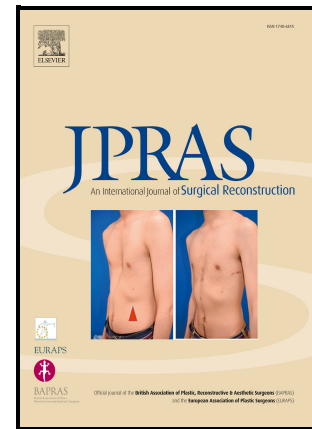


Preoperative ultrasound evaluation for decompressive surgery for occipital neuralgia: preliminary results of a case-control study
SHORT RUNNING HEAD: Ultrasound evaluation for occipital neuralgia

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PII: S1748-6815(25)00356-0

DOI: <https://doi.org/10.1016/j.bjps.2025.05.040>

Reference: PRAS9660

To appear in: *Journal of Plastic, Reconstructive & Aesthetic Surgery*

Received date: 9 January 2025

Accepted date: 18 May 2025

Please cite this article as: Ilaria Baldelli, Federico Zaottini, Riccardo Picasso, Federico Pistoia, Hamedani Mehrnaz, Giorgio Raposio, Michele Piccioli, Carlo Martinoli and Edoardo Raposio, Preoperative ultrasound evaluation for decompressive surgery for occipital neuralgia: preliminary results of a case-control study
SHORT RUNNING HEAD: Ultrasound evaluation for occipital neuralgia, *Journal of Plastic, Reconstructive & Aesthetic Surgery*, (2025)
doi:<https://doi.org/10.1016/j.bjps.2025.05.040>

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TITLE:

Preoperative ultrasound evaluation for decompressive surgery for occipital neuralgia: preliminary results of a case-control study.

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SHORT RUNNING HEAD

Ultrasound evaluation for occipital neuralgia

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FINANCIAL DISCLOSURE STATEMENT

The authors have nothing to disclose; this includes any actual or potential conflict of interest including any financial, personal or other relationships with other people or organizations related to the submitted work.

SUMMARY

Although no consensus yet exists on the pathogenesis of occipital neuralgia, alterations in the soft tissues surrounding the occipital nerves have been recently demonstrated. To investigate potential differences of these tissues between patients and healthy subjects, we decided to use ultrasound in the study of peripheral neurological pathology. Twenty patients who underwent occipital nerve decompression surgery were included in this retrospective observational study. A group of twenty-two healthy volunteers was considered as a control group for ultrasound evaluation of the occipital region. Ultrasound was performed to detect morphological alterations and to measure the cross-sectional areas of the greater occipital nerve and the occipital artery in both groups. Pain during the preoperative examination was significantly higher in patients. Ultrasound-detected pathological alterations of the greater occipital nerve were observed in 16 out of 20 patients and 3 out of 22 healthy subjects. Patients suffering from occipital neuralgia could also present perineural fibrosis and morphological alterations of the occipital artery, which were generally not detected in healthy subjects.

During surgery, the appearance of the soft tissues was documented, and the crossing point between nerve and artery was checked. In all subjects evaluated, the greater occipital nerve was bilaterally identified throughout its course. Moreover, in 17 patients out of 20, the preoperative location of pain in the occipital region matched with the contact point the greater occipital nerve with the occipital artery observed during surgery. . Ultrasound appears to be a useful tool for the preoperative evaluation of the trigger points in the treatment of the occipital neuralgia.

Keywords: Migraine surgery, greater occipital nerve, neuralgia, ultrasound.

Introduction

Occipital neuralgia (ON) is a widely described disabling neuropathy^{1,2} characterized by recurrent moderate to severe jabbing pain localized in the occipital region, with possible radiation anteriorly to the face and posteriorly to the neck. Although the origin of occipital neuralgia has been thought to derive from nociceptors in the neck-occipital area since 1926, the concept of cervicogenic headache remains controversial^{3,4}. Despite its prevalence in the population, occipital neuralgia is still underdiagnosed, and no consensus exists among specialists regarding its pathogenesis. This leads to different treatment options without a defined, shared treatment algorithm.

Typically, initial treatment involves non-steroidal anti-inflammatory drugs, acetaminophen, tricyclic antidepressants, opioids, serotonin-norepinephrine reuptake inhibitors, anticonvulsants, or ergo-derivatives, which may help alleviate symptoms but are not always effective and often have only a transient effect. Infliximab may be cautiously beneficial in neglected patients with cervicogenic headache⁵.

More invasive treatments, such as local anesthetics, corticosteroids, botulinum toxin injections, radiofrequency treatments, or implantable nerve stimulators, have demonstrated good pain relief but with limited duration and a certain number of complications. Moreover, long-term results are often unknown^{4,6,7}.

Surgical therapy for occipital neuralgia was introduced by Martin and Fagan in 1964⁸, consisting of the sectioning of the occipital nerves (greater, lesser, third) to achieve relief from occipital pain, accepting a certain degree of sensation loss in the scalp.

After the in-depth studies of Guyuron et al.⁹, surgical approaches shifted from denervation to nerve-decompression surgeries¹⁰⁻¹³. The obliquus capitis inferior muscle (OCinf), trapezius muscle (TM), semispinalis muscle (SM), and the occipital artery (OA) began to be sectioned due to their potential role in greater occipital nerve entrapment, with surgical benefits reaching 80%. A combination of

these compression conditions may contribute to the symptoms, and incomplete treatment may therefore not lead to satisfactory results^{14,15}. There are six potential compression points that have been carefully elucidated for the GON¹⁶. However, common anomalies of the GON occur. In particular, it often branches caudal to the nuchal line, unexpectedly necessitating an extension of the dissection or making it particularly challenging¹⁷.

Recently, soft tissues surrounding the occipital nerves have been evaluated in detail as potentially involved in nerve compression. For example, the trapezius fascia of patients who underwent occipital decompression surgery was found to be highly thickened and fibrotic¹⁸. Recent studies also showed several anomalies of the OA^{19,20}.

