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Clinical outcomes of middle fossa craniotomy for superior semicircular canal dehiscence repair

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OBJECTIVE Superior semicircular canal dehiscence (SSCD) is a rare disorder characterized by the formation of a third opening in the inner ear between the superior semicircular canal and the middle cranial fossa. Aberrant communication through this opening causes a syndrome of hearing loss, pulsatile tinnitus, disequilibrium, and autophony. This study analyzed the clinical outcomes of a single-institution series of patients with SSCD undergoing surgical repair by the same otolaryngologist and neurosurgeon.

METHODS All patients who underwent SSCD repair at the University of California, Los Angeles, between March 2011 and November 2014 were included. All patients had their SSCD repaired via middle fossa craniotomy by the same otolaryngologist and neurosurgeon. Outcomes were analyzed with Fisher's exact test.

RESULTS A total of 18 patients with a mean age of 56.2 years (range 27–84 years) and an average follow-up of 5.0 months (range 0.2–21.8 months) underwent 21 cases of SSCD repair. Following treatment, all patients (100%) reported resolution in ≥ 1 symptom associated with SSCD. Autophony ($p = 0.0005$), tinnitus ($p = 0.0059$), and sound- and/or pressure-induced dizziness ($p = 0.0437$) showed significant symptomatic resolution. Following treatment, 29% (2/7) of patients developed imbalance, 20% (1/5) of patients developed sound- and/or pressure-induced dizziness, and 18% (2/11) of patients developed aural fullness. Among patients with improved symptoms following surgical repair, none reported recurrence of symptoms at subsequent follow-up visits.

CONCLUSIONS SSCD remains an underdiagnosed and undertreated condition. Surgical repair of SSCD using a middle fossa craniotomy is associated with a high rate of symptom resolution. Continued investigation using a larger patient cohort and longer-term follow-up could further demonstrate the effectiveness of using middle fossa craniotomy for SSCD repair.

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KEY WORDS superior semicircular canal dehiscence; vertigo; autophony; tinnitus; skull base

Vestibular and auditory symptoms can be caused by a dehiscence in any of the semicircular canals.[6] Under normal conditions, the inner ear is filled with fluid and is encased by dense otic capsule bone with only 2 mobile windows: the oval window and the round window. A dehiscence acquired in either the superior, posterior, or lateral canal can result in the creation of a third opening in the inner ear, causing a variety of symptoms.

A syndrome of vestibular and auditory dysfunction resulting from a dehiscence in the bone covering the superior semicircular canal was only recently identified.[6,16] Superior semicircular canal dehiscence (SSCD), which is more clinically common than the lateral and posterior forms, was first described in 1998 by Minor et al.[19] This rare disorder is characterized by a fistula between the superior semicircular canal and the temporal fossa. The aberrant communication between the superior semicircular canal and the middle fossa results in abnormal vestibular function that is sensitive to sound and intracranial pressure changes.[2] The creation of this "third mobile window" into the inner ear causes abnormal stimulation of the vestibular system and its ensuing auditory manifestations.[17]

ABBREVIATIONS SSCD = superior semicircular canal dehiscence.

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Possible vestibular symptoms of SSCD include oscillopsia, pressure-induced vertigo (Hennebert sign),[6] sound-induced vertigo, and chronic disequilibrium (Tullio phenomenon).[2,6,17] Common auditory symptoms include pulsatile tinnitus, hearing loss,[17] and aural fullness.[15] If the dehiscence develops close to the CSF space, a common clinical manifestation is autophony (i.e., the abnormal amplification of internal body sounds—self-generated sounds such as the heartbeat and eye movement).[21,24] Patients with SSCD have been known to experience vestibular or auditory symptoms either together or individually.[6] However, the physiological basis of why symptoms vary among patients is not yet fully understood.[15,17]

An estimated 1%–2% of the general population have an extremely thin bone overlying the superior canal. Yet in a study of asymptomatic subjects, Carey et al. found a 0.7% incidence of SSCD in a collection of randomly selected temporal bones from 596 cadavers.[5] However, incidental findings may occur in cases of asymptomatic patients following exposure of the temporal bone during surgery[4,12] or with cross-sectional imaging of the temporal bone.[9]

Current theories of the potential etiologies include developmental abnormalities, congenital defects, chronic otitis media with cholesteatoma, fibrous dysplasia, or a high-riding jugular bulb.[6] Yet, the cause of SSCD remains unclear in a majority of cases.[11]

