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REVIEW

Collet-Sicard syndrome: a scoping review

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Abstract

Collet-Sicard syndrome (CSS) is the unilateral palsy of the cranial nerves (CN) IX, X, XI, and XII. To our knowledge, no review describes the characteristics of patients diagnosed with CSS. Therefore, this review aims to collect and describe all cases in the literature labeled as CSS. We performed a scoping review of the literature and conducted a database search in Embase and PubMed. We included articles and abstracts with case reports or case series of patients with CSS diagnosis. We classified the cases into two groups: "CSS", referring to patients presenting exclusively with IX-XII nerve involvement, and "CSS-plus", which corresponds to cases with CSS and other neurological impairments. We included 135 patients from 126 articles, of which 84 (67.7%) were male. The most common clinical manifestations reported were dysphagia and dysphonia. The most common etiology was tumoral in 53 cases (39.6%) and vascular in 37 cases (27.6%). The majority of patients showed partial or total improvement, with just over half receiving conservative treatment. The most frequent anatomic space was the jugular foramen (44.4%) and the parapharyngeal retrostyloid space (28.9%). Approximately 21% of the patients had other CN impairments, with the seventh and eighth CN most frequently compromised. We conclude that although there is a need for greater rigor in CSS reporting, the syndrome has a clear utility in identifying the localization of jugular foramen and parapharyngeal retrostyloid space pathology.

Keywords Collet-Sicard syndrome · Review · Scoping review · Tumor · Skull base · Lower cranial nerves

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Introduction

Collet-Sicard Syndrome (CSS) was first described in 1915 by a French otolaryngologist named Fréderic Justin Collet, who named this clinical entity [19, 26]. Independently, in 1917, the French radiologist and neurologist Jean-Athanase Sicard described the same syndrome [26, 112]. CSS is defined as unilateral palsy of the cranial nerves (CN) IX, X, XI, and XII. This syndrome includes dysphagia, absent gag reflex, impaired taste and sensation over the posterior third of the tongue, dysphonia, tongue deviation, paralysis of the palate, hemianesthesia of the larynx, pharynx, and soft palate, weakness of trapezius, and sternocleidomastoid muscle [120, 127]

CSS is a rare and understudied class of disease. No prevalence or incidence is reported in the literature. This syndrome has various etiologies, including malignant and benign neoplasms, vascular disease, and trauma, among others [2]. To our knowledge, no review describes the characteristics of patients diagnosed with CSS. Thus, this scoping review aims to collect and describe all cases in the literature labeled as CSS.

Materials & methods

We carried out a Scoping Review of the Literature. This study was conducted and reported in accordance with the PRISMA extension for Scoping Reviews (PRISMA-ScR 2018) [124]. We conducted a database search in October 2021 using Embase terms "'collet Sicard syndrome'/exp OR 'collet Sicard syndrome'" and PubMed terms "Collet Sicard" OR "Collet Sicard Syndrome". We did not limit the search according to publication date. We included articles and abstracts written in English, Spanish, Portuguese, German, and French, with case reports or case series of patients with a Collet-Sicard diagnosis. We excluded review articles without any patient description. A total of 231 articles were extracted from both databases and gray literature; 88 were duplicated, and 18 fulfilled the exclusion criteria. Only 126 articles fulfilled the inclusion criteria and 135 patients were included in the analysis (Appendix Table 5).

We collected data considering the cervical spaces described by *Varoquaux* et al. and *Kamalian* et al., who divided them into suprahyoid and infrahyoid [47, 126]. Suprahyoid spaces regarded for the analysis were: prevertebral space, parotid space, masticator space, retropharyngeal space, pharyngeal mucosal space, and parapharyngeal space. Tenso-vascular-styloid fascia divided this last space into pre-styloid and retro-styloid spaces (PRS). The retro-styloid space refers to the carotid sheath in the suprahyoid space [47] (Fig. 2).

Furthermore, we assigned an additional classification to CSS etiologies, considering those cases that started outside of the cranium as extracranial, those that formed within the bone such as metastases, as intraosseous, and those that started within the cranium as intracranial. More than one label could be attached to the same case if more than one of the aforementioned spaces was compromised.

Two independent reviewers (MP and MA) extracted the data from each article and the discrepancies were solved in a discussion between them. Data were recorded in a Microsoft Office 365, Microsoft Inc., Redmond, Washington, United States Excel spreadsheet. We classified the cases into two groups: "CSS", which refers to patients presenting exclusively with IX-XII cranial nerve involvement, and "CSS-plus", which corresponds to cases presenting with the original syndrome description and other neurological impairments.

For the statistical analysis, the normal distribution of quantitative variables was assessed using the Kolmogorov–Smirnov test. Variables with normal distribution were described with mean and standard deviation, while those with non-normal distribution were described with median and interquartile range. Categorical variables were described with their absolute and relative frequencies. The differences in categorical variables were assessed with X^2 test and Fisher's exact test, and the Mann–Whitney U test was used for continuous variables. Statistical significance was met at $p<0.05$. Data was analyzed using the SPSS, International Business Machines Corporation, Armonk, New York, United States, personal license Version 26 for Mac iOS.

