Cerebellar Thoracic Outlet Syndrome (CTOS) who had associated neurological lesions as a result of the hypoperfusion and hypometabolism of Certain areas of the brain and cerebellum. Chronic hypoxia was found to produce Symptomatic neurological syndromes such as Parkinson's disease (SPD), Chorea, Ballism and Athetosis. The surgical technique was found to have excellent results in 96% of CTOS patients. In a follow-up study of patients with CTOS / SPD, 90.6% of the symptoms Suffered by the patients, were eliminated or showed improvement after surgery. No improvement was shown in 9.4% of the operated patients.

This article describes the role of compression of the vertebral arteries, subclavian, internal mammary, internal carotid, brachial plexus, the coil (crease) and kink of the vertebral and basilar arteries in the absence of blood supply and oxygen to the brain, cerebellum and the basal ganglia of the brain. Chronic hypoxia produces different results, depending on the area of the brain and cerebellum are affected. When chronic hypoxia affects the basal ganglia in the putamen, there is a decrease in dopamine production causing the symptoms of Parkinson Symptomatic. When hypoxia affects the caudate nucleus, causes the symptoms of chorea, when it affects the thalamus or hypothalamus, it produces the tribalism and by affecting the lenticular nucleus, athetosis occur. This compression can be caused by the anterior scalene muscles and the cervical ribs at the level of C6-C7 vertebrae, by the sternocleidomastoid muscles at Atlas, for minor pectoral

muscles (steal syndrome) to the subclavian arteries and the coil (crease) and kink of the vertebral arteries and congenital cause.

The lack of blood supply to the cerebellum, basal ganglia and brain, is the cause of Cerebellar Thoracic Outlet Syndrome (CTOS) and its neurological complications, among which are: ipsilateral paralysis, Parkinson's disease symptoms, the disease Alzheimer's and other functional. We are currently conducting several studies to extend our understanding of this phenomenon. 'Cerebellar Thoracic Outlet Syndrome' (CTOS), Parkinson's disease, Alzheimer's disease and other

The term Thoracic Outlet Syndrome (TOS) was introduced in the medical literature by Peet and Anderson in 1956, characterized as a neurological disorder (1). In 1983 the first patient was operated with our surgical technique (described below) in a patient suffering from this syndrome and two of its neurological complications, ipsilateral paralysis and Parkinson's disease, and was disabled in a wheelchair. The excellent results obtained in this patient postoperatively led the author to propose two theories: 1) that the TOS is a disorder, primarily vascular and neurological complications 2), that the Parkinson's symptoms is a functional problem and not organic to the core level basal brain. This disorder affects the cerebellum and other brain areas by hypertrophy of the anterior scalene muscle at the level of C6-C7 cervical vertebrae (Fig. 1.2) and as a result of transient compression of the vertebral arteries, subclavian, internal mammary, internal carotid and brachial plexus. This syndrome may also result from compression of the vertebral and internal carotid arteries by the sternocleidomastoid muscles and at the level of the Atlas (Fig. 1, 2); for minor chest muscles with severe compression of the subclavian arteries, fibromyositis of the trapezius muscles and the coil (crease) and kink of the vertebral arteries (Fig. 3), basilar and internal carotid. For this reason, the author called this syndrome Cerebellar Thoracic Outlet Syndrome (CTOS).

Objectives

Vascular compression of the Cerebellar Thoracic Outlet Syndrome has been confirmed at Mount Sinai Hospital in Miami since 1985 with Digital Intravenous Injection (IVDSA) (Fig. 4.5), with the PET scan (Fig. 6.7) and the Transcranial Doppler (1994), which clearly demonstrates compression of the vertebral and internal carotid arteries and the Single Photo Emission Computed Tomography (SPECT Brain). This transient intermittent compression affects the normal functioning causing insufficient blood supply to the cerebellum and the brain. The results of this condition is intermittent functional disorders that result from inadequate blood level of the brain capillaries and arterial angiotensin II peptides. In cases where post-operatively there is only a partial recovery is that you probably had a heart attack in the arterioles and capillaries causing irreversible damage in the area. We should also note that the years do not necessarily come to produce disease organic lesion. We have seen cases of patients who have had the disease for 27 years and have recovered fully after the operation, as well as cases of patients who have been ill

for shorter periods with organic lesions in certain areas and have only partially recovered.

The many and varied vascular and neurological symptoms produced in the manner described become progressively more severe until complications occur such as paralysis ipsilateral, the threat of gangrene in the arms, the progressive loss of vision, Parkinson's disease, Alzheimer's functional and others.