Although it is proven that soft tissue aberrations exist at the occipital site in patients undergoing nerve decompression surgery for occipital neuralgia, it is difficult to demonstrate their absence in tissues taken from healthy subjects who do not require any type of surgery in the occipital region. A growing body of literature supports ultrasound evaluation to help diagnose and manage common neurological diseases²¹. This study aims to investigate sonographic differences in the occipital region between healthy subjects and patients undergoing nerve decompressive surgery and then compare the observed data with those collected intraoperatively in affected subjects.

Patients and Methods

Patients of both sexes who underwent occipital nerve decompression surgery between September 2021 and May 2022 were included in this retrospective observational study. The inclusion criteria were as follows: a neurological diagnosis of occipital neuralgia (tension headache, chronic daily headache, or migraine) according to the criteria established by the ICHD-3, with 15 or more episodes per month in the occipital region, in patients suffering from side effects of drug treatments or who did not benefit from any medication. Patients were required to be aged ≥ 18 years, in good general health, and able to identify the exact location of the sore point where the headache usually begins with the tip of a finger. The specific involvement of the greater occipital nerve was evaluated by the

semiological maneuver described in a previous work of our group²². Exclusion criteria included previous surgeries in the neck-occipital region or a diagnosis of cluster headache, episodic tension-type headache, or secondary headache. Healthy volunteers, i.e., those not suffering from recurring headaches, were considered as a control group for ultrasound evaluation of the occipital region. Patients' demographic data (age, sex, BMI) and clinical data (headache symptoms and duration, comorbidities) were collected. All participants received a detailed explanation of the study design, and written informed consent was obtained from all respondents in accordance with the guidelines provided in the current version of the Declaration of Helsinki²³. The authors adhered to the STROBE guidelines for case-control studies.

Anatomy

The greater occipital nerve (GON) is a sensory nerve that arises from the dorsal ramus of the C2 spinal nerve and crosses the suboccipital triangle, which is formed by the rectus capitis posterior major, the superior oblique muscle, and the inferior oblique muscle. The nerve then ascends to the fascia between the inferior oblique muscle (IOM) and the semispinalis capitis (SM). After piercing the SM, it continues upward on the deep side of the trapezius muscle (TM). The GON becomes superficial just below the superior nuchal line as it passes through the aponeurotic sling formed by the insertions of the TM and the sternocleidomastoid muscle. In this area, it runs close to the ipsilateral occipital artery (OA). The GON innervates the skin of the scalp, the occipital region, and the galea aponeurotica²⁴.

Ultrasound Assessment

Ultrasonography was performed on all subjects in the study using an Aplio I800 Canon (i800 platform, Canon Medical Systems, Otawara, Japan) ultrasound system with a linear matrix probe operating at a frequency of 18-5 MHz. The ultrasound examination was conducted bilaterally in the control group and either mono- or bilaterally in the patient group, depending on the reported

symptoms. The control group was independently evaluated by two operators to assess the degree of operator-dependent variability in the results.

The GON was scanned along its entire course, from the suboccipital triangle to its perforation of the trapezius fascia and entry into the subcutaneous tissue. The distal subcutaneous portion of the occipital artery (OA), where it intersects with the divisional branches of the GON, was also examined. The study aimed to identify morphological alterations of the GON (such as focal enlargement, distortions, changes in nerve and perineural tissue echogenicity) and the OA (such as caliber irregularity, wall thickening, thrombosis) in both patients and the control group. In addition, a quantitative analysis of the GON and OA was performed. The cross-sectional area (CSA) of the GON was measured at two key points: superficial to the inferior oblique muscle (OCinf) (Figure 1) and between the aponeurosis of the trapezius muscle (TM) (Figure 2) and the semispinalis capitis (SM), to assess nerve swelling because of compression within the semispinalis or at the piercing point of the TM aponeurosis²⁵. The caliber of the OA was measured at the crossing point between the artery and the subcutaneous distal divisional branches of the GON (Figure 3), with the aim of evaluating whether arterial ectasia could be associated with the painful syndrome in patients suffering from migraines and tension headaches, because of a vascular-nerve conflict. During the ultrasound assessment, a skin marker was placed over the crossing point between the GON and the OA to verify the consistency of the ultrasound findings during subsequent surgery. Both patients and healthy controls were asked to rate any painful symptoms triggered by the ultrasound probe pressure on the occipital area, using a scale from 0 to 10.

Decompressive Surgery

Surgical decompression of the greater occipital nerves in the nuchal region was performed as a one-day surgery procedure under local anesthesia with deep sedation. Without requiring a haircut, a horizontal incision of a few centimeters was made at the upper nuchal line. A blunt dissection was then performed to identify the GON (Fig. 4). Arterial vessels near the nerves (Fig. 5), which often

intertwine with them, were meticulously coagulated to ensure accurate neurolysis. The senior surgeon completed an intraoperative anatomy form for each patient, documenting the appearance of the tissues encountered, including the presence or absence of fibrotic fascia and muscle, macroscopic nerve changes such as flattening or edema, and interactions between the OA and the GON, as well as any irregularities of the occipital vessels. The procedure was completed with a continuous nylon overlock suture.

Statistical Analysis

Statistical analysis was performed using SPSS for Windows, version 22. Descriptive statistics, including means, standard deviations, absolute numbers, and frequencies (%), were used to describe the variables. Kolmogorov-Smirnov tests were conducted to assess the normal distribution of the data. If the data were normally distributed, an independent samples t-test was used to compare the quantitative data between the experimental group and the healthy control. If the data were not normally distributed, the Mann-Whitney non-parametric test was applied. A p -value of < 0.05 was considered statistically significant.

For the healthy control group, interrater reliability was analyzed by calculating Cronbach's alpha (α), with an alpha value above 0.7 indicating good consistency. The level of agreement was assessed using intra-class correlation (ICC) and an inter-item correlation matrix with a 95% confidence interval (95% CI). The agreement between pairs of raters was categorized using the proposed cut-off values²⁶.

