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Case report

Red ear syndrome and lesser occipital nerve block

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Abstract

Introduction
Red ear syndrome (RES) is a rare condition characterized by episodes of burning pain and redness of the external ear. Its pathophysiology remains unclear, and both peripheral mechanisms, involving the upper cervical roots, and central mechanisms, involving the trigeminal autonomic reflex, have been proposed.

Case report
We report a 44-year-old man with a 3-year history of left ear pain and redness, occurring five times per week and lasting more than 60 minutes, sometimes triggered by touching the tragus. He also had migraine ipsilateral to the red ear, but the red ear attacks did not occur during the migraine attacks. There were no cranial autonomic symptoms, and neurological and otorhinolaryngological examinations, including magnetic resonance imaging, were normal. On examination, there was tenderness over the lesser occipital nerve, and its anesthetic blockade led to the complete disappearance of symptoms for ten days. He was then treated with amitriptyline 25 mg/day, with excellent response, and is currently asymptomatic. We also review the literature on the mechanisms, differential diagnosis and treatment of RES.

Conclusion
The response to the lesser occipital nerve blockade suggests that upper cervical afferents, possibly through convergence in the trigeminocervical complex, may contribute to RES in selected patients. Tenderness over the upper cervical nerves should be investigated in patients with RES, and peripheral nerve blocks deserve further study in this condition.

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Introduction

Red ear syndrome (RES) is a rare condition described by Lance in 1996 and is characterized by episodic ear redness and burning pain lasting seconds to hours, with triggers including touch, heat, or neck movements (1,2). The pathophysiological mechanisms are still unclear. The proposed theories include peripheral dysfunction of the C2-C3 roots and greater auricular nerve, as well as dysregulation in the trigeminal autonomic reflex, involving brainstem connections between the trigeminal nerve and the facial parasympathetic outflow. We present a case of RES in a 44-year-old man with a 3-year history of paroxysmal otalgia and redness of the left ear. A review of the literature highlights the association of RES with migraine, trigeminal autonomic cephalalgias (TACs), as well as potential pathophysiological mechanisms involving the trigeminal autonomic reflex (1–3).

Case report

A 44-year-old man presents with a 3-year history of severe unilateral left ear pain, occurring five times per week, lasting more than 60 minutes. During attacks, his left ear becomes erythematous (Figure 1), and he reports a burning sensation. Sometimes touching the tragus region triggers the pain. Occasionally, he has left-sided headaches, ipsilateral to the red ear, that fulfill the ICHD-3 criteria for migraine; however, the red ear attacks do not occur during the migraine attacks. In his family history, his father has migraines. There were no associated autonomic signs, such as lacrimation or ptosis. Previous treatment with gabapentin was ineffective. Neurological and otorhinolaryngological examinations, including magnetic resonance imaging, showed no abnormalities. During the examination, he felt pain on palpation of the lesser occipital nerve. He underwent an anesthetic blockade of the nerve, with disappearance of symptoms for ten days. After that, he was medicated with amitriptyline 25 mg/day with excellent response. He is currently asymptomatic.



Figure 1. During the pain attack. A) Right ear (normal) and B) Left ear (erythematous).

Discussion

RES is characterized by episodic ear redness and burning pain lasting seconds to hours, with triggers including touch, heat, or neck movements (2). Our patient's symptoms were consistent with frequent, severe, unilateral attacks, sometimes triggered by touching the tragus. The most relevant finding in this case was the tenderness over the lesser occipital nerve (LON), followed by the complete disappearance of symptoms for ten days after its anesthetic blockade.

The great auricular nerve, branch of the C2-C3 roots, and the auriculotemporal nerve, branch of the trigeminal nerve, contribute to the sensory innervation of the external ear (1,2). The LON, which originates from C2, is one of the nerves that supply the posterior part of the scalp, adjacent to the ear (4). The ear and its neighboring regions receive both cervical and trigeminal sensory inputs. The vascular supply of the ear arises from the external carotid circulation with trigeminal sensory innervation, while the vasomotor control is mediated by sympathetic vasoconstrictor tone (2). The sensation of ear obstruction was hypothesized to result from swelling of the external auditory canal due to vasodilation mediated by the trigeminal autonomic reflex (3). However, this was not a complaint in the present case.

The proposed pathophysiological mechanisms of RES can be divided into peripheral theories and central theories. Peripheral theories attribute RES to dysfunction of the upper cervical nerves, mainly the C3 root, whereas central theories propose a dysregulation of the brainstem trigemino-autonomic circuits (2). The convergence of nociceptive information processing at the level of the trigeminal nucleus caudalis and the dorsal horns of the upper cervical spine is a possible anatomical basis linking the peripheral and central mechanisms of RES (2,5). It may also explain why symptoms often extend beyond the territory of a single nerve (5).

In healthy volunteers, Hoffmann et al.(6) showed that the greater occipital nerve blockade with lidocaine reduced the trigeminal nociceptive activation on functional magnetic resonance imaging, suggesting that the interaction between occipital and trigeminal inputs occurs at the C2 level of the trigeminocervical complex. Although the LON was not studied, a similar interaction may explain the response to the blockade in our patient.