Unlike what was believed until now (2), we concluded that Parkinson's disease is more functional than organic. This is due to intermittent irrigation and inadequate oxygenation of the cells that produce dopamine level of the basal ganglia (putamen, caudate and globus pallidus) and the cortex of the brain, caused by a complication of Cerebellar Thoracic Outlet Syndrome (CTOS) . This mechanism has been demonstrated with the PET Scan at McMaster University in Hamilton (Canada) in 1985 (Fig. 6.7), in June 1990 with the Single Photo Emission Computed Tomography (SPECT Brain) and in 1994 with Transcranial Doppler Ultrasound as a result of arterial compression in the Thoracic Outlet and the internal carotid and vertebral arteries to the entry of the brain (3). The putamen is irrigated by the middle cerebral artery branch of the carotid and posterior cerebral artery, terminal branch of the basilar artery. The posterior cerebral arteries supplement irrigation to different areas of the forebrain.

When it affects irrigation and oxygenation of the putamen by compression of the vertebral arteries and internal carotid tremor occurs Parkinson's Disease Symptomatic. When it affects the caudate nucleus, chorea occurs. This has been corroborated by the great improvement of 40 patients with chorea, after eliminating the compression of the vertebral and internal carotid arteries. As shown in Figure 1, the internal carotid arteries via the Circle of Willis, supply the anterior portion of the brain and the vertebral arteries supply the posterior and several areas of the anterior brain (4). Symptoms of Symptomatic Parkinson Disease (SPD) disappear when the patient is asleep or sedated, but increase with hyperabduction maneuvers and arterial compression by stress. If there is permanent damage to the substantia nigra, the symptoms would not disappear with rest and / or sedation. Symptoms are present at all times when there is an organic arterial injury, toxic injury or hemodynamic Parkinsonism due to coil (crease) and the kink of the vertebral and basilar arteries, as was discovered by the author in June 1990 (Fig . 3). Approximately 25% of patients with CTOS / SPD are associated with Alzheimer's disease.

It is important to emphasize that we are referring and treating Parkinson's disease, diagnosed by independent neurologists and by our assessment team, which includes neurologists.

Although they share some common symptoms, Symptomatic Parkinson's disease differs from secondary parkinsonism, a disorder caused by organic lesions such as vertebral-basilar insufficiency (VBI), injuries caused by toxic drugs and / or hemodynamic Parkinsonism.

When there is an organic disorder, circulatory failure is partially compensated through the circle of Willis even permanent symptoms may be present, not always. For example, try a case where a bullet injured his right carotid artery, we inserted a plastic artery Dacron and yet the patient died that night. The autopsy revealed a total brain damage. The left carotid artery was not enough to offset the movement of the brain through the circle of Willis. In 35% of cases, the estate is unable to provide the necessary circulation to the brain when there is damage in one of the carotids. The factors that affect total cerebral circulation are the blood pressure in the brain, the venous pressure, intracranial pressure, blood viscosity and the degree of active contraction or dilation of cerebral arterioles. The caliber of the arterioles is controlled by local vasodilator substances including products of metabolism and self-regulation of circulating peptides such as angiotensin II and vasomotor nerves.

Through the use of Positron Emission Tomography (Pet Scan) in 1985, we noted that we see a reduction in the amount of dopamine produced at the putamen in Parkinson's patients when compared with the normal subject (Fig. 6, 7). The technique of ¹³³Xe and the Pet Scan have been applied to study several diseases, including epilepsy, memory deficit, agnosia, dementia, Huntington's disease, chronic schizophrenia and manic depression. Interesting is that in all these diseases, the PET scan has proven inadequate glucose metabolism (Fig. 6.7). Although perhaps only a coincidence, this seems a pattern similar to that described in cases of CTOS / Symptomatic Parkinson's, Alzheimer's disease and others. In fact, we operated on six patients with CTOS and psychological disturbances, secondary to hypoxia, and two patients with CTOS / psychological disturbances and Parkinson's disease. All experienced great improvement in psychological disturbances and symptoms of CTOS. Another interesting situation is that of patients with CTOS and Parkinson's disease with chorea have been healing and significant improvement in 40 patients operados. Trece cases involve patients with the diagnosis of CTOS / Epilepsy. Of these, eleven were cured after the operation without the need to prescribe drugs, and two of them needed drugs but have not returned to have more seizures.