The rate of pathological GON alterations in patients compared to healthy subjects was calculated by chi-square test.

Results

Twenty patients (13 females, 7 males) and 22 volunteers (13 females, 9 males) were enrolled in this retrospective observational study. In all subjects, the GON was bilaterally identified throughout its course. The patients underwent nerve decompression surgery of the occipital region, predominantly

on the right side, performed by the same surgeon. Demographic and clinical data are presented in Tables 1 and 2.

For the measurements taken by the two observers, the Cronbach's Alpha value for the six dimensions was 0.701 (ICC Average Measures = 0.701, 95% CI 0.454 to 0.859), indicating acceptable consistency. The inter-item correlation matrix demonstrated correlations between pairs of variables, with a strong correlation observed for the cross-sectional area of the GON ($r = 0.928$) (Table 3).

No statistically significant differences were found between healthy subjects and those suffering from headaches regarding nerve diameter. However, for the OA diameter, a statistically significant difference was found only on the right side (Table 2). The laterality of the disease (1 on the left, 5 on the right, and 14 bilateral) may influence the interpretation of these measurements. When considering only bilateral cases, no significant difference in OA diameter (mm) was found for either the right or left side ($p = 0.133$ for the right and $p = 0.255$ for the left) compared to healthy controls.

During the ultrasound examination, the patient group reported an average pain score of 5.1 ± 2.9 on the right side and 2.06 ± 2.5 on the left (on a scale from 1 to 10), compared to 0.1 ± 0.4 on both sides in the healthy subjects ($p = 0.005$ and $p = 0.001$, respectively). GON pathological alterations were observed in 16 patients and 3 healthy subjects. Ultrasound revealed GON thickening as it passed through the TM fascia (Figure 6) in more than half of the patients. Perineural fibrosis along the subcutaneous branches of the GON (Figure 7) and irregular OA caliber with multifocal strictures and ectasia were less frequent. The nerve appeared rarely enlarged proximally to the SM and more hypoechogenic compared to the contralateral, unaffected side. Multiple alterations involving both the nerve and arteries were identified in 5 patients. In the control group, 3 out of 22 cases showed some alterations: in particular, 2 people had nerve focal thickening while piercing the TM aponeurosis, and 1 exhibited a tortuous and irregular caliber OA. The rate of pathological GON alterations in patients compared to healthy subjects was found to be statistically significant (chi-square statistic with Yates correction = 16.0418; p -value < 0.05).

There was a great correspondence (16 out of 20) between surgical and ultrasound findings in the patient group. The most frequent alterations included perineural fibrosis, OA anomalies (ectasia, multifocal narrowing, wall thickening), and GON focal swelling. Detailed results are shown in Table 1. Additionally, a high match rate (17 out of 20) was found between the preoperative location of pain in the occipital region as indicated by the patient and the contact point of the GON with the OA observed during surgery (Figures 8, 9, 10).

Discussion

This study compared ultrasound assessments of the occipital region in subjects with occipital neuralgia with intraoperative findings observed during subsequent nerve decompression surgery. While many anatomical studies on cadavers and in vivo patients undergoing nerve decompression have detailed the course of the GON through vertebral joints, muscles, aponeuroses, and their relations with occipital vessels²⁷⁻²⁹, the evaluation of soft tissues surrounding the occipital nerves has been less frequent. Only a few studies have detailed the ultrasound pathological findings of the occipital nerve in occipital neuralgia, primarily focusing on detecting nerve enlargement by measuring it superficial to the OCinf and proximal to the SM³⁰⁻³².

In our study, despite some patients showing differences between the two examined sides and a nerve diameter > 4mm², as reported in previous clinical studies³¹, the mean CSA values measured at two points did not differ significantly from the control group. This discrepancy may be partly due to the long duration of symptoms (mean duration = 25.7 years) and high age (mean age = 46.8 ± 14.6 yo; range = 19 – 70 yo). in our patients. Long-standing nerve compression and irritation, **in fact**, may lead to axonal loss, increased intraneural fibrotic tissue, and reduced edema, which together could cause a pseudo-normalization of nerve CSA. The main finding in our cohort was focal thickening of the distal GON at the TM fascia crossing point, where nerve measurement was hindered by perineural fibrosis obscuring the nerve margins. **Therefore, reduced CSA values in our patients**

agree with the results present in the literature on the inverse correlation between age and CSA values of peripheral nerves in extremities^{33,34}.

Despite the unclear pathogenic mechanism, our study demonstrates that soft tissues surrounding the occipital nerves in patients with occipital neuralgia show macroscopic alterations absent in healthy patients. Both ultrasound and intraoperative assessments revealed perineural fibrosis, particularly in the final course of the GON, where it becomes superficial, passing through the TM fascia and the area surrounding the GON-OA contact point.

In our study, thanks to the use of a matrix probe with a higher resolution than those used in previous works³⁵, we were able to measure the GON between the fascia of the trapezius and the SM of the head. We included this site because in the case of compression of the GON at the point where it perforates the fascia or at the point where it crosses the OA, the nerve widens proximally. However, the distal fascicles of the GON can also be visualized above the OA and, occasionally, signs of fibrosis can be seen.

The results indicate a good level of consistency between the two observers in their quantitative measurements across six dimensions. **The agreement between observers on the measurement of nerve size was better for the proximal measurement point, at the level of the obliquus inferior capitis, because the nerve is larger, making a systematic measurement error less likely. In the distal portion, the nerve appeared flat, without the typical honey-comb appearance. However, using the indentation on the surface of the capitis semispinalis as a reference point, where the GON crosses the muscle from deep to superficial, increases confidence in the identification and measurement of this nerve.**

However, results also highlight the challenges in accurately assessing the OA in patients, particularly given the complex morphological changes involving the tunica intima and media of the OA¹⁹. Although the mean artery diameter may not significantly differ from controls, multifocal enlargements, strictures, and a winding course of the artery complicate the interpretation. The poor inter-observer agreement underscores the difficulty in obtaining consistent measurements due to

variations in ultrasound probe pressure, which can limit the reliability of subcutaneous tissue measurements. Additionally, the analysis did not account for the laterality of the disease, which could affect the interpretation of the results.

