外伤性视神经病变-视神经病变

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外伤性视神经病变的诊治

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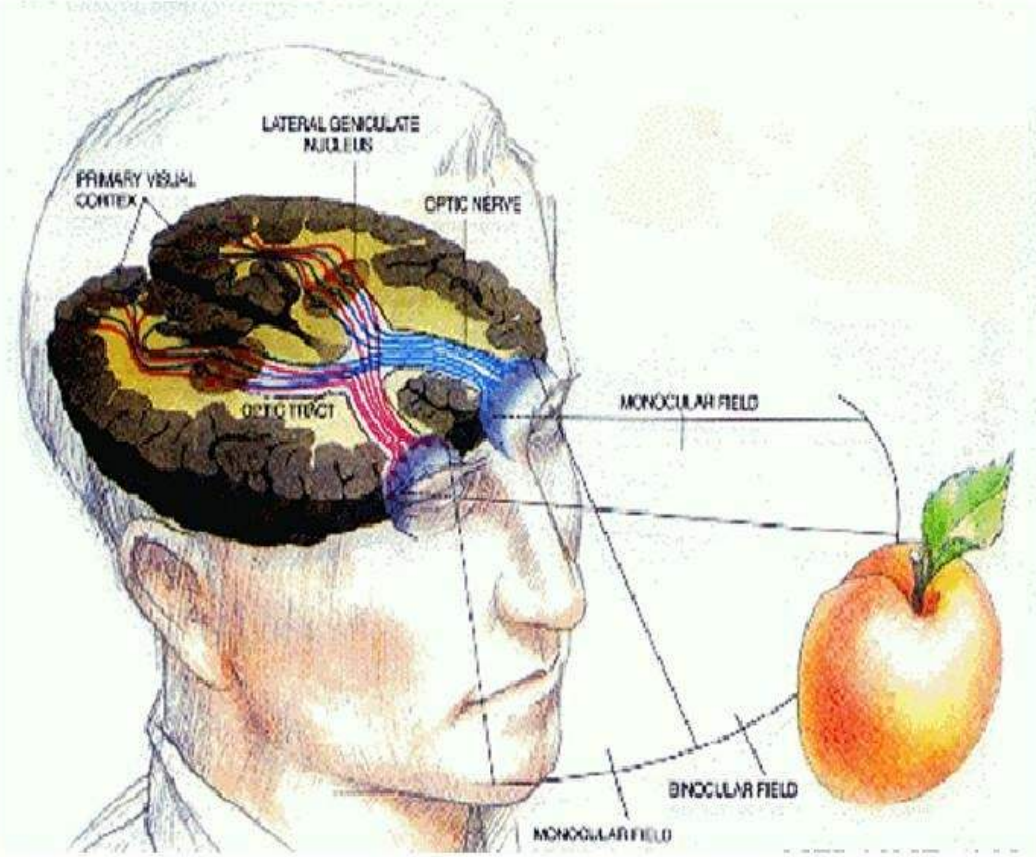
概念

外伤性视神经病变(traumatic optic neuropathy，TON)是临床常见的一类眼外伤，主要指眼眶外上方额、颞部突然遭受钝性外力作用后而导致的视神经病变



中华医学会眼科学分会神经眼科学组.我国外伤性视神经病变内镜下经鼻视神经管减压术专家共识（2016年）.中华眼科杂志.2016,52(12):889-893

发展

2000年前

Hippocrate在对脑外伤患者做记录时，即指出前额部受到撞击，可发生同侧的视力障碍。

1879年

Berlin对此病进行了较系统的描述，并称此视力障碍为视神经管骨折。人们称之为视神经间接损伤，以区别锐物刺入而造成的直接损伤。

1969年

Walsh和Hoyt进一步明确了此病的定义，称之为外伤性视神经病变(traumatic loss of vision which occurs without external or initial ophthalmolscopic evidence of injury to the eye or its nerve)，即外伤后没有外部或最初眼底镜下眼球、视神经损伤的表现，而有视力丧失。

2016年

我国外伤性视神经病变内镜下经鼻视神经管减压术专家共识

病因

车祸伤占首位(50%-65%)，其次为坠落伤和摔伤(13%-28%）
受伤部位最常见于眉弓外侧部和颞侧，其次为眶周和头颅。

刘杰，马志中，郭金凤，等. 交通伤所致外伤性视神经病变的流行病学特点[J. 眼外伤职业眼病杂志，2008，30(5)：344—346. DOI：10. 3760 / cma. j. issn. 2095—1477. 2008. 05. 003.
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2000 年北京同仁医院统计 364 例致伤原因

