Case Report

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Horner's Syndrome due to a Spontaneous Internal Carotid Artery Dissection treated with Casper Stent

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Abstract

Horner syndrome is a constellation of neurological findings consisting of ipsilateral ptosis, miosis and anhidrosis. Partial Horner syndrome, comprising ipsilateral ptosis and miosis in the absence of anhidrosis, is a well-documented but uncommon manifestation of internal carotid artery dissection. Internal carotid artery dissection (ICAD) is a rare entity that either results from traumatic injury or can be spontaneously preceded or not by aminor trauma such as sporting activities, is a common cause of ischaemic stroke in young patients. As many as 40 to 50% of patients with carotid dissection will develop an ipsilateral Horner's síndrome.

Keywords: Horner's syndrome; Carotid artery dissection; Anisocoria; Miosis; Ptosis; Oculosympathetic pathway; Carotid artery stenting; Dual antiplatelets

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Introduction

Horner syndrome (HS) was first described by Francois Pourfour du Petit in 1727 but was named after a Swiss ophthalmologist Johann Friedrich Horner in 1869. HS is noted in about 25% of patients with carotid artery dissection (CAD). An association with intrinsic vessel structure has been reported in connective tissue disease, pregnancy and the postpartum period, and in infectious and inflammatory disease. Non-invasive diagnostic modalities including magnetic resonance imaging, computed tomography, and duplex ultrasonography have become an alternative to digital subtraction angiography, however this still remains the gold standard, with better detection of thrombus and collateral circulation.

Case Report

A 45-year-old male bends his neck and feels moderate pain in the cervical region while playing with his dog, 2 weeks ago who was treated with analgesics to control the pain. However 2 days before going to the emergency service to the clinic the right craniocervical pain intensifies not yielding with common analgesics.

The patient has no significant medical history, does not report allergies, occasionally consumes alcohol, and denies drug use. The examination of the cranial nerve territory showed a right myosis and ipsilateral ptosis. The rest of the neurological examination was normal. The haematological and biochemical profiles were normal. The electrocardiography trace showed sinus rhythm at a rate of 64bpm. The patient was diagnosed with a painful right-sided, incomplete Horner's syndrome based on the anisocoria and ptosis. He was hospitalized to complete the study. An angiotomography and magnetic resonance image of the brain and MRA of the extra- and intracranial arteries were performed. It revealed a decrease in the caliber of the right ICA with an eccentric and irregular stenosis (string sign) (yellow and White arrows) **(Figure 1)**.

The patient was started on double platelet antiaggregation with acetylsalicylic acid plus clopidogrel for one week, then was scheduled for endovascular surgery in the right internal carotid cervical segment, correcting the arterial dissection with recovery of the normal caliber of the artery, after three days his clinical condition improved and he was discharged home. The ptosis persists and the cervical pain improves being mild and controlled with NSAIDs, The rest of the neurologic examination was normal. It is indicated to continue double platelet antiaggregation for 3 months and single antiplatelet therapy with aspirin was continued indefinitely **(Figure 2)**.

Procedure

Two days after admission, a cerebral and cervical vessel angiographic study was performed, using a trans femoral