Interestingly, the pain elicited during the ultrasound examination, significantly present in the patient group, corresponded intraoperatively with the intersection point between the GON and OA. This is in line with our previous work about a semiological maneuver we described as an aid in the preoperative selection of patients with ON. A dense fibrous tissue incorporating the occipital nerve and/or the presence of a close to the nerve altered occipital artery were, in fact, common findings³⁶. Although the role of intra- or extracranial vascularization in headache pain remains unclear, the detected macroscopic vascular remodeling, confirmed by optical and electron microscopy, may be an important indicator for future research.

Despite the existence of successful sonographic visualization of the GON³⁷, this is, to our knowledge, the first study to compare ultrasound and intraoperative findings. We demonstrate an 80% match rate between them, confirming that ultrasound is a valuable tool for preoperative assessment in the clinical evaluation of occipital neuralgia. **The discrepancy detected may stem from the variable appearance of fibrosis and fibrous bands on ultrasound. Fibrotic tissue becomes clearly visible when it distorts the subcutaneous tissue architecture, causing attenuation of the ultrasound beam. However, in some patients, the fibrous bands may be too small or thin to be detected, even with high-resolution probes. Additionally, prolonged compression can cause nerve axon degeneration, leading to a reduction in the nerve's cross-sectional size, which may obscure a key indicator of nerve pathology — the edematous swelling of the nerve.**

Conclusion

Our study confirms the presence of ultrasound-detected pathological GON alterations, perineural fibrosis and morphological alterations of the OA (findings confirmed intraoperatively) in patients suffering from ON, which were generally not detected in healthy subjects. Furthermore, ultrasound

appears to be able to confirm the correspondence between pain and the point of nerve/artery intersection, which can therefore be confirmed as a trigger site in the preoperative phase.

This data highlights once again the importance of ultrasound as a support tool in the diagnosis and preoperative planning of neurological diseases, guiding the surgeon in selecting the correct surgical target for the treatment of the ON.

Ethical approval

Not required.

Conflict of interest and funding statements.

None to declare.

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Figure Legend



Figure 1. 1a - The diagram shows the relevant anatomy of the greater occipital nerve (yellow structure) from the suboccipital triangle to the subcutaneous divisional branches on the side of the occipital tuberosity. The suboccipital triangle is bounded by the Obliquus capitis inferior muscle (OCinf), the Obliquus capitis superior muscle (OCsup) and the rectus capitis posterior major muscle (RCP). The greater occipital nerve arises from the dorsal branch of the C2 spinal nerve. It enters the suboccipital triangle passing between the OCinf and the semispinalis capitis muscle (S, not fully represented in the diagram). More cranially, the nerve pierces S, and enters the cleavage plane

between S and the aponeurosis of the trapezius muscle (visualized in transparency). At the level of the superior nuchal line (dashed, curved line) it perforates the aponeurosis of the trapezius and releases some terminal branches into the subcutaneous tissue. The latter cross the divisional branches of the occipital artery (OA). The black rectangle indicates the position of the ultrasound probe with which image B is obtained. 1b - The ultrasound image shows measurement of the diameters of the greater occipital nerve (demarcated by four calipers on the image) in between the obliquus capitis inferior muscle (OCinf) and the semispinalis muscle (S). At this level the nerve is visualized in short axis and appears oval, differently from Fig.2. This measurement site was chosen to assess whether the nerve changes in size in patients suffering from neuralgia as a result of compression in the semiplinalis muscle or more distally.

O= occipital bone; M= mastoid process of the temporal bone; RCPm = rectus capitis posterior minor muscle Spl= splenius capitis; white arrowheads= trapezius muscle.

