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GUIDELINE

Diagnosis and Treatment of Venous Reflux Disorders of Head and Neck

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INTRODUCTION

Venous reflux disorders of the head and neck comprise a spectrum of syndromes characterized by impaired intracranial and extracranial venous drainage. Clinically, the most commonly encountered entities include cerebral venous thrombosis (CVT), cerebral venous sinus stenosis (CVSS), and internal jugular vein stenosis (IJVS), with the latter two arising from either intrinsic or extrinsic factors[1]. In recent years, advances in neuroimaging and the broader adoption of minimally endovascular techniques have accelerated progress in this field. While CVT is supported by relatively mature guidelines and consensus frameworks, evidence for CVSS and IJVS remains heterogeneous and clinical practice varies across centers. Based on a systematic review of the literature and multidisciplinary expert experience, this guideline focuses on CVSS and IJVS, offering unified, practice-oriented recommendations for diagnostic workup and standardized treatment pathways. CVT is beyond the scope of this article.

CLINICAL FEATURES OF CVSS/IJVS

Venous reflux disorders of head and neck show marked clinical heterogeneity in both presentation and course. Patients often report one or more non-specific symptoms that overlap with common neurological, otologic, and ophthalmologic presentations. Imaging findings may also be influenced by posture, flow status, and acquisition technique, contributing to under-recognition, misclassification, and substantial variability in management.

CVSS most frequently involves the unilateral or bilateral transverse–sigmoid sinus region and may also affect the superior sagittal sinus. It can occur in the setting of head trauma, intracranial tumors, arachnoid granulations, thrombus, or sustained intracranial hypertension. Clinical manifestations are typically related to raised intracranial pressure, with headache as the leading complaint, often accompanied by papilledema, reduced visual acuity or other visual disturbances, and tinnitus[2]. Tinnitus is frequently pulsatile and may reflect hemodynamic disturbance associated with collateral flow or jet-like flow across the stenosis that impacts the adjacent sigmoid sinus and its bony wall, potentially contributing to sigmoid sinus wall dehiscence (SSWD) and sigmoid sinus diverticulum (SSD).

IJVS most often reflects extrinsic compression, particularly by the styloid process or the C1 transverse process; intrinsic contributors such as jugular valve abnormalities have also been described. Intracranial pressure is normal or only mildly elevated in most patients with IJVS. A typical clinical profile consists of recurrent or persistent neuro-otologic and neurocognitive complaints, including tinnitus cerebri, tinnitus, sleep disturbance, dizziness, headache, cognitive difficulties, neck discomfort, and autonomic symptoms, with tinnitus cerebri and sleep disturbance reported particularly frequently; a minority may develop visual impairment or papilledema. IJVS has also been reported in association with several central nervous system disorders, including transient global amnesia, transient monocular blindness, primary exertional headache, and Parkinson's disease.

DIAGNOSTIC EVALUATION OF CVSS AND IJVS

Evaluation for suspected CVSS or IJVS should follow a structured pathway centered on venous imaging, accompanied by systematic assessment of intracranial hypertension and visual involvement. When establishing hemodynamic significance is required to guide subsequent treatment decisions, DSA-based venography with manometry is recommended.

Noninvasive imaging assessment

On non-contrast head CT/MRI, most patients with CVSS show no specific abnormalities; nonetheless, ancillary features suggestive of raised intracranial pressure should be reviewed, including empty sella, posterior globe flattening, optic nerve sheath distension/tortuosity, and optic disc protrusion. MR black-blood thrombus imaging technique (MRBTI) facilitates differentiation of acute/subacute intraluminal thrombosis and identification of arachnoid granulations, thereby supporting discrimination among CVSS, chronic thrombosis, and hypoplasia. In tinnitus-dominant presentations, mastoid/temporal bone CT can help evaluate SSWD and SSD. Basic audiovestibular testing (e.g., pure-tone audiometry, vestibular function testing, and auditory evoked potentials) is also recommended to exclude inner-ear or vestibular disorders.