Results

We included a total of 126 articles and 135 patients diagnosed with CSS (Fig. 1, PRISMA diagram). Of them, 84 (67.7%) were male, with a median age at diagnosis of 55 [IQR, 41.0—67.0]; 40 (32.3%) were female, with a median age of 50 [IQR, 37.0—62.0]. The most common clinical manifestations were dysphagia ($n=92$, 68.15%), dysphonia ($n=78$, 57.78%), tongue deviation ($n=60$, 44.44%), palate paralysis ($n=54$, 40.00%), and trapezius weakness ($n=48$, 35.56%) (Table 1).

The principal etiology was tumoral with 53 cases (39.6%), with glomus jugulare ($n=11$, 20.75%) and metastatic prostate adenocarcinoma ($n=8$, 15,1%) as the most frequent tumor types. This is followed by vascular etiology, with 37 cases (27.6%). Internal carotid dissection was the most reported cause in this subgroup ($n=20$, 54.1%), followed by jugular thrombosis ($n=5$, 13.5%), and granulomatosis with polyangiitis ($n=5$, 13.5%). Traumatic etiology was the third most common cause of CSS with 32 cases (23.9%), in which condylar ($n=18$, 56.25%) and Jefferson fracture ($n=7$, 21.9%) represented the most frequent trauma-related injuries. Other congenital, iatrogenic, and infectious causes were less frequent.

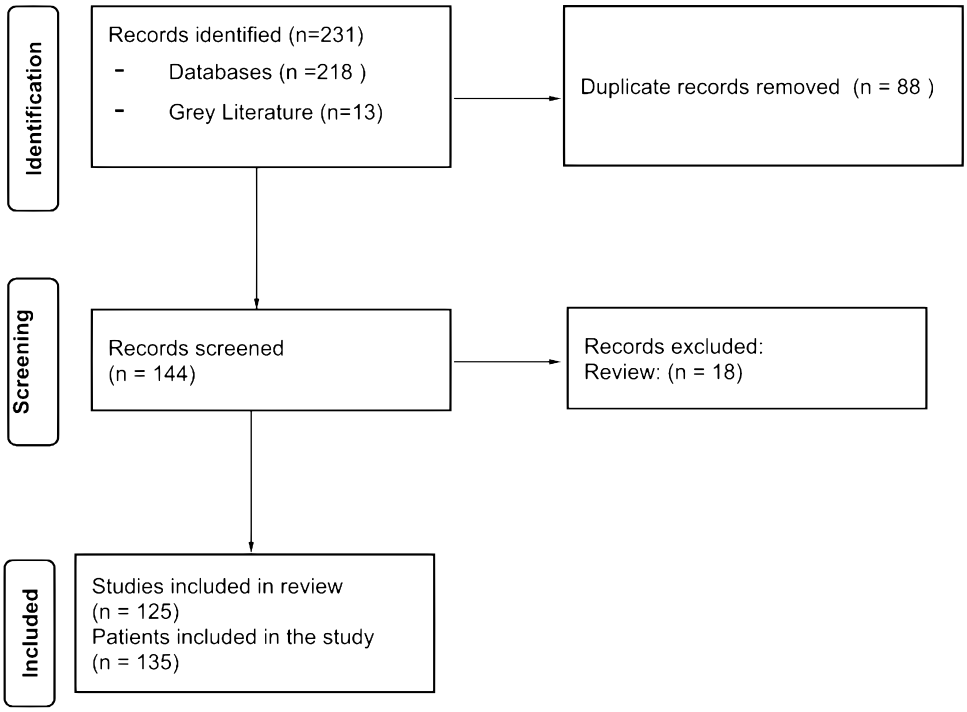

Fig. 1 PRISMA flow diagram

The rest of the specific etiologies are shown in Table 2. Half of the patients underwent conservative treatment, and the majority showed partial or total improvement The majority of patients showed partial or total improvement, with just over half receiving conservative treatment. Follow-up was made in 48.1% of patients. Additional CN impairment was present in 21% of patients. Other general characteristics of the patients included are shown in Table 1.

Table 1 Comparison between CSS and CSS-plus

	Total *N* (%) *N* (%)	CSS *N* (%)	CSS-plus *N* (%)	*P* value
		106 (78.5)	29 (21)	
Age*	53 (37—63)	53.50 (38.25—62.00)	55.00 (37.00—67.00)	0.956
Sex				0.077
Female	40 (32.3)	29 (28.7)	11 (47.8)	
Male	84 (67.7)	72 (71.3)	12 (52.2)	
General Cause				0.245
Congenital	3 (2.2)	2 (1.9)	1 (3.4)	
Iatrogenic	5 (3.7)	5 (4.8)	0 (0.0)	
Infectious	4 (3.0)	2 (1.9)	2 (6.9)	
Trauma	32 (23.9)	27 (25.7)	5 (17.2)	
Tumor	53 (39.6)	38 (36.2)	15 (51.7)	
Vascular	37 (27.6)	31 (39.5)	6 (20.7)	
Space				
Jugular Foramen	60 (44.4)	45 (42.5)	15 (51.7)	0.373
Parapharyngeal Retrostyloid Space	39 (28.9)	31 (29.2)	8 (27.6)	0.861
Prevertebral Space	27 (20.0)	24 (22.6)	3 (10.3)	0.142
Prestyloid Parapharyngeal Space	19 (14.1)	13 (12.3)	6 (20.7)	0.193
Hypoglossal Foramen	12 (8.9)	9 (8.5)	3 (10.3)	0.498
Foramen Magnum	4 (3.0)	3 (2.8)	1 (3.4)	0.625
Parotid Space	2 (1.5)	1 (0.9)	1 (3.4)	0.385
Retropharyngeal Space	1 (0.7)	0 (0.0)	1 (3.4)	0.215