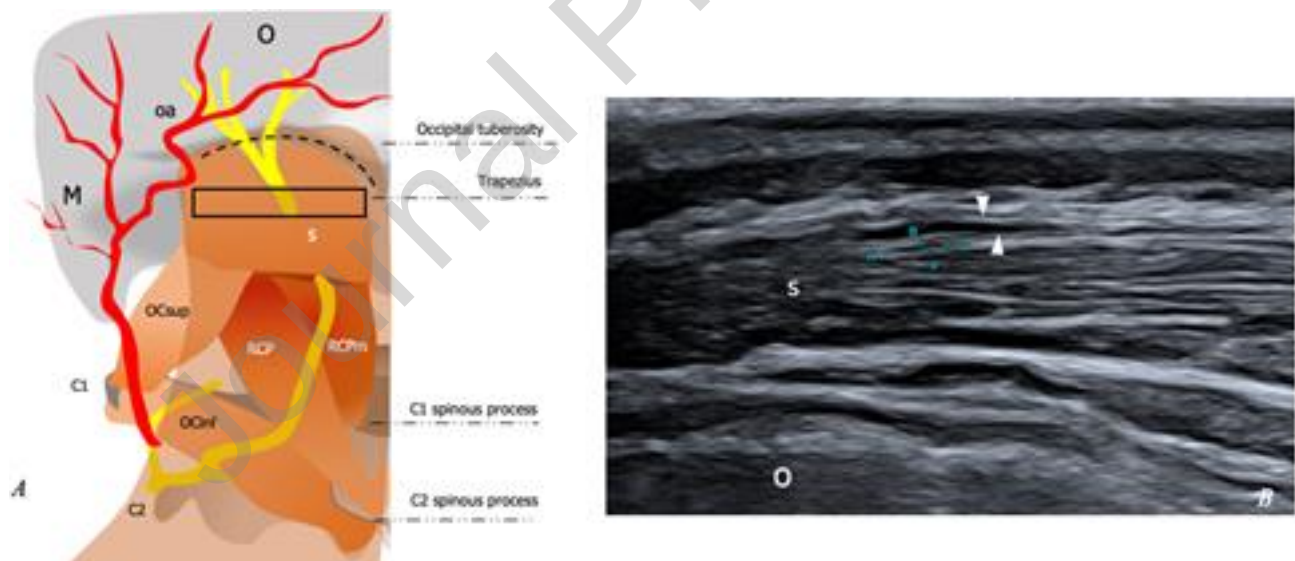


Figure 2. 2a - See the diagram legenda in figure 1a. 2b - The ultrasound image shows measurement of the diameters of the greater occipital nerve (demarcated by four calipers on the image) in between the semispinalis muscle (S) and the aponeurosis of the trapezius muscle (white arrowheads). At this level the nerve is visualized in short axis and appears flattened. This measurement site was chosen to assess whether the nerve changes in size in patients suffering from neuralgia as a result of compression at the point where it crosses the aponeurosis of the trapezius.

O= occipital bone

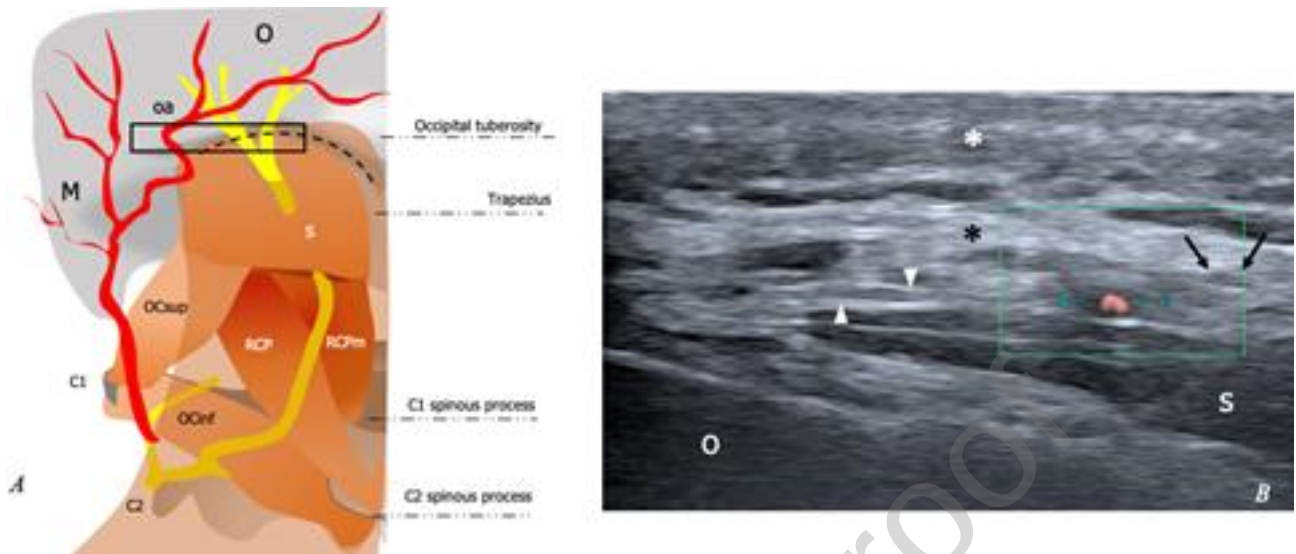


Figure 3. 3a - See the diagram legenda in figure 1a. 3b - The ultrasound image shows the measurement of the occipital artery (red dot within the Power Doppler box) over the aponeurosis of the trapezius (white arrowheads), where it is crossed superficially by a branch of the greater occipital nerve. The distance measured appears greater than the flow signal within the artery, as the vessel wall is included. Note the bi-layered ecotexture of the subcutaneous tissue of the posterior scalp: the superficial layer (white asterisk) is more hypoechoic and homogeneous than the deep layer (black asterisk), the latter being richer in connective tissue.

O= occipital bone; S= semispinalis muscle



Figure 4. Blunt dissection of the right GON (yellow arrow).

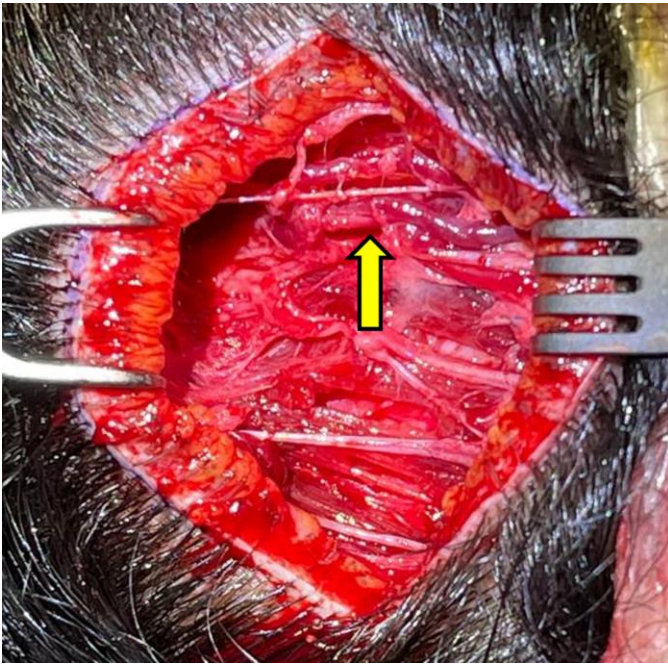


Figure 5. Right OA (yellow star).