Contrast-enhanced MRV delineates venous morphology and cross-sectional area and represents a preferred modality for diagnosis and follow-up in both CVSS and IJVS. Jugular venous Doppler ultrasound enables real-time, dynamic assessment of internal jugular vein (IJV) lumen caliber, flow parameters, and valvular reflux. Measurements are recommended during quiet respiration, using the maximal diameter at end-expiration, with bilateral comparison and systematic evaluation across the J1-J3 segments. A Valsalva maneuver may be added when needed to further assess stenosis severity and valve function. Head-neck CTV can further characterizes stenosis morphology and collateral compensation, and clarifies the anatomic relationship between the IJV and the C1 transverse process or styloid process. CTV is often advantageous when bony compression is suspected.

Invasive imaging assessment

Digital subtraction angiography (DSA) remains the reference standard for diagnosing CVSS. Direct retrograde cerebral venography with manometry (DRCVM) provides key information on stenosis severity, collateral drainage, and trans-stenotic pressure gradients. This assessment is recommended when noninvasive imaging cannot adequately define the stenosis or collateral compensation, or when endovascular therapy is being considered. DSA also helps exclude alternative causes such as arteriovenous malformations and dural arteriovenous fistulas. When MRV/CTV demonstrates bilateral filling defects at the transverse–sigmoid junction, or unilateral transverse and/or sigmoid sinus hypoplasia accompanied by contralateral filling defects, early DSA with manometry should be considered. Intravascular ultrasound may be used as an adjunct in selected cases to better characterize intraluminal pathology and support intra-procedural decision-making.

Intracranial hypertension and ophthalmologic assessment

In patients with suspected CVSS, after contraindications are excluded, lumbar puncture should be performed to measure the CSF opening pressure and obtain CSF studies to rule out secondary etiologies. CSF biochemistry and cytology are typically non-specific in CVSS, whereas the opening pressure is often elevated. For CVSS patients with intracranial hypertension, a comprehensive ophthalmologic evaluation is recommended, including visual acuity, visual fields, fundus examination with assessment of papilledema, and optical coherence tomography (OCT). Optic nerve sheath ultrasound may be added when available. A similar ophthalmologic assessment is also recommended routinely in IJVS to support risk stratification and evaluation of treatment response.

TREATMENT AND MANAGEMENT OF CVSS/IJVS

Management of CVSS and IJVS should be individualized and stepwise.

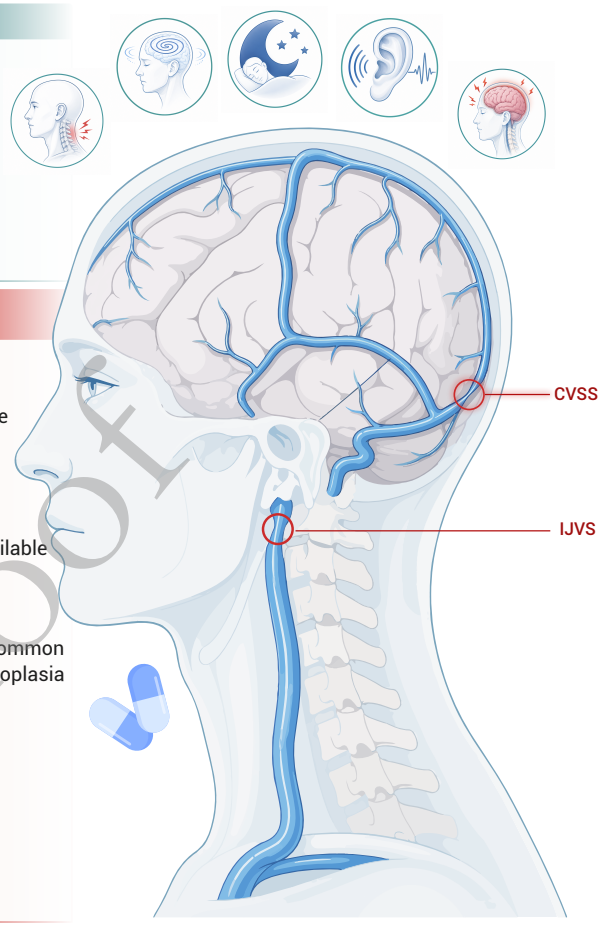
Diagnosis and Management of CVSS and IJVS