Table 1 (continued)

	Total *N* (%) *N* (%)	CSS *N* (%)	CSS-plus *N* (%)	*P* value
Side				0.845
Left	69 (56.6)	55 (56.1)	14 (58.3)	
Right	53 (43.4)	43 (43.9)	10 (41.7)	
Treatment				
Surgical	24 (18.3)	17 (16.3)	7 (25.9)	0.190
Conservative	73 (55.7)	58 (55.8)	15 (55.6)	0.984
Radiotherapy	23 (17.6)	16 (15.4)	7 (25.9)	0.158
Endovascular	7 (5.3)	4 (3.8)	3 (11.1)	0.135
Chemotherapy	5 (3.8)	1 (1.0)	4 (14.8)	0.006
Improvement				0.103
Total	22 (23.4)	16 (21.3)	6 (31.6)	
Partial	54 (57.4)	47 (62.7)	7 (36.8)	
None	18 (19.1)	12 (16.0)	6 (31.6)	
Sympathetic	5 (3.7)	1 (1.0)	4 (14.3)	0.007
Follow up	65 (87.8)	49 (87.5)	16 (88.9)	0.621
Signs and Symptoms				
Dysphagia	92 (68.1)	76 (71.70)	16 (55.2)	0.365
Hoarseness or Dysphonia	78 (57.8)	67 (63.21)	11 (37.9)	0.812
Tongue Weakness or Deviation	60 (44.4)	50 (47.17)	10 (34.5)	0.606
Palate deviation or paralysis	54 (40.0)	44 (41.51)	10 (34.5)	0.863
Trapezius Weakness	48 (35.6)	39 (36.79)	9 (31.0)	0.679
Paralysis of the vocal cord	43 (31.8)	33 (31.13)	10 (34.5)	0.508
Gag reflex absent	42 (31.1)	34 (32.08)	8 (27.6)	0.800
Dysarthria	37 (27.4)	30 (28.30)	7 (24.1)	0.779
Weak Sternocleidomastoid	31 (23.0)	27 (25.47)	4 (13.8)	0.894
Tongue atrophy	28 (20.7)	20 (18.87)	8 (27.6)	0.494
Sternocleidomastoid atrophy	15 (11.1)	11 (10.38)	4 (13.8)	0.860
Shoulder weakness	13 (9.6)	10 (9.43)	3 (10.3)	0.737
Trapezius atrophy	13 (9.6)	9 (8.49)	4 (13.8)	0.864
Loss sensation larynx	9 (6.7)	7 (6.60)	2 (6.9)	0.673
Tongue Fasciculations	9 (6.7)	7 (6.60)	2 (6.9)	0.589
Taste impairment	5 (3.7)	5 (4.72)	0 (0.0)	0.424

*Median (IQR)

Table 3 shows the anatomic distribution and its main etiologies. The most common anatomic spaces were the jugular foramen (44.4%), and the parapharyngeal retrostyloid space (28.9%). The jugular foramen was most commonly affected by tumors (53.3%), and the PRS by vascular pathology (61.5%) (Fig. 2). The prevertebral anatomic space was most commonly affected by traumatic injury (74%). Most of these lesions were located in the extracranial (41.4%) and intraosseous (30.8%) regions.

Table 1 shows the characteristics of CSS compared to CSS-plus. Approximately 21% of the patients had other CN impairments, with the seventh and eighth CNs most frequently affected (Table 4).

Discussion

We conducted a scoping review to characterize CSS, focusing on the affected anatomical spaces and clinical characteristics. Since its first description in 1915, we found 126 studies describing CSS in 135 patients.

Etiology

Gutierrez et al. described in 2015 the main causes of CSS in 51 cases extracted from PubMed. They reported that the most common etiology was metastasis, while our study

Table 2 Specific cause of Collet-Sicard syndrome

	n	(%)
Tumor		
Glomus	11	8.15
Metastatic Prostate Adenocarcinoma	8	5.93
Metastatic breast adenocarcinoma	5	3.70
Other Metastatic disease	5	3.70
Lower cranial nerves Schwannoma	5	3.70
Multiple Myeloma	4	2.96
Unspecified	4	2.96
Metastatic lung Adenocarcinoma	2	1.48
Metastatic colorectal carcinoma	2	1.48
Hemangiopericytoma	2	1.48
Adenocarcinoma of unknown primary origin	1	0.74
Meningioma	1	0.74
Inflammatory pseudotumor	1	0.74
Fibrosarcoma	1	0.74
Malignant Perivascular Epithelioid Cell Tumor	1	0.74
Vascular		
Internal Carotid Dissection	20	14.81
Granulomatosis with Polyangiitis	5	3.70
Jugular Thrombosis	5	3.70
ICA Fibromuscular Displasia	2	1.48
ICA Aneurysm	2	1.48
Panarteritis nodosa	2	1.48
APA ischemia following cardiovascular surgery	1	0.74
Congenital		
Eagle's syndrome	2	1.48
Occiput C1-C3 fusion, hypoplastic dens, left hypertrophic C1 and C2 Transverse Processes	1	0.74
Infectiuos		
Abscess/osteomyelitis	3	2.22
Viral infection	1	0.74
Iatrogenic		
Catheter-related jugular vein thrombosis	3	2.22
Post-coiling carotid aneurysm	1	0.74
Cervicofacial emphysema after cryoblockade to the mandibular division of the trigeminal nerve	1	0.74
Trauma*		
Occipital Condyle Fracture	18	13.33
Jefferson fracture	7	5.19
Unspecified	4	2.96
Petrous fracture	3	2.22
Clivus fracture	3	2.22
Styloid process fracture	1	0.74

*Cases could present with one or more injuries

found that glomus jugulare was the most frequent etiology in the tumoral group. In contrast to Gutierrez et al., we also found congenital disease (Developmental abnormalities of the craniocervical junction, Eagle's syndrome) as a possible cause [48, 76, 114].