Previous reports of nerve blockades in RES showed variable results. Selekler et al.(7) described a patient with tenderness over the great auricular nerve during the attacks, who remained free of symptoms for eight weeks after the blockade of this nerve. Lance reported pain relief lasting only a few hours after C2-C3 root blockades (1). Tenderness over the occipital nerve has also been described in a patient with RES, with pain behind the ear (7). In our patient, the LON tenderness and the response to its blockade suggest that upper cervical afferents contributed to the attacks. A response to a nerve blockade alone does not prove the origin of the symptoms, but it indicates that cervical afferent pathways may be a therapeutic target in selected patients with RES. Lance proposed that, in cases associated with upper cervical disorders, an antidromic discharge along the C3 root could release vasodilator peptides, causing pain and vasodilation of the ear (1). The release of vasodilator substances, such as substance P, CGRP and nitric oxide, has been attributed to trigeminovascular activation in migraine-related RES, and the overlap between neurons of the trigeminal nucleus caudalis and neurons located within the C1 and C2 dorsal horns may explain pain that extends beyond the trigeminal domain (8). However, the pathophysiology of RES remains unclear (2), and in our case this mechanism is hypothetical, since CGRP was not investigated and the response to the blockade does not confirm it.

Primary RES occurs more often in younger patients and is frequently associated with migraine, whereas secondary RES occurs more often in older patients, in association with upper cervical spine disorders, temporomandibular joint dysfunction or TACs (9,10). In a series of eight migraineurs, mostly children, red ear episodes occurred in close temporal relationship with migraine attacks, but also in isolation (8). The association between migraine and RES can be a sign of hyper parasympathetic activation via trigeminal-autonomic reflex during migraine (10). In migraineurs, red ear episodes occurring outside migraine attacks have been proposed as "acephalgic" migraine attacks, and the ipsilaterality between the red ear and the headache supports a relationship between both conditions (10). Our patient has migraine fulfilling the ICHD-3 criteria and a paternal history of migraine, which suggests a migrainous susceptibility. However, attacks triggered by local stimuli and a later onset have been more associated with upper cervical pathology than with migraine (11), and the LON tenderness and the response to its blockade suggest that a cervical peripheral component also contributed to the attacks.

The differential diagnosis of RES includes TACs, occipital neuralgia, secondary RES and other causes of ear erythema, such as trauma, connective tissue or autoimmune disorders and erythromelalgia (Table 1) (12). Paroxysmal hemicrania may present as otalgia with ear erythema (3). In our patient, the frequency of five attacks per week and the duration of more than 60 minutes do not fit paroxysmal hemicrania or SUNCT/SUNA, and the absence of cranial autonomic symptoms or restlessness, as well as the location of the pain in the ear, do not fit cluster headache (4). Occipital neuralgia may involve the LON and is commonly associated with tenderness over the nerve and temporary relief after anesthetic blockade (4). However, its pain is shooting or stabbing, lasts from seconds to minutes and is located in the posterior scalp (4), unlike the burning ear pain with erythema lasting more than 60 minutes in our patient. Secondary RES, related mainly to upper cervical spine disorders and temporomandibular joint dysfunction, should be investigated with brain and cervical spine MRI (2).

Table 1. Differential diagnosis of red ear syndrome

Disorder	Attack duration	Auricular erythema	Cranial autonomic symptoms	Distinguishing features
Red ear syndrome	Seconds to hours	Characteristic	Usually absent	Burning ear pain with erythema; may be triggered by touch, heat or neck movement
Paroxysmal hemicrania	2–30 minutes	May occur	Present	More than 5 attacks per day; absolute response to indomethacin
Cluster headache	15–180 minutes	May occur	Present, and/ or restlessness	Severe orbital, supraorbital or temporal pain; from one attack every other day to eight per day
SUNCT/SUNA	1–600 seconds	Rarely reported	Present	Stabbing attacks, at least once a day
Occipital neuralgia	Seconds to minutes	Not part of the criteria	Not part of the criteria	Shooting or stabbing pain in the posterior scalp; tenderness over the nerve; temporary relief with anesthetic blockade
Secondary RES	Variable	Characteristic	Variable	Upper cervical spine disorders or temporomandibular joint dysfunction; brain and cervical spine MRI

SUNCT/SUNA: short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing / with cranial autonomic symptoms; MRI: magnetic resonance imaging. Based on references (2–4).

The treatment of RES remains empirical, and many patients are refractory (2,9). The treatment options include NSAIDs (e.g., ibuprofen), gabapentin, tricyclic antidepressants, such as amitriptyline, calcium channel blockers and beta-blockers (2), greater auricular nerve blocks (6), and treatments directed to the associated primary headache (5). Amitriptyline 25 mg at night reduced the intensity of symptoms by half in one of Lance's patients (1). Our patient's response to amitriptyline suggests that it may be a useful therapeutic option in RES, particularly when migraine is associated, since migraine preventive treatments have shown benefit mainly in migraine-related attacks (2). However, this response is not specific to RES.

Thus, in our patient, both cervical peripheral inputs and central mechanisms related to migraine may have contributed to the attacks. The response to the LON blockade does not establish a causal relationship, but suggests that upper cervical afferents should be considered in the evaluation of patients with RES, and that peripheral nerve blocks deserve further study in this condition.

Conclusion

RES is a heterogeneous syndrome with primary (migraine-related) and secondary (structural or TAC-associated) forms (9). Clinicians should consider RES in patients with recurrent ear pain and redness, particularly if triggers are present, and should look for tenderness over the upper cervical nerves, since peripheral nerve blockade may be useful in selected cases. Further research is needed to clarify the role of upper cervical afferents and of the trigeminovascular pathways in RES (2,13).

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