① When to suspect CVSS/IJVS

Non-specific neurological, otologic, or ophthalmologic symptoms

CVSS-dominant phenotype
- Headache
- Papilledema
- Visual disturbance
- Pulsatile tinnitus

IJVS-dominant phenotype
- Tinnitus cerebri / tinnitus
- Sleep disturbance
- Dizziness
- Headache
- Cognitive complaints
- Neck discomfort



② Diagnostic evaluation and lesion characterization

Noninvasive venous imaging
- CE-MRV
- Head-neck CTV
- Jugular Venous Doppler ultrasound
- MRBTI
- Temporal bone CT for tinnitus-dominant presentations
- Audiovestibular tests when indicated

Intracranial hypertension and ophthalmologic assessment
- Lumbar puncture / CSF opening pressure
- Visual acuity
- Visual field
- Fundus examination
- OCT
- Optic nerve sheath ultrasound when available

Hemodynamic confirmation
- DSA-based venography
- Manometry
- Venous pressure gradient
- Collateral drainage assessment
- IVUS in selected cases

Lesion characterization

CVSS
- Transverse–sigmoid sinus stenosis is common
- Arachnoid granulation, thrombus, or hypoplasia
- Collateral flow / jet-like flow
- Raised intracranial pressure phenotype

IJVS
- Internal jugular vein stenosis
- Styloid process compression
- C1 transverse process compression
- Jugular valve abnormality
- Collateral venous drainage

Decision before intervention
- Symptom–imaging concordance
- Hemodynamic significance
- Individual visual risk and symptom burden

③ Stepwise management and follow-up

Endovascular therapy
- Venous sinus stenting for selected CVSS
- Selected IJVS intervention when hemodynamically significant
- VPG-guided decision-making
- Peri-procedural antithrombotic therapy

Medical therapy
- ICP-lowering therapy
- Symptom control
- Anticoagulation if thrombosis or hypercoagulable state is present

Surgical and other interventions
- Styloidectomy or C1 transverse process decompression for extrinsic IJVS
- Sigmoid sinus wall reconstruction for selected tinnitus-related lesions
- Optic nerve sheath fenestration for acute or progressive visual compromise

Follow-up and surveillance
- Clinical reassessment
- Repeat venous imaging within 6–12 months
- Ophthalmologic follow-up when indicated
- Reassessment and individualized management

Core principle: Intervention should be considered when symptoms, anatomical lesions, and hemodynamic significance are concordant.

CE-MRV: contrast-enhanced magnetic resonance venography
CVT:cerebral venous thrombosis
ICP: intracranial pressure
MRBTI: magnetic resonance black-blood thrombus imaging
VPG: venous pressure gradient.
CSF: cerebrospinal fluid
CTV: computed tomography venography
IJVS: internal jugular vein stenosis
OCT: optical coherence tomography
CVSS: cerebral venous sinus stenosis
DSA: digital subtraction angiography
IVUS: intravascular ultrasound
ONSF: optic nerve sheath fenestration

Figure 1. XX

Medical therapy targets intracranial hypertension and symptom control. Endovascular or surgical interventions should be considered when a dominant outflow lesion is identified and hemodynamic significance is established, particularly in patients with visual risk or disabling symptoms.

Medical therapy

Intracranial pressure–lowering therapies. The carbonic anhydrase inhibitor acetazolamide reduces CSF production and has a diuretic effect. It appears more beneficial for mild visual decline and severe papilledema, whereas improvement in headache is less consistent. Topiramate has antimigraine properties, may promote weight loss, and has mild carbonic anhydrase–inhibitory activity. Other dehydration/diuretic agents can be used as adjuncts, including furosemide, hydrochlorothiazide, and potassium-sparing diuretics, although overall efficacy is often less robust than that of acetazolamide or topiramate. Hypertonic glycerol and mannitol may also help reduce intracranial pressure and venous hypertension.