We found that the most prevalent tumoral causes were glomus jugulare and metastatic tumors. Glomus jugulare represents 0.6% of all head and neck tumors [49]. Although rare, glomus jugulare neoplasm is the most frequent tumor of the jugular foramen, constituting 60–80% of all such cases [45]. In concordance with Jaiswal et al. we found glomus jugulare predominantly in females [45]. Its usual clinical presentation consists of pulsatile tinnitus, hearing loss, vertigo, and in some cases, lower CN impairment [45, 59]. However, we believe its slow-growing and locally invasive behavior explains why it is the most frequent cause of CSS, given the close vicinity between the jugular and hypoglossal canal.

Among metastatic tumors, prostate and breast cancer were the most frequent primary neoplasms that presented with CSS, in alignment with Opie et al. [79]. This finding is probably related to prostate adenocarcinoma being the most frequent primary tumor metastasizing to the skull base [35, 57].

Internal carotid artery dissection represented the most common specific cause overall, and the most frequent within the vascular etiology group. Carotid dissection occasionally presents with paresis of the cranial nerves and, to a greater extent, with lower cranial nerve compromise, with the XII CN being the most affected one [36, 38, 67, 68, 120]. In the literature, we found two hypotheses to explain the pathophysiology of lower CN palsy secondary to internal carotid dissection. The first is localized ischemia of the small vessels affecting the lower cranial nerves [36, 38]. The second and most accepted hypothesis is intramural hematoma formation due to arterial dissection. This hematoma can compress the lower cranial nerves leading to CSS [36, 38, 115]. Interestingly, most of the patients in our study diagnosed with CSS due to internal carotid artery dissection had some recovery, supporting the intramural hematoma hypothesis, as compression carries a better prognosis than ischemia [38]. The parapharyngeal retrostyloid space was the most frequent anatomical space affected in the vascular group. In this space, the lower cranial nerves, sympathetic chain, and carotid sheath can be found, which in turn contains the cervical or C1 segment of the internal carotid artery according to the Bouthillier classification. This anatomical proximity explains how intramural hematoma formation causes compression in the last four CNs [115, 121] (Fig. 3).

Table 3 Anatomic space and general cause

Anatomic Space	Tumor *N* (%)	Vascular *N* (%)	Trauma *N* (%)	Infectious* *N* (%)	Iatrogenic *N* (%)	Congenital *N* (%)	Total *N* (%)
Jugular Foramen	32 (53.3)	12 (20.0)	11 (18.3)	3 (5.0)	2 (3.3)	0 (0.0)	60 (44.4)
Parapharyngeal Retrostyloid Space	5 (12.8)	24 (61.5)	4 (10.3)	2 (5.1)	2 (5.1)	2 (5.1)	39 (28.9)
Prevertebral Space	6 (22.2)	0 (0.0)	20 (74.0)	1 (3.7)	0 (0.0)	0 (0.0)	27 (20.0)
Prestyloid Parapharyngeal Space	11 (57.9)	3 (15.8)	2 (10.5)	0 (0.0)	2 (10.5)	1 (5.3)	19 (14.1)
Hypoglossal Foramen	8 (66.7)	1 (8.3)	2 (16.7)	1 (8.3)	0 (0.0)	0 (0.0)	12 (8.9)
Foramen Magnum	2 (50.0)	0 (0.0)	1 (25.0)	1 (25.0)	0 (0.0)	0 (0.0)	4 (3.0)
Parotid Space	2 (100.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	2 (1.5)
Retropharyngeal Space	1 (100.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	1 (0.7)