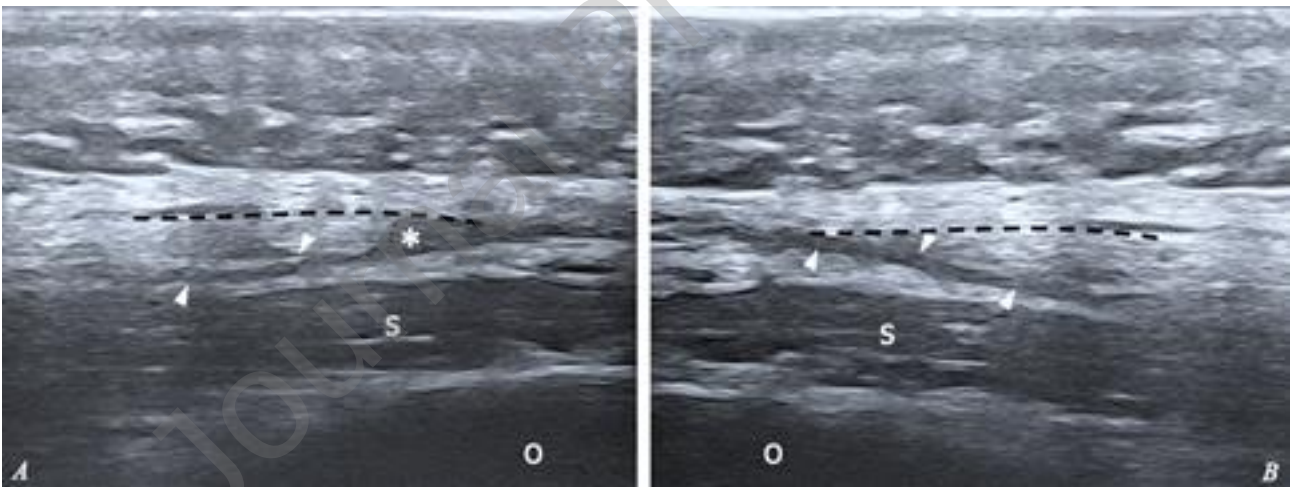


Figure 6. The two ultrasound images show the long-axis view of the greater occipital nerve (white arrowheads) in the area where it crosses the aponeurosis of the trapezius (dashed black line), on the symptomatic side (6a) and on the contralateral side (6b). Note the fusiform bulge (white asterisk) along the course of the greater occipital nerve, consistent with a small neuroma.

S= Semispinalis muscle; O= Occipital bone

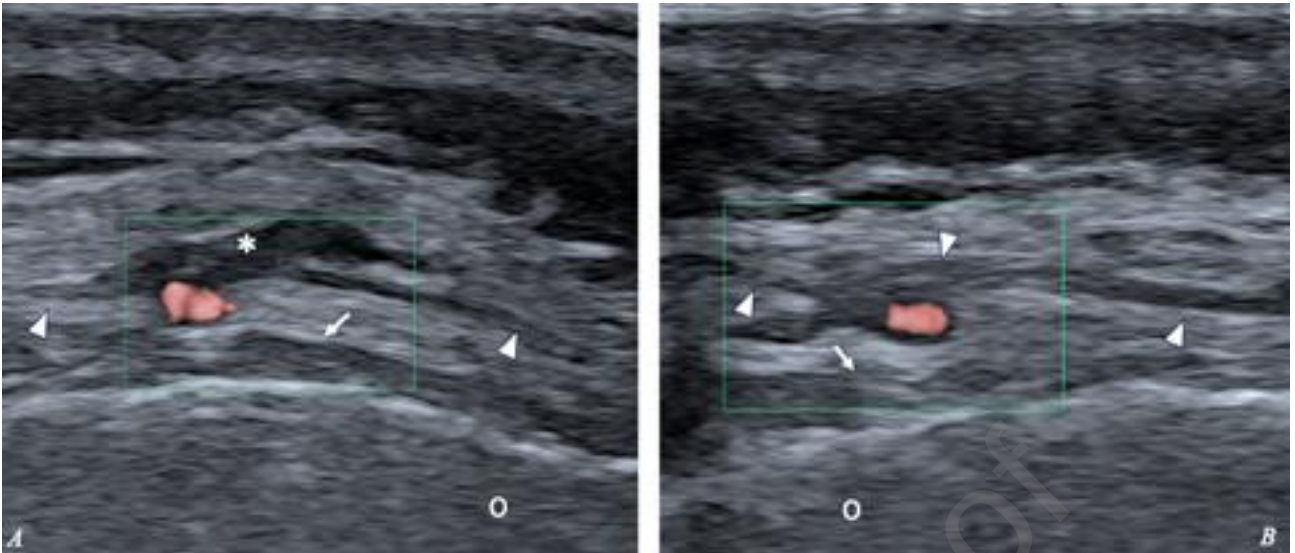


Figure 7. The images show the long axis view of a distal branch of the great occipital nerve (white arrowheads) in the subcutaneous tissue at the level of the superior nuchal line. On the affected side (7a), there is hypoechoogenic fibrotic tissue (white asterisk) surrounding the nerve at the point where it runs superficially to the occipital artery (red dot in the colored Power Doppler box). In 7b, contralateral normal findings. White arrow: aponeurosis of the trapezium that inserts on the occipital bone (O).