The AD has been described in medical literature as caused by structural lesions. We have operated 30 patients with Alzheimer's "functional" associated with CTOS and have achieved cure rates of 88%. The Alzheimer functional is a complication of Cerebellar Thoracic Outlet Syndrome (CTOS) in which there is damage to arteries as occurs in Alzheimer's organic. We also diagnosed with early Alzheimer's disease, where there is the CTOS and the patient says suffer from temporary absences and / or slight disorientation. This has been demonstrated clinically since 1990 and Single Photo Emission Computed Tomography (SPECT Brain) by decreased blood flow in brain areas parieto-temporal, parieto-occipital, temporal and frontal. It has also been confirmed by publications of technological advances in nuclear medicine. Twenty percent of patients with CTOS / SPD had Alzheimer's disease. We also operated 17 patients with MS and 12 of these have shown great improvement.

Using our surgical technique, remove the compression of the vertebral arteries at the level of C6-C7 and entry into the brain at the level of vertebral and internal carotid arteries, with excellent results in 3,800 procedures performed in over 1,300 patients suffering from CTOS and its neurological complications. Table 1 represents the percentage of patients operated on CTOS / SPD and its results. After surgery, patients continue taking the drugs for Parkinson's disease: L-dopa, and Eldepryl Amantidine chloride, whose doses are tapered off gradually until symptoms improve or until, in some cases, medication is unnecessary. In all cases, physical therapy is given to patients before and after surgery.

Methodology

1. CTOS / symptomatic Parkinson: Twenty patients were diagnosed with SPD CTOS and developed severe stress due to transient compression by the vertebral artery by the anterior scalene muscles and sternocleidomastoid.

2. Patients with CTOS / SPD under general endotracheal anesthesia with our maneuver, developed the following symptoms:

- * A. high blood pressure
- * B. supra-ventricular arrhythmia
- * C. Cheyne Stokes respirations as a result of cerebral hypoxia.

Our maneuver includes:

1. Holding a deep breath
2. Bend your head up on the right or left shoulder. After our surgical technique, all the above symptoms disappear.

Ten patients had early symptomatic CTOS and Parkinson (ESPD).

Fourteen patients had severe compression of the subclavian and internal mammary arteries for minor chest muscles, cyanosis, coldness, pain and absent radial pulse. All symptoms disappeared after the operation.

Four patients had severe compression of the subclavian arteries with hyperabduction of "steal syndrome". After bilateral section of the pectoralis minor muscles, the symptoms disappeared.

Twenty patients with CTOS / SPD and hypertension did not need treatment after surgery.

The regular maneuvers Doppler revealed an 89% compression of the subclavian arteries in patients with CTOS / SPD and 94% compression using our maneuver.

Transcranial Doppler with our move from the vertebral arteries, internal carotid and its branches, showing a decreased blood flow, which he was normal in neutral position.

The compression of the vertebral arteries occurs in the opposite direction to where the tremor begins CTOS / SPD, as demonstrated with Doppler studies and Trans-cranial Doppler.

We believe that the study of Brain SPECT should be performed each CTOS patients with severe, particularly in patients with symptoms of Parkinson's symptoms. The Brain SPECT studies were positive for Parkinson's disease (Table 2). Surgical treatment of CTOS prevent the neurological complications associated with our technique and stop the progress of these diseases.

Etiology, diagnosis and treatment and complications CTOS

The etiology of this disorder can be first, congenital, caused by cervical ribs, fibrosis and muscle bands. We have discovered the coil (crease) and the kink of the vertebral arteries as a cause congenital in 84% of patients with CTOS / SPD through 600 "Intravenous Digital Subtraction Angiography (IVDSA) made. The second cause is acquired: first, by hypertrophy of the scalene muscle, sternocleidomastoid and pectoralis minor, second, by occupational roles such as in cases of secretaries, teachers, postmen, workers in industrial machines, severe stress, etc.. And thirdly, by stretching the brachial plexus, all producing compression of the vertebral arteries, subclavian, internal mammary and internal carotid arteries leading to the temporary lack of oxygen to the brain and cerebellum.

We conducted trials in over 10,000 patients and 1,500 CTOS CTOS in patients with symptoms of Parkinson Symptomatic. Of the surgical procedures performed 3.800, more than 1,300 patients were patients with CTOS and 400 patients also had SPD and other associated neurological complications.

To diagnose this syndrome, we do a complete medical history and the four diagnostic maneuvers, the Adson, hyperabduction, hyperextension and a new maneuver we have developed.