Cases may present with one or more spaces affected simultaneously

*Not including viral infection

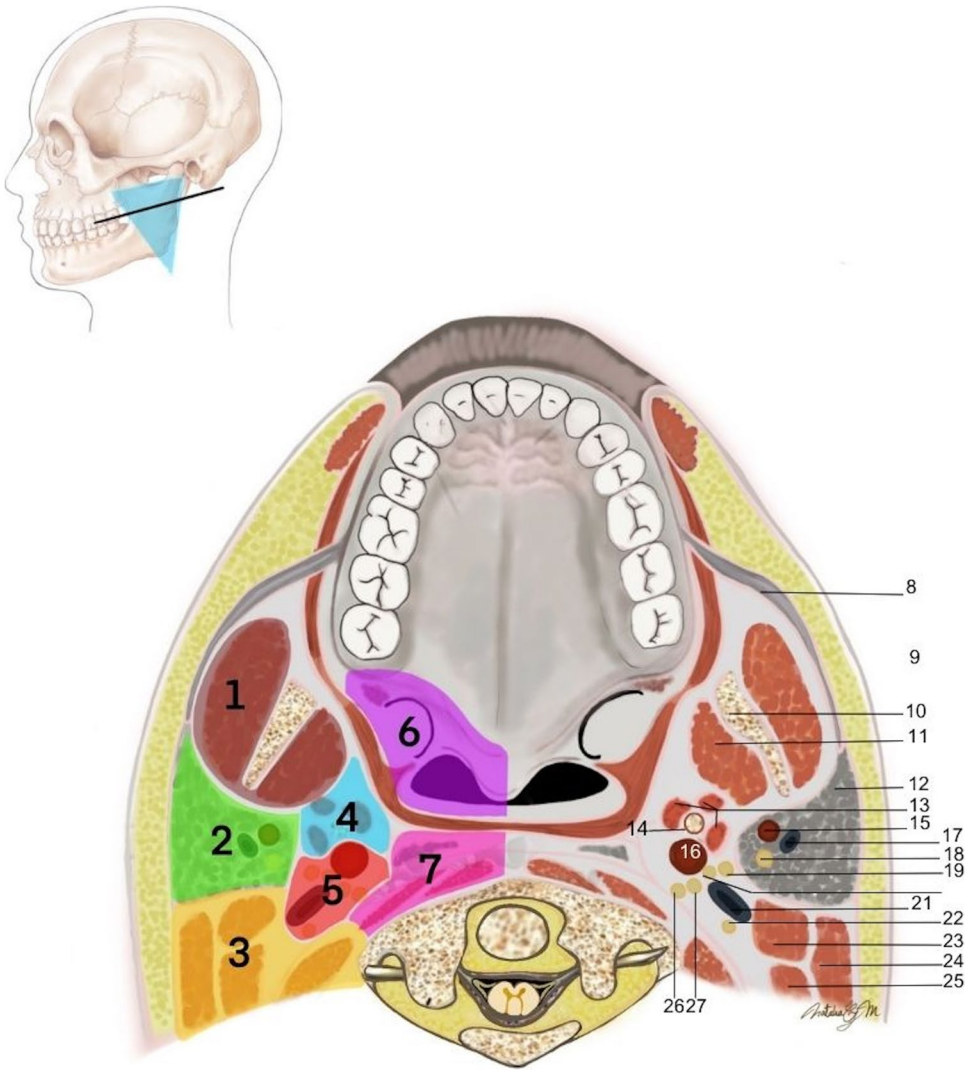

Fig. 2 Relationship of the retrostyloid parapharyngeal space with adjacent cervical spaces. Axial cut a2 the level of the parotid gland. 1. Masticatory space, 2. Parotid space, 3. Parapharyngeal mucosal space, 4. Parapharyngeal prestyloid space, 5. Parapharyngeal restrostyloid space, 6. Pharyngeal mucosal space, 7. Prevertebral space. 8. Parotid duct 9. Masseter muscle 10. Mandible 11. Medial pterygoid muscle 12. Parotid gland 13. Stylopharyngeal muscle, Stylologlossus muscle, Stylohyoid muscle 14. Styloid process 15. External carotid artery 16. Internal carotid artery. 17. Retromandibular vein 18. Facial nerve 19. Hypoglossal nerve 20. Accessory nerve 21. Internal jugular vein 22. Cervical sympathetic trunk 23. Digastric muscle 24. Sternocleidomastoid muscle 25. Longissimus muscle 26. Glossopharyngeal nerve 27. Vagus nerve. Illustration created using Procreate version 5.3.4

Table 4 Additional cranial nerve impairment

Cranial Nerve	
Facial Nerve	17 (12.6)
Vestibulocochlear Nerve	8 (5.9)
Trochlear Nerve	3 (2.2)
Trigeminal Nerve	2 (1.5)
Abducens Nerve	2 (1.5)
Oculomotor Nerve	1 (0.8)
Optic Nerve	1 (0.8)

Lastly, the third most frequent etiology was traumatic injury. This group had two predominant injuries: condyle fracture and Jefferson fracture. Jefferson fracture or atlas fracture represents 1% of all spinal injuries [108]. Domenicucci et al. evaluated post-traumatic CSS cases, finding that the most frequent mechanism of injury for the Jefferson fracture is the axial loading along the axis of the cervical spine, which can happen in conjunction with mechanisms of lateral bending and rotation [26]. The last four cranial nerves pass through the space between the transverse process of the atlas and the styloid process. In a Jefferson fracture, the transverse process comes closer to the styloid process compressing and stretching those cranial nerves resulting in CSS [26] (Fig. 4). On the other hand, condyle fracture, when there is dislocation of this structure, causes tearing of the lower cranial nerves, making the recovery of these patients more difficult compared with the Jefferson fracture [26].

Cranial nerve and clinical manifestations

The typical clinical manifestations of CSS described by Lian et al. were vocal cord paralysis, dysphagia, impairment of taste in the posterior third of the tongue, weakness of the tongue, and sternocleidomastoid [1, 63]. However, in our study, the most frequent clinical manifestations were dysphagia, dysphonia, tongue deviation, palate paralysis, and trapezius weakness. We consider that the variability in the clinical manifestations can contribute to initial misdiagnosis and delay in achieving a correct diagnosis.