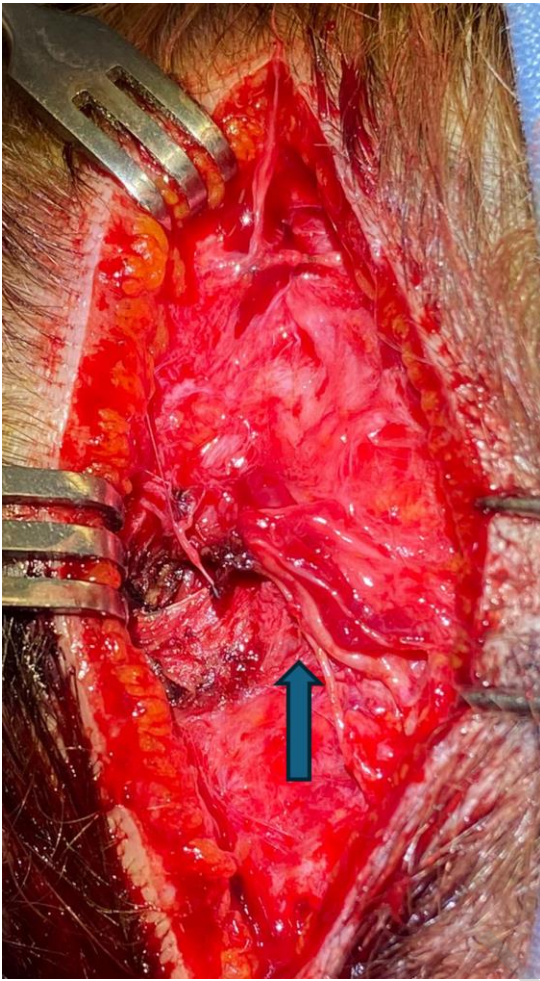


Figure 8. Point of contact between the GON and the OA (blue arrow).

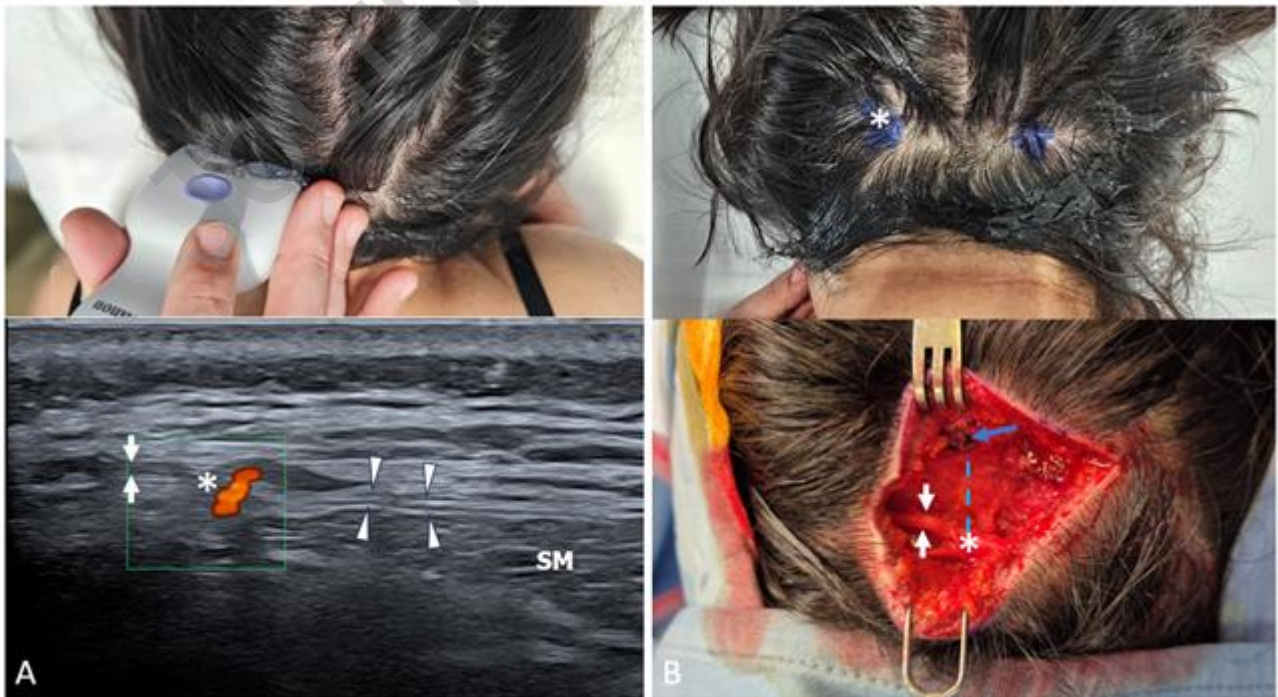


Figure 9. A: Ultrasound longitudinal view of the left GON (white arrowheads and arrows) located between the trapezius aponeurosis and the semispinalis muscle (SM). The segment of the GON, proximally to its crossing with the left occipital artery (red Doppler signal) and the trapezius fascia, appears normal (white arrowheads). However, distal to the crossing point (white asterisk), the nerve appears enlarged (white arrows). Additionally, at the crossing point, the occipital artery is ectatic, with a hypoechoic halo surrounding it. However distal to the crossing point (asterisk), the nerve appears enlarged (white arrows). Also, at the crossing point, the artery appears ectatic with hypoechoic halo surrounding it. **B:** The enlarged GON (white arrows) crosses (asterisk) the occipital artery (blue dotted line), which appears barely coagulated (blue arrow) in the image.