The diagnosis of SSCD is based on a combination of clinical signs and symptoms, audiometric and vestibular testing, and high-resolution CT.[18,20] Some patients with SSCD are either asymptomatic or do not require treatment,[16] and the avoidance of triggers may be sufficient to prevent recurring symptoms.[4] However, for patients with debilitating symptoms, surgical repair may be an effective option.[16] Following surgery, most patients experience an improvement of sound- and/or pressure-induced vertigo, autophony, and hearing loss.[7,15,20] Although a number of published case series have investigated the outcomes of SSCD repair and have documented a high success rate, a majority of them focus on a particular symptom or a limited set of symptoms.[1,3,7,10,13,14,20,22] Without accounting for the entire spectrum of symptoms that patients could experience, many studies may not fully capture the possible outcomes of SSCD repair. Better knowledge regarding the effectiveness of surgical repair for SSCD is necessary for patients and physicians to make informed decisions regarding the management of this syndrome. The goal of this study was to assess the outcomes of SSCD repair. Herein, we present a series of patients from a single institution who underwent surgical repair of their SSCD by the same otolaryngologist and neurosurgeon. The procedure was followed by a detailed assessment of symptoms before and after surgery, as well as comparison with prior literature.

Methods

Selection of Patients

Patients who underwent surgical treatment for SSCD by the same otolaryngologist and neurosurgeon at Ronald Reagan University of California, Los Angeles (UCLA) Medical Center between March 2011 and November 2014 were retrospectively identified. Diagnosis of SSCD was based on patient presentation and audiological and vestibular evaluations, including vestibular-evoked myogenic potential response and Valsalva maneuvers, and confirmed with high-resolution CT imaging. The diagnosis of SSCD was made when both clinical signs and symptoms localized the dehiscence to a single side. Surgical repair was only offered to patients with debilitating auditory and vestibular symptoms that could not be adequately managed by avoiding triggers. Patients who presented with bilateral SSCD had their most symptomatic dehiscence repaired. Patients who received sequential SSCD surgery on their contralateral dehiscence were included in this study.

Collection and Analysis of Data

A comprehensive chart review was performed to collect patient demographic data and to assess clinical symptoms. The auditory and vestibular symptoms recorded included autophony, aural fullness, hearing loss, imbalance, sound- and/or pressure-induced dizziness, tinnitus, and headaches. Status of symptoms following treatment was based on subjective patient responses elicited at each follow-up. In our assessment of outcomes, we reviewed each patient's symptoms at the latest follow-up possible. For patients who received sequential SSCD surgery, their postoperative symptoms and follow-up period were not evaluated until after their second surgery. Categorical variables were compared using Fisher's exact test, with 2-tailed p values less than 0.05 considered statistically significant. All statistical analyses were performed using SPSS version 22.0 (IBM Corp.).

Surgical Technique

Plugging of the SSCD was performed through a middle fossa craniotomy, as previously described.[23] In summary, an approach through the temporal bone of the skull was used, using the zygomatic arch as the most inferior aspect of the craniotomy and the external auditory canal as one-third of the distance from the most anterior and two-thirds of the distance from the most posterior aspect of the craniotomy. High-magnification microdissection was then used to elevate the temporal lobe off the middle fossa floor to expose the arcuate eminence, and the canal dehiscence was identified on the petrous portion of the temporal bone. Once visualized, the dehiscence was directly plugged with temporalis fascia and bone wax and then sealed into place with fibrin glue.

Human Subjects Committee Approval

This study was approved by the Human Subjects Committee of the University of California, Los Angeles (UCLA) Human Research Protection Program.

Results

Patient Characteristics

Between March 2011 and November 2014, 21 cases of SSCD surgery were performed in 18 patients. Patients included 6 men (33%) and 12 women (67%) with a mean age of 56.2 years (range 27–84 years). The left ear was repaired in 9 patients (50%), the right ear was repaired in

6 patients (33%), and 3 patients (17%) had bilateral repair. Nine patients (50%) presented with bilateral SSCD. For patients who underwent sequential surgical treatment of their contralateral dehiscence, their surgeries were spaced a mean of 4.3 months (range 3.5–6 months) apart. The mean postoperative follow-up period was 5.0 months (range 0.2–21.8 months). Patient information is summarized in Tables 1 and 2.

Preoperative and Postoperative Symptoms

The most common preoperative symptoms that patients experienced upon presentation included autophony (89%), sound- and/or pressure-induced dizziness (72%), tinnitus (67%), imbalance (61%), and hearing loss (56%). The most common postoperative symptoms that patients continued to experience at last follow-up included sound- and/or pressure-induced dizziness (33%), autophony (28%), and imbalance (28%). Most patients reported a period of imbalance or dizziness that lasted for weeks following surgical treatment of their SSCD. A detailed summary of patient symptoms is provided in Table 3. When the preoperative group was compared with the postoperative group, autophony ($p = 0.0005$), sound- and/or pressure-induced dizziness ($p = 0.0437$), and tinnitus ($p = 0.0059$) all showed statistically significant improvements within the patient cohort (Table 4).