Twenty-nine patients (21%) presented other cranial nerve impairments in addition to IX, X, XI, and XII. The VII CN was be the most frequent additional CN affected in cases reported as CSS. In this review, it was affected in 17 patients (12.6%) [20, 48, 49, 70, 89, 110, 111, 117, 123, 131, 134]. Most of them (11 cases, 64.7%) presented an extracranial lesion, where the extratemporal course of the VII CN begins, in close proximity to the jugular foramen and PRS [51]. In five (29%) of these extracranial cases, there was more than one type of injury; within this subgroup, a concomitant intraosseous lesion was the most frequent (4 cases, 23.5%). The remaining cases of patients presenting with facial palsy had intraosseous compromise (9 cases, 52.9%), followed by intracranial injury alone (3 cases, 17.5%).

A group of patients with CSS and hearing loss did not have VIII CN impairment, but conductive hearing loss secondary

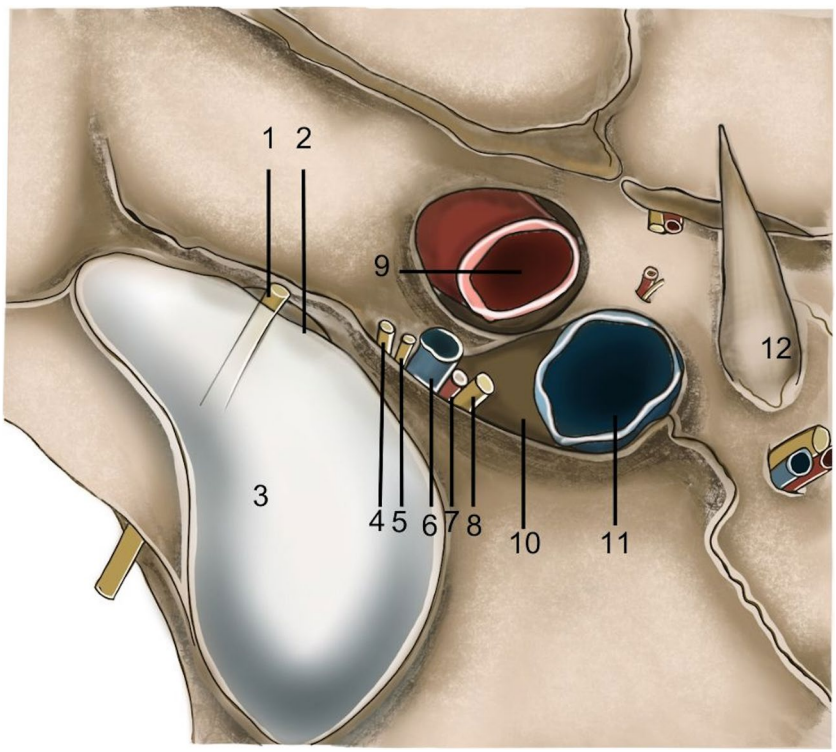

Fig. 3 Anatomical relationships of the lower cranial nerves and surrounding structures. 1. Hipoglossal nerve; 2. Hipoglossal canal; 3. Occipital condyle; 4. Glossopharyngeal nerve; 5. Vagus nerve; 6. Inferior petrosal sinus; 7. Posterior meningeal artery; 8. Accessory nerve; 9. Carotid artery; 10. Jugular foramen; 11. Internal jugular vein; 12. Styloid process. Illustration created using Procreate version 5.3.4