致伤原因	例数	百分比
交通事故	287	78.84%
坠落或跌倒	41	11.26%
其他	36	9.89%

287 例交通事故致伤分析

致伤原因	例数	百分比
汽车	45	15.68%
摩托车(两轮)	238	82.93%
自行车	4	1.39%

外伤性视神经病变诊断治疗进展 -宋维贤王怀洲，2007 - 2007年全国神经眼科学临床与基础新进展研讨会

损伤概率

视神经各段损伤概率为：
管内段71.4 %，眶内段16.7 %，球内段和颅内段共11.9 %。

Kumaran AM, Sundar G, Chye LT. Traumatic optic neuropathy: a review[J]. Craniomaxillofacial trauma & reconstruction, 2015, 8(1): 31-41

Traumatic Optic Neuropathy: A Review

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Craniomaxillofac Trauma Reconstruction 2015;8:31–41

Abstract

The aim of this article is to evaluate current literature on investigation and management of traumatic optic neuropathy (TON), propose recommendations for diagnosis and management, and explore novel future treatments. TON, though uncommon, causes substantial visual loss. Without clear guidelines, there is much ambiguity regarding its

In a study of 42 patients with TON,[8] the frequency of site of injury was: intracanalicular (71.4%) > orbital apex (16.7%) >

and if also relevant, included. A total of 2,686 articles were retrieved and 43 examined for relevance. Of these, 23 articles were included. TON is a clinical diagnosis. Visual-evoked potential is useful in diagnosis and prognosis. Computed tomography demonstrates canal fractures and concomitant injuries. Magnetic resonance images should be reserved for select and stable patients. Conservative treatment is appropriate in mild TON. Steroids are of questionable benefit and may be harmful. Surgery should be reserved for patients with radiological evidence of compression and individualized.

Keywords

- trauma
- neurop
- oculofacial trauma
- corticosteroid therapy
- optic nerve decompression
- neuroprotection and neuroregeneration

The optic nerve (ON) comprises axons of retinal ganglion cells (RCCs) and support cells. At 50 mm in length, it consists the following four segments: intraocular (1 mm), intraorbital (24 mm), intracanalicular (9 mm), and intracranial (16 mm). The ON may be injured in trauma, resulting in visual loss and this is known as traumatic optic neuropathy (TON). This occurs from either direct or indirect trauma and both primary and secondary mechanisms of damage have been proposed.[1–4]

In direct trauma, stress is applied directly to the ON and is often when orbital fracture fragments lacerate the it or when mechanical contusion/concussion.[5] The ON commonly sustains indirect trauma, where stress is transmitted through the oculofacial soft tissues and skeleton. The resultant coup-contrecoup forces damage the nerve at transitions between mobile and fixed segments. Commonly, this occurs at the junction of the intraorbital and intracanalicular segments. This results in compression and disruption of pial vessels within the canal, limiting vascular supply of the ON.[6,7]

In a study of 42 patients with TON,[8] the frequency of site of injury was: intracanalicular (71.4%) > orbital apex (16.7%) >

both (11.9%). This is supported by static loading studies which demonstrate that force applied to the superior orbital rim is transferred and concentrated on the orbital roof and optic canal.[8] The next most common site is within the anterior cranial fossa where the intracranial ON lies close to the falciform dural fold.[9]

Primary damage occurs when there is an immediate disruption (direct trauma) or shearing (indirect trauma) of RGC axons. The inflammation and vascular dysfunction that follows gives rise to secondary damage. Though the pathophysiologies of both mechanisms differ greatly, patients often have elements of both.

A 5-year British study of TON in the general population reported 121 cases.[10] Of those, 79% were male with a median age of 31 years and significantly, 21% were younger than 18 years. Common etiologies were falls (26%), motor vehicle accidents (21%), and assaults (21%). In the trauma setting, a 20-year study at the largest level 1 trauma center in Canada reported 0.4% of all trauma (injury severity score [ISS] > 12) patients had TON.[11] Of those, 76% were male with a median age of 33.5 years. Significantly, all patients with TON had head

similar results to adult studies.[12] Overall, 60% were males, common etiologies included motor vehicle accidents (62%) and sports injuries (22%). About 78% of cases were because of blunt trauma.