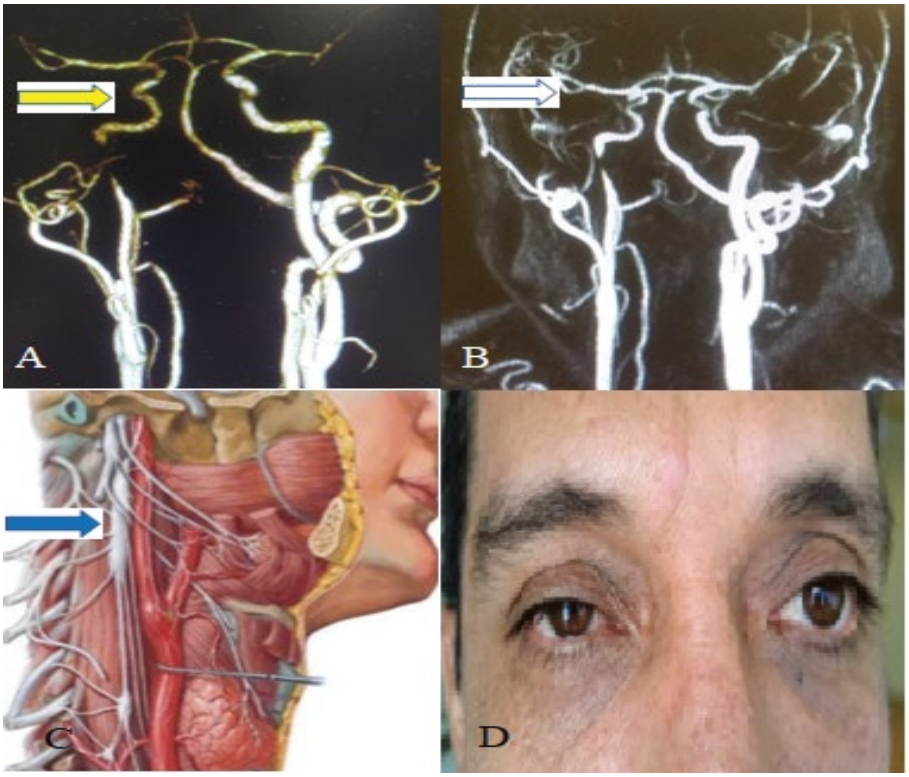

Figure 1 **(A)** study of cervical vessel angiotomography demonstrating the right internal carotid stenosis in the cervical segment (yellow arrow). **(B)** Angioresonance shows stenosis of the right internal carotide (white arrow). **(C)** Anatomic cutting of the right internal carotid artery showing inervation by the fibers of the sympathetic system that travels the arterial wall in an ascending form (blue arrow). **(D)** Patient image showing palpebral ptosis and right miosis).

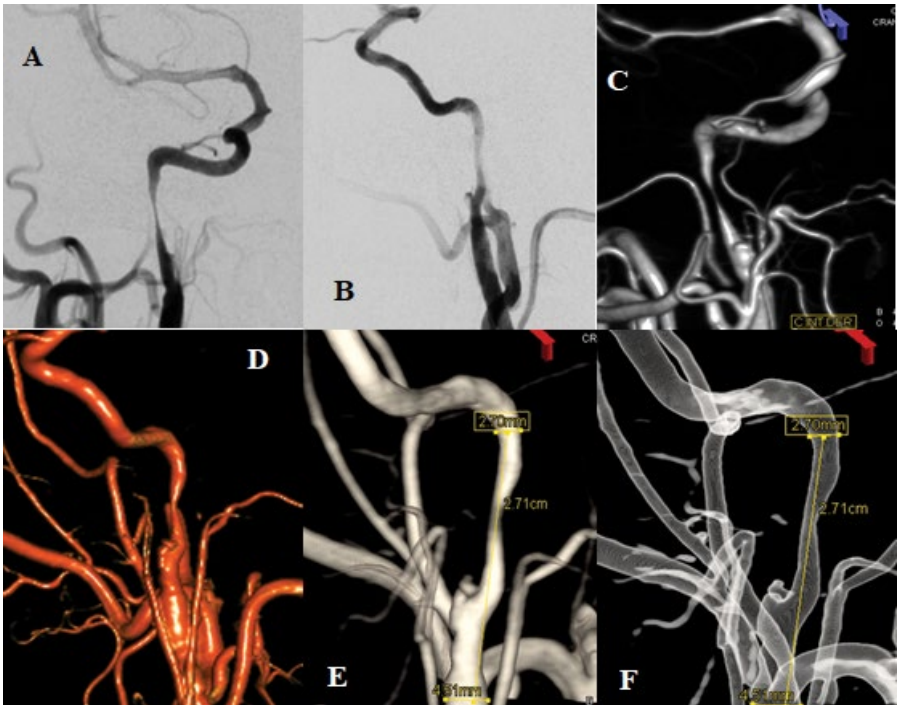

Figure 2 **(A, B)** Angiography posteroanterior and lateral proyections, common carotid injection, showing a dissection of the right internal carotid above the bulb to the start of the petrous segment. **(C-F)** 3D reconstruction showing the segment of arterial dissection with measurements and severe lumen stenosis of the right internal carotid.