All patients (100%) who had their dehiscence plugged experienced resolution of ≥ 1 of their primary complaints (Table 5). Autophony, aural fullness, imbalance, and tinnitus showed a high degree of response to surgical repair, with symptomatic resolution in approximately ≥ 70% of patients. Headaches represented the symptom with the least response, with only 40% of patients reporting resolution after surgery. Among patients with significantly improved symptoms, none reported symptom recurrence. Seven patients developed symptoms following surgical repair. For patients who presented without preoperative imbalance or sound- and/or pressure-induced dizziness, 29% and 20%, respectively, developed imbalance or dizziness following treatment. For patients who presented without preoperative aural fullness, 18% developed aural fullness following treatment. In all individuals in whom postoperative symptoms developed, no patient developed > 1 new symptom after surgery.

TABLE 1. Summary of patient clinical and demographic information

Variable	No. (%)
Mean age in yrs, range	56.2, 27–84
Sex	
M	6 (33)
F	12 (67)
Bilat SSCD	9 (50)
Repair approach	
Rt	6 (33)
Lt	9 (50)
Bilat	3 (17)
Mean follow-up in mos, range	5.0, 0.2–21.8

TABLE 2. Individual characteristics of 18 patients with SSCD

Case No.	Age, Yrs	Sex	Bilat SSCD	Operated Side	Follow-Up (mos)
1	52	F	Yes	Lt	8.09
2	62	F	No	Rt	0.20
3	37	F	No	Rt	21.83
4	44	F	No	Lt	9.86
5	77	M	No	Rt	0.43
6	56	F	Yes	Rt	6.74
7	55	M	Yes	Bilat	5.79
8	57	F	Yes	Bilat	0.33
9	84	M	No	Lt	6.48
10	57	F	Yes	Lt	6.05
11	55	F	No	Lt	3.06
12	64	F	Yes	Bilat	3.81
13	65	M	No	Lt	3.02
14	69	M	No	Rt	3.45
15	56	F	Yes	Lt	4.04
16	32	M	No	Rt	3.95
17	27	F	Yes	Lt	0.36
18	55	F	Yes	Lt	1.68

Discussion

In our cohort of patients who underwent surgical treatment for SSCD, all patients (100%) showed improvement of their clinical symptoms. Except for an initial period of imbalance and dizziness after surgery, most patients reported an immediate improvement in their symptoms following treatment. This result is similar to what has been previously reported.[1,15] The strength of our study lies in the fact that the same 2 surgeons performed all surgical aspects of the dehiscence repair for all patients in our cohort. This allowed the surgery to be more standardized among patients, by reducing intraoperative variability and limiting the effect of the surgeon's own technical expertise on the surgical outcome. To our knowledge, this is the largest review of a single-institution series in which the entire cohort was operated on by the same key individuals. Additionally, our meticulous reporting of symptoms before and after surgical repair provides an insight into the degree of symptom resolution that can be expected and identifies the specific instances where symptoms developed postoperatively.

Symptom Resolution

Most symptoms of SSCD showed a high degree of resolution following surgery, with autophony, aural fullness, imbalance, and tinnitus being completely resolved in approximately ≥ 70% of our patients. Our results are very similar to what has been previously reported by Crane et al., who noted that autophony resolved in 72% of their study cohort.[7] Other studies also support the effectiveness of surgical plugging as a treatment modality for SSCD repair.[1,4,7,10,13–15,20,22] Our overall rate of symptom resolution (100%) is similar to the range of 72%–100% that has been reported in the literature (Table 6).[1,3,7,10,13,14,20,22] The