Iatrogenic TON is an understudied cause of postoperative blindness. It can occur during orbital surgery,[13] Le Fort I osteotomies,[14] maxillofacial fracture fixation,[15,16] and endoscopic sinus surgery.[17–19] In patients with chronic sinusitis, the ON may protrude and complicate sphenoidal sinus surgery. Among 260 patients with chronic inflammatory sinus disease, ON protrusion occurred in 28% of cases. Overall, 12% of ON indented the sinus wall and 8% of them coursed through it.[20]

Risk factors for TON include loss of consciousness and injury to the superolateral orbital region.[21] Spontaneous improvement in vision can occur and time from injury to the presentation should be documented. Concomitant head injury can leave the patient obtunded, making assessment difficult and delaying the diagnosis.[22] Decreased visual acuity (VA) is observed with 40 to 60% of patients presenting with light perception or worse.[21,23] This is associated with a relative afferent pupillary defect (RAPD), except in cases of symmetrical bilateral TON.[24] There may be impaired color and RAPD. However, in cases of posterior injuries (more common), a normal fundus is seen. In a study of 27 patients, 89% had a normal fundus.[25] ON avulsions may be visualized on funduscopy as well. Optic atrophy may develop but is only evident after 4 to 6 weeks.[26]

At present, no clear indications or contraindications for choice of imaging or treatment of TON have been proposed. In addition, treatment (corticosteroids and surgery) largely minimizes damage but in the future, neuroprotection and neuroregeneration may be possible.

Materials and Methods

A search of PubMed, MEDLINE (Medical Literature Analysis and Retrieval System Online), PROSPERO (International prospective register of systematic reviews), CENTRAL (the Cochrane library), and EMBASE (Elsevier) electronic databases for publications with content matching the term "traumatic optic neuropathy" was performed. Articles with abstracts and full text available, published in the past 10 years, written in English, and limited to human adults (older than 18 years), elected. All study designs were acceptable except case and series with fewer than 10 patients. All abstracts valuated for relevance. References of these studies valuated as well and if relevant, included. Articles on estigation, management (conservative, medical, surgi- combined), and future treatment (neuroprotection uroregeneration) of TON were included.

Results

The electronic database search yielded 2,689 articles (PROSPERO, 0; CENTRAL,7; PubMed/MEDLINE, 2,682; and Embase,0). Of these, 43 articles had available abstract and full text, were written in English, not older than 10 years, and were limited to human adults. Of these articles, nine were found to be relevant. The references of these articles were evaluated, and further 14 articles were added. Hence, a total of 23 articles were reviewed in this study. This has been summarized in ►**Fig. 1**.

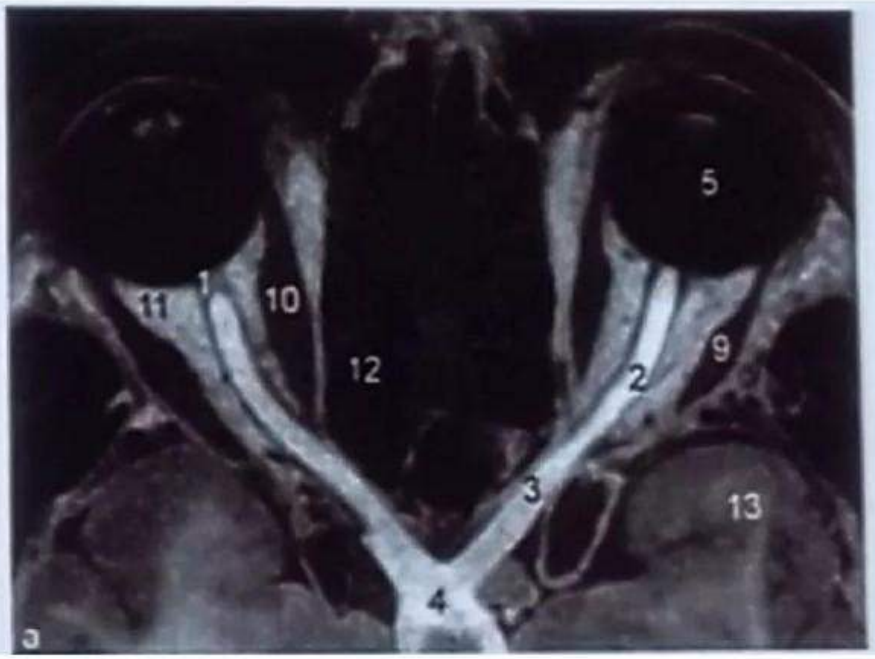
The details of these studies have been summarized in ►**Table 1**. There were zero randomized controlled clinical trials, one meta-analysis, seven prospective studies, six retrospective studies, five reviews, three case series, and one expert opinion which resulted in a mean level of evidence of 2.8. The level of evidence was determined according to the definitions stated in ►**Table 2**.