approach and with general anesthesia, which demonstrated dissection of the right internal carotid in the cervical segment above the carotid bulb until the start of the petrous segment whose diameter The distal carotid artery was 2.7mm, proximal 4.7mm and the dissection extension was 27.0mm, with a decrease in the lumen of the artery by approximately 85%, with little blood passage to the right cerebral hemisphere, the irrigation of this The hemisphere is provided by the left internal carotid through the anterior communicant and the vertebrobasilar system through the posterior communicant. The patient returned to bed expecting to complete the double Antiplatelet therapy (aspirin 100mg and clopidogrel (Plavix) 150mg) for 6 days before the stent placement. The day of surgery was done under general anesthesia and foley catheter, we placed an 8f femoral introducer on the right side and advanced with a Ballast™ long sheath 088 guide catheter with the help of a hydrophilic guide, reaching the cervical segment internal carotid passing the bulb, and an angiographic study is carried out, the diameter of the internal carotid artery proximal to, at and distal to the lesion, as well as the length of the target lesion, were measured and recorded. The delivery system was then advanced supported by a Traxcess 14 microware (MicroVention Terumo, Tustin, California, United States) under fluoroscopy and positioned with the radiopaque inner shaft markers (distal and proximal ends of the stent) distal and proximal to the target lesion. The Casper Stent (MicroVention, Terumo, Tustin, California, USA) was then deployed under constant fluoroscopy by slowly with drawing the delivery catheter and holding the delivery wire in a secure stationery position. Final angiographic control shows that the Casper stent is adequately deployed from the petrous segment at the distal level, covering the entire extension of the arterial dissection of the internal carotid artery, with the artery having recovered its light, with adequate blood flow to the right cerebral hemisphere, there was no distal embolization or dissection and the rest of the vessels present **(Figure 3)**.

Discussion

Spontaneous ICAD has been reported to cause various combinations of cranial nerve palsies involving upper as well as lower cranial nerves [1]. The clinical manifestations of the ICAD can be local symptoms and signs suchas unilateral headache (periorbital and frontotemporal, facial, or anterior neck pain), Horner's síndrome (myosis, ptosis, and anhydrosis), and cranial nerve paralysis. Horner's síndrome is due to compression, stretching, or hypoperfusion of the sympathetic fibres within the carotid wall [2]. SCAD can cause a variety of symptoms, however headache, Horner's syndrome, and ischaemic stroke are the most common. Headache occurs in 70% of SCAD cases [3] and neck pain is also seen in 26% cases [4] Horner's syndrome is induced by