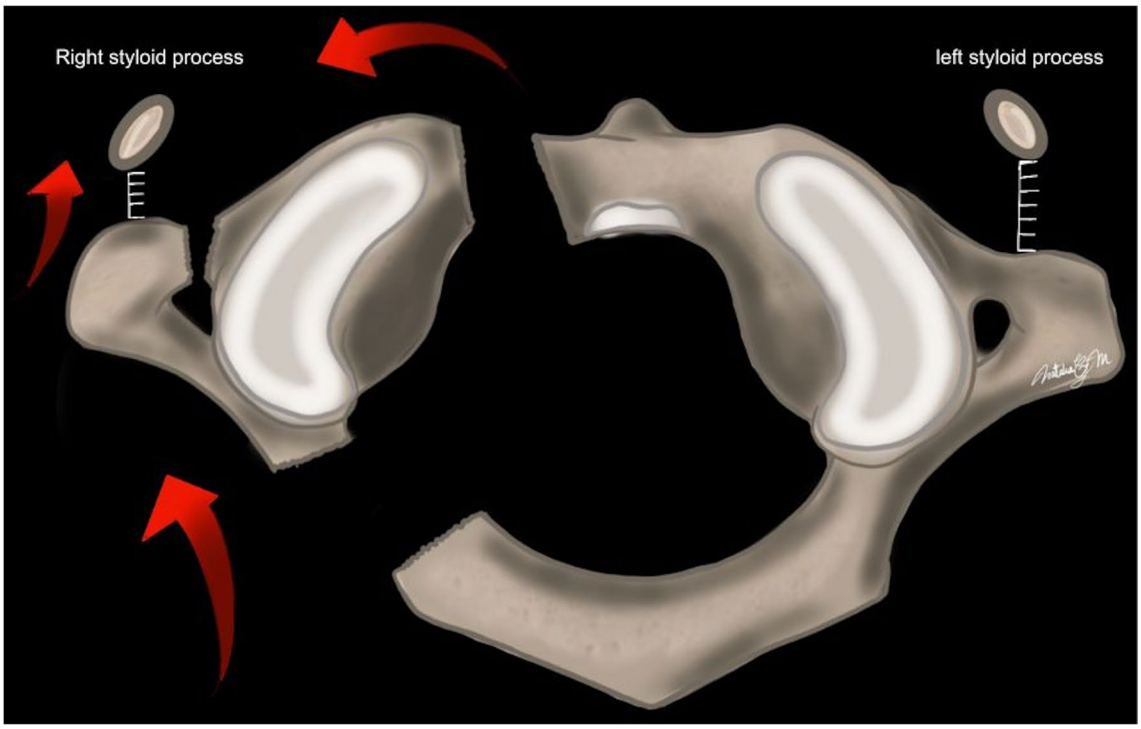

Fig. 4 Relationship between the styloid process and Jefferson fracture. A Jefferson fracture decreases the space between the right lateral mass and the ipsilateral styloid process, where the IX, X, XI CN are located. Illustration created using Procreate version 5.3.4

to otitis media [49, 103–105, 107, 111, 122]. Therefore, when a patient presents with CSS and hearing loss, it is crucial to evaluate the auditory system and not assume that the hearing loss is a consequence of VIII CN damage.

Higher cranial nerve involvement (i.e. oculomotor, trochlear, trigeminal, and abducens) was observed in patients with systemic diseases such as granulomatosis with polyangiitis or in high-energy traumatic brain injury [10, 22, 62, 85].

Compromised anatomical spaces

The most common anatomical spaces impaired in CSS were the jugular foramen [94] and the parapharyngeal retrostyloid space, consistent with the most frequent etiologies identified, considering that glomus jugulare and internal carotid dissection are usually located in those spaces. However, a significant percentage (41 patients, 30%) of cases comprised other anatomical spaces.

In contrast to previous literature, the cases we found did not have retroparotid space compromise. Collet was the first and only author to mention the retroparotid space in connection with CSS in 1915. Since then, the term has been used in educational texts and case reports of Villaret syndrome, but not in CSS cases [14, 30, 113]. Our findings suggest that PRS describes more thoroughly one of the spaces affected in CSS than the term "retroparotid space" does. Therefore, we suggest that this last term should be discontinued when referring to CSS.

There was no statistically significant difference between CSS and CSS-plus in terms of etiology, clinical manifestations, anatomical space, and treatment. This means that the impairment of other CN, in addition to the IX-XII, does not significantly affect the disease course. Indeed, there was no statistically significant difference between the impairment of VII-XII, VIII-XII, or IX-XII CN.

The only statistically significant difference between the two groups was sympathetic fiber impairment; however, the compromise of the last four CNs and ipsilateral sympathetic impairment is known as Villaret syndrome [14, 30], which leads us to conclude that the cases with these clinical characteristics were mislabeled as CSS.

The limitations of this study are as follows: first, the sample size is limited, which may explain the absence of any statistically significant differences. Second, we performed a systematic literature search of all cases, however, some cases could be published in non-indexed journals, and some diagnosed cases in clinical practice may not be published, possibly leading to underestimation.

In conclusion, CSS is a rare pathology. The most commonly affected anatomical locations are the jugular foramen and parapharyngeal retrostyloid space. Hence, pathologies in those spaces should be ruled out in patients presenting with CSS. Tumoral etiology should also be considered in patients with clinical signs of lower CN impairment without a history of trauma. Furthermore, we found a lack of standardization when reporting the syndrome in the literature, which makes apparent the need for greater rigor in reporting CSS.