In addition to the above, we use the following diagnostic studies:

3. Imaging of the spine and thorax
4. Doppler ultrasonography with regular exercises and our maneuver in the upper extremities
5. Electromyography (EMG)
6. Study of nerve conduction velocity (NCV)

We added the following diagnostic studies:

7. Somatosensory evoked potentials of short latency of the upper limbs (SEP), which we identified as a specific diagnostic tests in the diagnosis of SPD due to the alteration of P14 waves caused by lack of oxygen and irrigation to the putamen and cortex brain, since 1984
8. Digital Intravenous Injection (IVDSA) head and neck, since 1985
9. Positron Emission Tomography (PET) scan for patients with Parkinson's disease since 1985
10. Single Photo Emission Computed Tomography (SPECT Brain), since 1990 (Table 3)
11. Transcranial Doppler ultrasonography with our maneuver, since 1994

Symptoms produced by CTOS

Headaches (migraine), neck, chest and arm numbness, chest, dyspnea transient memory deficits, dysphagia, dizziness, tinnitus, urinary incontinence, slurred speech, loss of consciousness, paralysis ipsilateral, severe stress, joint temporomandibular, amaurosis fugax, tachycardia, dysmenorrhea, heavy bleeding during menstruation, paresis, snoring and others.

Symptoms caused by Parkinson

Tremors, impaired writing, sialorrhea, seborrhea, shuffling gait, chorea, rigidity, aspect of monkey, and sexual impairment.

Complications CTOS

A. Described by other authors

12. Aneurysm of the subclavian and vertebral arteries
13. Thrombosis

B. Described by the author (11)

14. Ipsilateral paralysis
15. Loss of temporary or permanent vision
16. Full compression of the subclavian artery with impending gangrene of the upper extremities
17. Symptomatic Parkinson's Disease

18. Early Alzheimer's Disease
19. Pulmonary complications
20. Alzheimer's Disease Functional
21. Epilepsy
22. Multiple Sclerosis
23. Psychological disturbances due to hypoxia
24. Parkinsonism hemodynamic
25. Symptomatic early Parkinson's disease (ESPD)

Differential Diagnosis

This diagnosis includes cervical spondylitis, Pancoast tumor, spinal cord tumor, angina, myositis, herniated disc, carpal tunnel syndrome, etc.. The author has also pointed out: the CVA (Stroke), Takayasu's disease, paralysis ipsilateral basilar insufficiency lobby (VBI), "steal syndrome" of the subclavian, parkinsonism hemodynamic, symptomatic Parkinson's disease early (ESPD), Alzheimer Functional Alzheimer Organic, Early Alzheimer's, Epilepsy, Multiple Sclerosis, psychological disorders due to hypoxia and organ damage whose symptoms mimic those of Parkinson's disease.

Recommended treatment

Under general endotracheal anesthesia and sterile conditions, place the patient in supine position with a serum bottle under the chest. Bilateral neck incisions, 4 cm above the clavicle. The incision is 4 cm in length and is made transversely in the area where you can feel the scalene muscles.

26. The sternocleidomastoid muscle are sectioned bilaterally with electro-coagulation of the cervical level C6-C7
27. The pre-scalene fat is dissected
28. When the jugular vein in front of the anterior scalene muscle is separated
29. The phrenic nerves were then dissected
30. The anterior scalene muscles are snipped at C6-C7, and also the insertion of the cervical
31. The interspinous ligament is cut at the same level
32. When stage III cervical ribs, the accessions are fought and cut the distal end of the cervical rib
33. When a "subclavian steal syndrome", is performed bilateral section of the pectoral muscles under
34. A thin drain "Penrose" is set and left for about two days
35. The surgical wounds were sutured with 5-0 Dacron

Conclusions

The benefit of this surgical technique appears to disappear immediately and vascular and neurological compression caused by anterior scalene muscle at C6-C7, and entry-level brain atlas by sterno-mastoid muscle. This has been demonstrated in patients with CTOS, Symptomatic Parkinson's, Alzheimer Functional Parkinson Symptomatic early psychological disturbances due to hypoxia, epilepsy, multiple sclerosis, Parkinsonism hemodynamic and threat of gangrene of the upper extremities. The superiority of this technique over other techniques used to treat this condition is demonstrated by a comparative study of surgical results presented in Table 3.

We have presented the Cerebellar Thoracic Outlet Syndrome (CTOS) as a common denominator associated with neurological damage caused mainly from lack of oxygen to the brain and cerebellum, followed by metabolic disorders, vasomotor and Angiotensin II to which we have obtained excellent results improvements and cures reinforced by an etiology, pathophysiology and proposed a new surgical technique without any fatality during treatment.

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