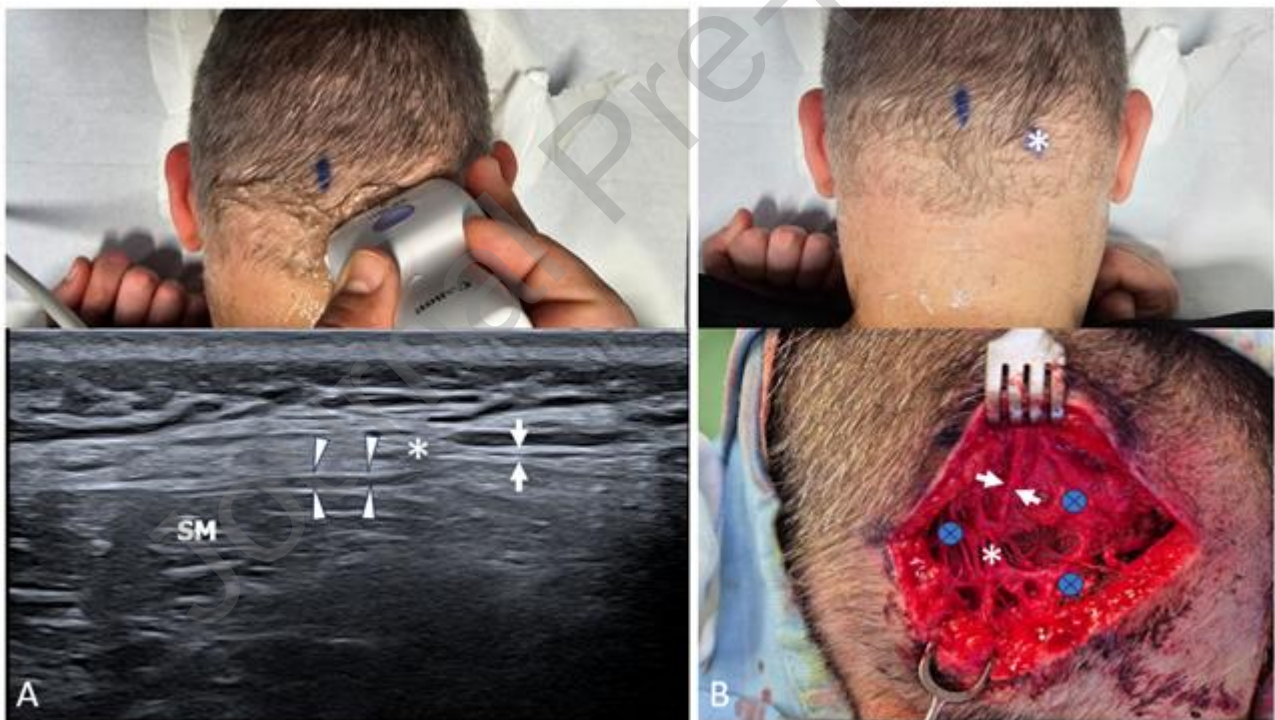


Figure 10. A: Ultrasound longitudinal view of the right GON (white arrowheads and arrows) between the trapezius aponeurosis and the semispinalis muscle (SM). Proximally to the crossing point (white asterisk), the nerve appears swollen (white arrowheads), while the artery does not appear enlarged. The compression causing the intraneural edema is likely related to the pressure exerted by the fascia. Distal to the crossing point, the nerve diameter

appears normal (white arrows). B: The fibrotic band (blue dots) surrounds the GON (white arrows). The occipital artery is not clearly visible near the nerve at the crossing point (white asterisk).

Tables

Table 1. Demographic and clinical data of Patients and Control Group.

Demographic and clinical data	Patients		Control Group	
	N.	%	N.	%
Sex				
males	7	35.0	9	40.9
females	13	65.0	13	59.1
Comorbidities				
yes	8	40.0	3	13.6
no	10	60.0	19	86.4
GON pathological alterations during ultrasound				
yes	16	80.0	3	13.6
no	4	20.0	19	86.4
Laterality of the surgery				
right	5	25.0		
left	1	5.0	-	-
bilateral	14	70.0		
Correspondence between pain and GON/OA crossing point				
yes	17	85.0	-	-
no	3	15.0	-	-
Tissues pathological alterations during surgery				
perineural fibrosis	15	75.0	-	-
vascular ectasia	10	50.0	-	-
close contact OA/GON	16	80.0	-	-
GON thickening	8	40.0	-	-
GON bifurcation	1	5.0	-	-
Correspondence between surgical and ultrasound findings				
yes	16	80.0	-	-
no	4	20.0	-	-

Table 2. Demographic and clinical data of Patients and Control Group

Demographic and clinical data	Patients (n=20)		Control Group (n=22)		p	95% Confidence Interval of the Difference	
	Mean	± ds	Mean	± ds		Lower	Upper
Age (years)							
Total	46.8	14.6	41.9	15.4	p = 0.299	18.641	32.759

Males	51.7	16.1	41.3	16.9			
Females	44.2	13.6	42.3	15.0			
BMI						-2.233	1.618
Total	22.1	3.1	22.4	3.1	p = 0.748		
Males	24.3	3.1	24.3	1.4			
Females	20.9	2.4	21.1	3.3			
Pain during ultrasound							
right	5.1	2.9	0.2	0.4	p = 0.001	3.444	6.491
left	2.6	2.5	0.1	0.4	p = 0.005	1.314	3.766
Elapsed time onset (years)	25.7	16.4	-	-			
GON CSA (mm ²) IOM							
right	1.9	0.9	2.0	0.6	p = 0.617	-6.381	0.383
left	1.8	0.8	1.6	0.7	p = 0.388	-0.271	0.683
GON CSA (mm ²) TM-SM							
face							
right	2.1	1.1	1.9	1.2	p = 0.476	-0.482	1.013
left	1.9	0.8	1.7	0.8	p = 0.442	-0.321	0.722
OA diameter (mm)							
right	1.9	0.6	2.3	0.4	p = 0.043	-0.660	-0.011
left	2.0	0.7	2.3	0.7	p = 0.134	-0.731	0.101

Table 3: reliability statistics and Inter-Item Correlation Matrix

CRONBACH'S ALPHA		CRONBACH'S ALPHA BASED ON STANDARDIZED ITEMS			N OF ITEMS	
0.701		0.707			6	
Inter-Item Correlation Matrix						
	CSA GON (mm ²) MOI ¹ R1	CSA GON (mm ²) between FT and MS ² R1	Major diameter (mm) AO ³ R1	CSA GON (mm ²) MOI ¹ R2	CSA GON (mm ²) between FT and MS ² R2	Major diameter (mm) AO ³ R2
CSA GON (mm ²) MOI ¹ R1	1.000	0.303	0.074	0.928	0.158	0.328
CSA GON (mm ²) between TM and SM ² R1		1.000	0.056	0.155	0.681	0.513
Major diameter (mm) AO ³ R1			1.000	0.090	0.135	0.282
CSA GON (mm ²) MOI ¹ R2				1.000	0.172	0.143
CSA GON (mm ²) between TM and SM ² R2					1.000	0.281
Major diameter (mm) AO ³ R2						1.000