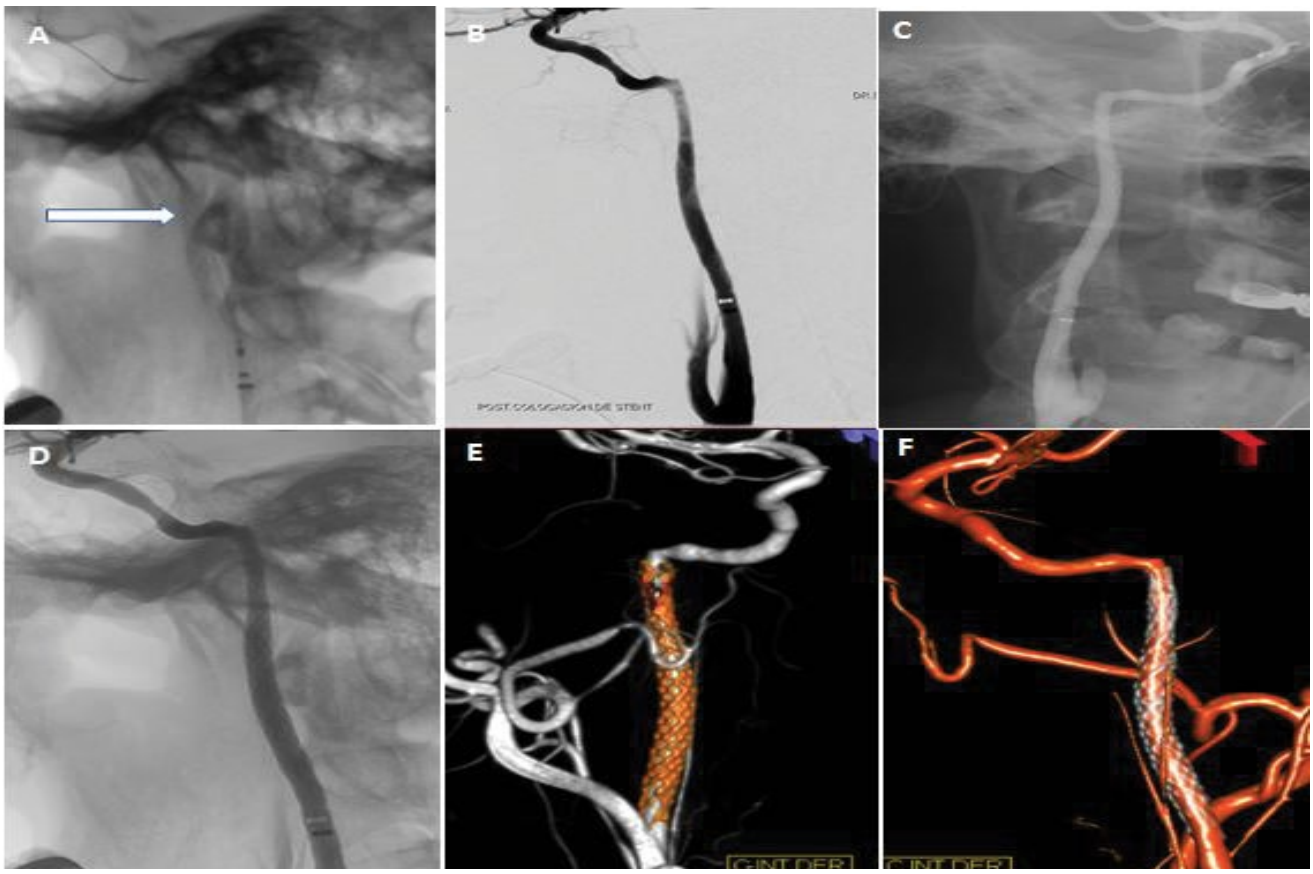

Figure 3 **(A)** Deployment of the Casper stent covering the entire area of the right internal carotid stenosis. (White arrow). **(B-D)** Immediate post-Casper stent placement lateral, oblique projections show close apposition of the stent to the wall of the artery. **(E, F)** Post-stent postero-anterior and lateral projection with 3D reconstructions' with wide patency of the stented artery.

damage to the sympathetic pathway distal to the superior cervical ganglion and is seen in 28–58% of cases [5]. Transient ischaemic attack or ischaemic stroke are seen in 67% of patients with SCAD as a consequence of artery-to-artery thromboembolism from the site of dissection [6].

Horner syndrome is classified based on the anatomic location of the lesion in the oculosympathetic pathway into first-order (central), second-order (pre-ganglionic), and third-order (post-ganglionic) types [7]. The presence or absence of its various components (ptosis, miosis, and facial anhidrosis) may help to localize the responsible lesion. Sympathetic input to the face and eye arise from the superior cervical ganglion, which usually lies at the level of the second cervical vertebra. Fibers originating from this ganglion then form the nerves of the internal and external carotid arteries. Fibers traveling with the external carotid artery supply the arrectores pilorum muscles and sweat glands of the face. Accordingly, lesions occurring along the course of the ICA distal to this division of sympathetic fibers do not produce facial anhidrosis. The internal carotid nerve travels embedded in the fibers of the carotid sheath or in the immediately surrounding fascia to the cavernous sinus. Here the fibers communicate with the cranial nerves of the cavernous sinus. Fibers to the levator palpebrae muscle travel with the superior division of the oculomotor nerve. Fibers to the dilator pupillae muscle communicate with the ophthalmic division of the trigeminal nerve and reach their destination via the nasociliary and long ciliary nerves [8].

The early diagnosis and prompt treatment of carotid artery dissection are essential to prevent cerebral infarction and the progression of neurological symptoms [9]. Treatment options for ICA dissection include anticoagulation, antiplatelet therapy, surgery, and observation [7].