Of these 23 articles, 8 articles investigated the choice of imaging, 14 articles investigated choice of treatment, and 1 explored future treatments of TON. To supplement the review of these articles and substantiate or refute their claims, wherever appropriate, the authors have cited relevant studies

视神经解剖

视神经长约40mm 。分4段。
眶内段最长，呈S形弯曲，周围有海绵状眶内脂肪包绕，具有缓冲作用。
在视神经管内，视神经硬脑膜同周围骨壁紧密相联，因此，造成骨质变形的冲击力可以轻易的传向视神经。最容易受伤。

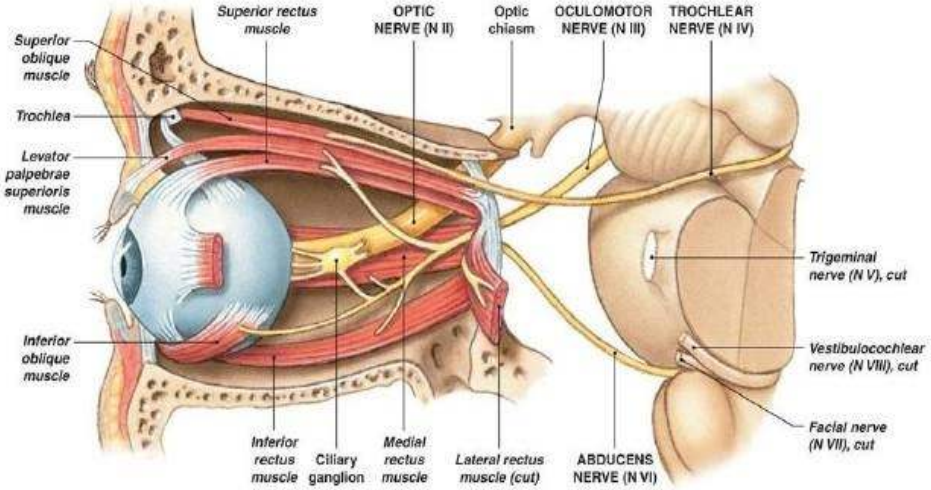
1. 球内段
2. 眶内段
3. 管内段
4. 颅内段



发病机制

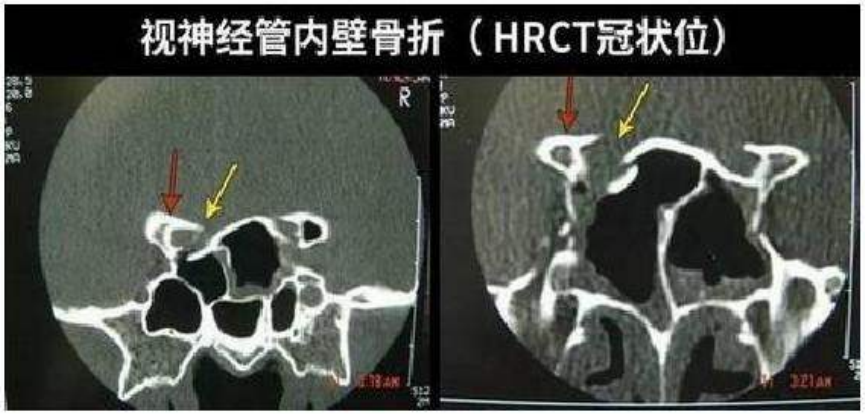
尚未完全明确。
目前认为分为原发性损伤和继发性损伤
(1)原发性损伤包括视神经断裂和视神经挫伤

(2)继发性损伤包括视神经水肿，局部血管受压或循环障碍



中华医学会眼科学分会神经眼科学组.我国外伤性视神经病变内镜下经鼻视神经管减压术专家共识（2016年）.中华眼科杂志.2016,52(12):889-893

原发性损伤是指外界钝力作用于额、颞部，因视神经管内段鞘膜与周围骨质紧贴，且视神经管空间狭小，钝力经过骨质传导而对视神经纤维造成冲击、震荡、剪切、扭曲等，致其断裂、撕裂或滋养血管破裂，甚至因神经管骨折碎片锐性切割等，导致视神经轴索中断，患者往往在受伤后即刻出现视功能严重受损。



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