Appendix

Table 5 Selected studies

Author	Year	Publication Type	Number of Cases
Ordoñez-Granja et al. [80]	2020	Article	1
Shahrvini et al. [108]	2020	Article	1
Ganesh et al. [34]	2019	Article	1
Simões et al. [114]	2019	Article	1
Dey et al. [24]	2019	Article	1
Krishnan et al. [53]	2019	Article	1
Zamudio Moya et al. [135]	2019	Letter to the editor	1
Komune et al. [52]	2019	Conference Abstract	1
Behravesh et al. [10]	2019	Conference Abstract	1
Subha et al. [117]	2018	Article	1
Saliou et al. [98]	2018	Article	1
Erben et al. [27]	2018	Article	1
Peters et al. [85]	2018	Article	1
Oushy et al. [82]	2018	Article	1
Sanchez-Lars et al. [99]	2017	Letter to the editor	1
Mayer et al. [69]	2017	Article	1
Barbiero et al.[6]	2017	Article	1
Neo et al. [75]	2017	Article	1
Kang et al. [48]	2016	Article	1
Schuster et al. [104]	2016	Conference Abstract	1
Jaiswal et al. [45]	2016	Article	1
Neo et al. [75]	2016	Conference Abstract	2
Schuster et al. [105]	2016	Conference Abstract	1
Mnari et al. [70]	2016	Article	1
Domenicucci et al. [26]	2015	Article	1
Rebattu et al. [92]	1925	Article	1
Bolender et al. [12]	1978	Article	1
Young et al. [133]	1992	Article	1
Legros et al. [62]	2000	Article	1
Caroli et al. [16]	2005	Article	1
Kwon et al. [56]	2011	Article	1
Gutierrez Rios et al. [37]	2015	Letter to the editor	1
Yüce et al. [134]	2015	Article	1
Barna et al. [7]	2015	Article	1
Taga-Senirli et al. [118]	2014	Article	1
Nunes et al. [76]	2013	Conference Abstract	1
Oliveira et al. [78]	2013	Conference Abstract	1
Zelenak et al. [136]	2013	Article	1
Mayer et al. [69]	2017	Article	2
Smith et al. [115]	2013	Article	1
Khalid et al. [49]	2013	Article	1
Dettling et al. [23]	2013	Article	1
Climans et al. [18]	2013	Letter to the editor	1
Lakshminaray et al. [58]	2013	Conference Abstract	1
Tanaka et al. [119]	2012	Conference Abstract	1
Hayward et al. [42]	2012	Article	1
Rajput et al. [89]	2011	Article	1
Petrović et al. [86]	2011	Article	1
Opie et al. [79]	2010	Article	1
Battaglia et al. [9]	2009	Article	1
Sibai et al. [111]	2009	Article	1
Lee et al. [60]	2009	Article	1
Romero Muñoz et al. [95]	2009	Conference Abstract	1
Lucato et al. [66]	2008	Article	1
Erol et al. [29]	2007	Article	1
Chacon et al. [17]	2006	Article	1
Basu et al. [8]	2006	Article	1
Mohr et al. [72]	2006	Article	1
Shine et al. [110]	2005	Article	1
Garcia-Escriva et al. [33]	2005	Article	1
Jimenez-Caballero [46]	2004	Article	1
Hsu et al. [44]	2004	Article	1
Rao et al. [90]	2004	Article	1
Walker et al. [129]	2003	Article	1
Willy et al. [131]	2003	Article	1
Paparounas et al. [83]	2003	Article	1
Heckmann et al. [43]	2000	Letter to the editor	1
Schmidt et al. [102]	2000	Article	2
Satoh et al. [100]	2000	Article	1
Connolly et al. [21]	2000	Article	1
Rüegg et al. [96]	1999	Article	1
Krystkowiak et al. [54]	1998	Article	1
Larson et al. [59]	1997	Article	1
Rees et al. [93]	1997	Article	1
Tappin et al. [120]	1996	Article	1
Ruiz et al. [97]	1995	Article	1
Kurebayashi et al. [55]	1995	Article	3
Comacchio et al. [20]	1995	Article	1
Sharma et al. [109]	1994	Article	1
Prick et al. [88]	1992	Letter to the editor	1
Wani et al. [130]	1991	Article	1
Silvestrini et al. [113]	1991	Article	1
Sehitoglu et al. [107]	1990	Article	1
Moss et al. [73]	1989	Article	1
Arita et al. [5]	1989	Article	1
Waespe et al. [128]	1988	Article	1
Hashimoto et al. [40]	1988	Article	1
Di Stasi et al. [25]	1986	Article	1
Malin et al. [68]	1984	Conference Abstract	1
Havelius et al. [41]	1982	Article	1
Nagata et al. [74]	1980	Article	1
Mohanty et al. [71]	1973	Article	1
Tomio et al. [123]	1991	Article	1
Olazabal et al. [77]	2014	Article	1
Evan et al. [31]	2021	Article	1

Table 5 (continued)

Author	Year	Publication Type	Number of Cases
Arasawa et al. [4]	2020	Article	1
Lian et al. [63]	2020	Article	1
Schuster et al. [104]	2015	Article	1
Rath et al. [91]	2014	Conference Abstract	1
Amar et al. [3]	2012	Article	1
Villatoro et al. [127]	2011	Article	1
Prashant et al. [87]	2003	Article	1
Kobayashi et al. [50]	1981	Article	1
Otto et al. [81]	2001	Article	1
Schattner et al. [101]	2009	Article	1
Schweinfurth et al. [106]	1993	Article	1
Wilson et al. [132]	1984	Article	1
Amar et al. [3]	2020	Article	1
Cabreira et al. [15]	2020	Article	1
Lee et al. [61]	2017	Article	1
Handley et al. [39]	2010	Article	1
Low et al. [65]	2018	Article	1
Al-Shabibi et al. [2]	2021	Article	1
Sokhi et al. [116]	2021	Article	1
Utheim et al. [125]	2015	Article	1
Erkan et al. [28]	2016	Article	1
Cugy et al. [22]	2014	Conference Abstract	3
Liu et al. [64]	2004	Article	1
Pelliccioni et al. [84]	1995	Conference Abstract	3
Olazabal et al. [77]	2014	Article	1
Bravo et al. [13]	2018	Article	1
Fidelis de Souza et al. [32]	2018	Article	1
Krystkowiak et al. [54]	1998	Article	1
Collet [19]	1915	Article	1
Sicard [112]	1916	Article	1
Benavides et al. [11]	2021	Conference Abstract	1

Authors contributions Maria Paula Aguilera-Pena and Maria A. Castiblanco: Conceptualization, Literature search, study selection, Writing, and analysis. Valentina Osejo-Arcos: Literature search, Preparation of Figs. 1–4, Preparation of Tables 1–3, Writing. Santiago Gutiérrez-Gómez: Writing—Review and editing. Rafael Aponte-Caballero: Writing—Review and editing. Juan F. Abaunza-Camacho: Writing—Review and editing. Natalia Guevara-Moriones: Illustrations. Camilo Armando Benavides-Burbano: Review and editing, Supervision. William M. Riveros-Castillo: Supervision, Project Administration. Javier M. Saavedra: Writing—Review & Editing, Supervision. All authors reviewed the manuscript.

Declarations

Ethical approval This review did not require approval by an ethics committee.

Conflict of interest None.

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