Carotid artery stenting (CAS) has been established as an alternative, minimally invasive technique for the treatment of ICAD, Self-expanding stents have been developed for the treatment of carotid lesions, including open-cell and closed-cell designs. The carotid stents are the available characteristics. Several studies have reported that closed-cell stents have some obvious advantages over open-cell stents, such as improved plaque coverage, lesser chance of plaque protrusion through the interstices, and consecutively decreased risk for distal embolization. The main disadvantage of a closed-cell stent design is their inability to conform to tortuous vessels. The ideal carotid stent should cover the entire plaque, provide good vessel wall apposition to the parent vessel, and be flexible and with good radial force. The body of the stent, with its characteristic design and radial force, provides an intrinsic anti-embolic effect as the stent struts prevent the escape of the disrupted plaque material through the interstices while maintaining the caliber and integrity of the vessel wall [10]. The Casper device is the first carotid stent designed with a nitinol double layer micromesh construction for sustained embolic protection. The braided nitinol construction confers the properties of shape memory and flexibility, and allows conformation to carotid anatomy while minimizing the chance of kinking. It is characterized by a closed cell design with open cell mechanical performance. The open cell external layer produces flexibility and conformability **(Figure 4)** and the inner micromesh with its diminutive pore size allows extensive plaque coverage,

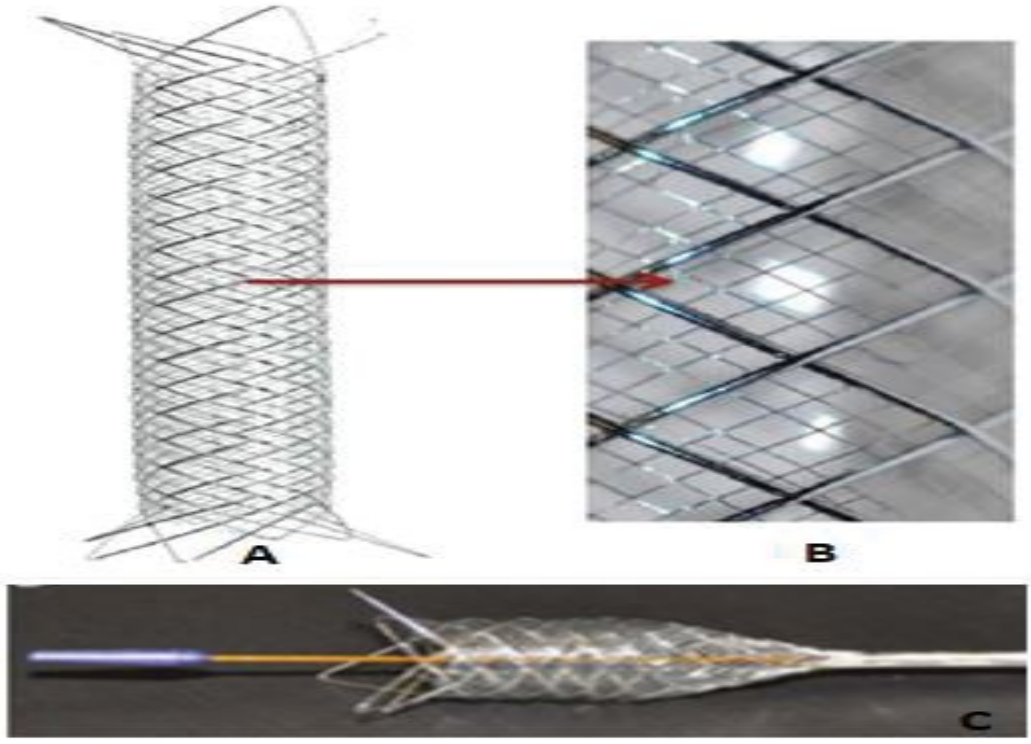

Figure 4 Casper device **(A)** Examples of a closed-cell dual-layer stent**. (B)** Magnifications show the 2 layers of the dual-layer stent. **(C)** Low profile 5.2 F rapid exchange catheter for all sizes.