Anticoagulation. There is currently no specific effective pharmacotherapy for IJVS. When a hypercoagulable state is present with intracranial or cervical venous thrombosis, timely anticoagulation is recommended.

Endovascular therapy

For patients with imaging evidence of focal CVSS, endovascular evaluation and treatment should be considered when any of the following clinical scenarios are present: (1) intracranial hypertension with papilledema and/or visual involvement[3]; (2) PT that severely impairs quality of life, after high jugular bulb and sigmoid sinus wall abnormalities have been excluded as the dominant cause; (3) chronic thrombus accompanied by symptoms of intracranial hypertension. In these settings, DRCVM is recommended to assess the venous pressure gradient (VPG). When manometry confirms that the stenosis is hemodynamically significant and concordant with the symptom profile, endovascular therapy can be considered, with venous sinus stenting preferred. VPG thresholds are not uniform. A cutoff of ≥10 mmHg is commonly used as a reference. In patients in whom alternative etiologies have been excluded and intracranial hypertension is strongly suspected to be primarily attributable to focal CVSS, a cutoff of ≥4 mmHg may also be considered.

For IJVS, stenting may be considered when a VPG is present across the stenotic segment. However, the optimal VPG threshold remains uncertain and requires further study. Because general anesthesia may reduce the measured pressure gradient, venography and manometry performed under local anesthesia are preferred when accurate VPG determination is required.

Peri-procedural medication. Prior to venous sinus stenting, anticoagulation or oral antiplatelet therapy with aspirin 100 mg once daily plus clopidogrel 75 mg once daily is recommended for at least 3 days. After stenting, antiplatelet therapy is recommended for 6–12 months or longer.

Surgical and other interventions

Bony decompression and other operations. In IJVS caused by confirmed extrinsic compression, resection of an elongated C1 transverse process or styloid process can be performed alone or in combination to relieve compression, restore internal jugular venous flow, and alleviate clinical symptoms[4]. Decompressive surgery may also reduce complications associated with internal jugular vein stenting. In selected cases, resection of perijugular muscles or other surgical approaches has been reported[5]. However, the current evidence is largely limited to isolated case reports or small series, and further clinical evaluation is needed to define efficacy, safety, and appropriate patient selection.

Sigmoid sinus wall reconstruction. For patients with PT or tinnitus cerebri, when a focal sigmoid sinus bony wall defect with an isolated SSD is confirmed as the symptomatic substrate, sigmoid sinus wall reconstruction may be considered after the dominant venous outflow lesion has been addressed.

Optic nerve sheath fenestration (ONSF). In CVSS and in a small subset of IJVS with intracranial hypertension causing acute, sudden visual loss or progressive visual decline, ONSF may be considered after comprehensive neuro-ophthalmologic evaluation.

Follow-up and surveillance

After endovascular or surgical interventions, regular clinical follow-up and repeat venous imaging are recommended within 6–12 months. In patients with intracranial hypertension or visual involvement, follow-up should include ophthalmologic assessments such as fundus examination, optic nerve sheath ultrasound, and OCT to evaluate response and detect recurrence.

Compared with arterial cerebrovascular disease, evidence for venous reflux disorders of head and neck—particularly CVSS and IJVS—remains limited. This guideline is therefore based mainly on case reports, case series, and observational cohorts, together with multidisciplinary expert consensus. Recommendations should be applied in the context of individual risk (especially visual involvement and intracranial hypertension) and concordant imaging/hemodynamic findings, with reassessment during follow-up. As prospective studies and standardized evaluation frameworks evolve, this guideline will be updated to incorporate new evidence.

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AUTHOR CONTRIBUTIONS

C.J.W., C.Z., and X.M.J. were responsible for conceptualization and supervision. L.L. was responsible for methodology, data curation, formal analysis, and drafting of the manuscript. H.L., C.H.L., J.W.G., Y.Z., and X.L.Z. contributed to critical revision, project administration, and resource allocation. All authors contributed to and approved the final manuscript.

DECLARATION OF INTERESTS

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