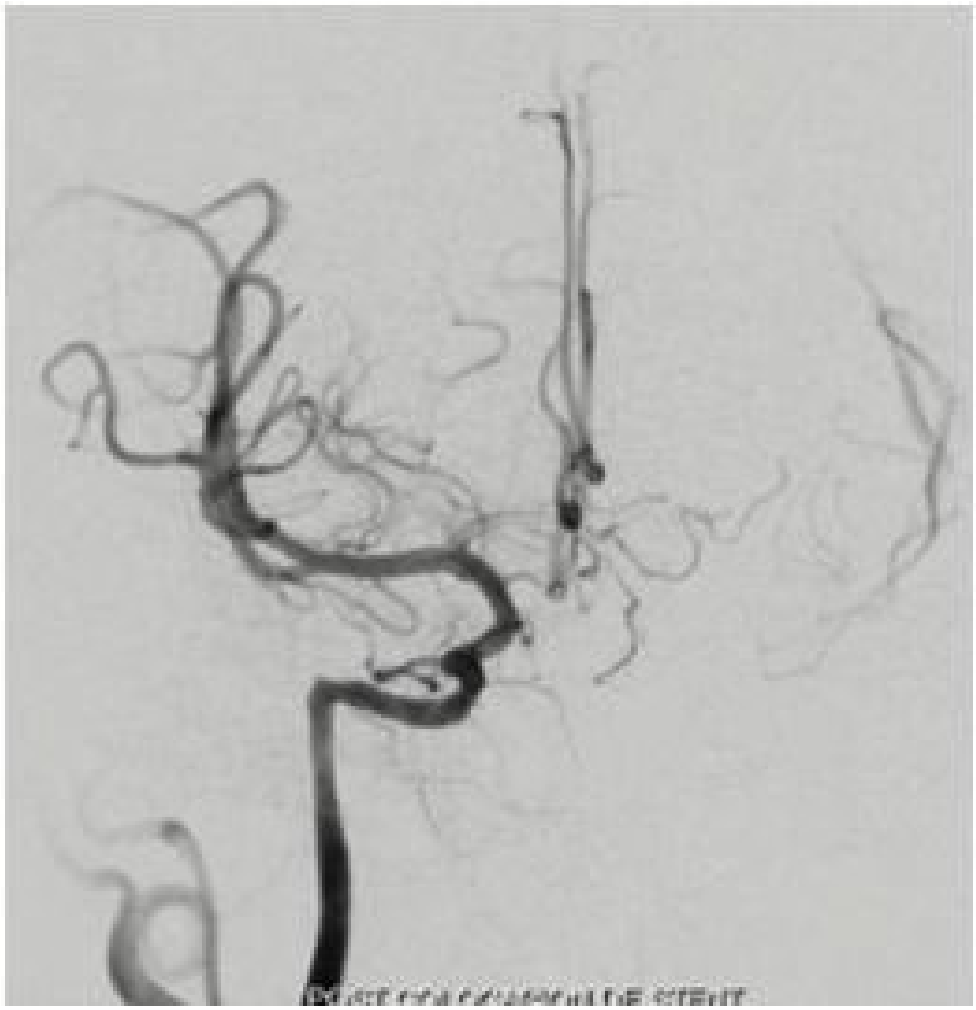

Figure 5 Adequate irrigation of the entire right cerebral hemisphere with the presence of all adjacent vessels.

minimizing the risk of plaque protrusion and embolization. This new Casper micromesh stent provides the advantage of sustained embolic protection with plaque stabilization and periprocedural stroke prevention. There were no technical complications. The low profile delivery system allowed rapid exchange for all sizes. The ability to re-sheath and reposition the stent is unique with this device and provided added comfort and confidence for the operator [11].

We must also bear in mind that Horner syndrome occurs after the placement of carotid stents in the treatment of atheromatous plaque stenosis, due to the distention of the artery walls irritating the fibers of the sympathetic system and it is also reported secondary to the formation of hematomas in the arterial wall after the distension of the artery walls by the stent meshes **(Figure 5)**.

Conclusion

Internal carotid artery dissection should be suspected in a patient with partial Horner Syndrome. Occasionally, HS can be the only manifestation in ICA dissection. Early diagnosis helps in the prompt initiation of treatment, which can prevent the devastating complications of carotid artery dissection. In this case we show an excellent performance of the Casper stent for the treatment of Internal carotid artery dissection (ICAD). The double layer micromesh stent has been designed to prevent plaque prolapse and to provide protection against emboli.

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