TABLE 3. Patients' symptoms before and after repair

Case No.	Autophony		Aural Fullness		Hearing Loss		Imbalance		Sound- /Pressure-Dizziness		Tinnitus		Headache	
	Preop	Postop	Preop	Postop	Preop	Postop	Preop	Postop	Preop	Postop	Preop	Postop	Preop	Postop
1	●	○	●	○	●	○	●	●	●	●	●	○	●	●
2	—	—	—	—	—	—	—	—	—	—	—	—	●	○
3	●	○	—	—	—	—	●	●	●	○	●	○	●	●
4	●	○	●	○	●	○	●	○	●	○	●	○	—	—
5	●	○	—	—	●	○	—	▲	●	○	●	○	—	—
6	●	●	—	—	●	●	●	●	—	▲	●	○	●	●
7	●	○	—	—	●	●	—	—	●	●	●	○	—	▲
8	●	○	—	—	●	○	●	○	●	○	—	—	—	—
9	●	●	—	—	●	○	●	○	—	—	●	●	—	—
10	●	○	●	●	—	—	●	○	●	○	—	—	—	—
11	●	●	—	—	—	—	●	○	●	●	—	▲	—	—
12	●	●	●	○	●	○	—	—	—	—	●	●	—	—
13	—	—	●	○	●	●	●	○	—	—	—	—	—	—
14	●	○	●	○	●	●	—	▲	●	●	—	—	—	—
15	●	○	—	—	—	—	●	○	●	●	●	○	—	—
16	●	●	—	▲	—	—	—	—	●	○	●	○	●	○
17	●	○	—	▲	—	—	●	○	●	○	●	○	—	—
18	●	○	●	○	—	—	—	—	●	○	●	○	—	—

● = present; — = not present; ○ = resolved; ▲ = developed.

reporting of symptom resolution was not identical across all studies and hence cannot be directly compared. However, all reported studies demonstrated a high degree of improvement in symptoms following SSCD repair. Given that most symptoms responded to surgical repair, our study suggests that the symptoms of SSCD can be improved with surgery. By focusing solely on the complete resolution of symptoms, this study does not account for the fact that patients may have experienced partial relief. Our study shows that surgical repair can be considered an effective treatment option for individuals with debilitating symptoms due to SSCD and can result in dramatic improvement of symptoms that substantially affect quality of life.

Factors Associated With Worse Outcomes

Niesten et al. noted that among female patients who underwent surgical treatment for SSCD, a history of migraines, bilateral superior canal dehiscence, and a larger dehiscence diameter were associated with a prolonged recovery.[20] In our study, only a small number of patients experienced the development of auditory and vestibular symptoms following surgery. The most common of these symptoms was the development of imbalance in 2 patients and the development of sound- and/or pressure-induced dizziness in 1 patient. In the single case where dizziness developed, the patient presented with bilateral SSCD. The development of symptoms in this patient may be attributed to the presence of the contralateral dehiscence rather than a result of surgery. As long as a contralateral SSCD remains, it is difficult to attribute the cause of the vertigo to a specific ear without additional tests. However, none of the patients who developed imbalance after surgery had bilateral SSCD. This is in contrast to the study by Mikulec et al., who noted that the single episode of imbalance that developed after surgery in their case series was in an indi-

TABLE 4. Overall symptoms of patient cohort before and after repair

Symptom	Preop		Postop		p Value
	No. w/ (%)	No. w/o (%)	No. w/ (%)	No. w/o (%)	
Autophony	16 (89)	2 (11)	5 (28)	13 (72)	0.0005
Aural fullness	7 (39)	11 (61)	3 (17)	15 (83)	0.2642
Hearing loss	10 (56)	8 (44)	4 (22)	14 (78)	0.0858
Imbalance	11 (61)	7 (39)	5 (28)	13 (72)	0.0922
Sound &/or pressure dizziness	13 (72)	5 (28)	6 (33)	12 (67)	0.0437
Tinnitus	12 (67)	6 (33)	3 (17)	15 (83)	0.0059
Headache	5 (28)	13 (72)	4 (22)	14 (78)	1.0000

TABLE 5. Symptom status in patient cohort following repair of SSCD

Symptom	No. Resolved (%)	No. Unresolved (%)	No. Developed (%)
Autophony	11 (69)	5 (31)	0 (0)
Aural fullness	6 (86)	1 (14)	2 (18)
Hearing loss	6 (60)	4 (40)	0 (0)
Imbalance	8 (73)	3 (27)	2 (29)
Sound/pressure dizziness	8 (62)	5 (38)	1 (20)
Tinnitus	10 (83)	2 (17)	1 (17)
Headache	2 (40)	3 (60)	1 (8)
0 symptoms	0 (0)	0 (0)	11 (61)
≥1 symptom	18 (100)	12 (67)	7 (39)
≥2 symptoms	14 (78)	8 (44)	0 (0)

vidual who had bilateral SSCD.[14] Agrawal et al. reported a period of vestibular hypofunction in 38% of patients 1 week after surgical repair, which decreased to just 11% after 6 weeks.[1] Therefore, the development of imbalance in our patients may represent a transient event not captured within our follow-up. With the current sample size, we are unable to assess for an association between bilateral SSCD and development of auditory and vestibular symptoms following treatment. Our study cohort also included 3 women who presented with a history of migraines. These patients all experienced resolution of at least 3 of their symptoms, and none developed any additional symptoms following surgery. As a result, our study could not confirm an association between a history of migraines and worse outcomes following SSCD repair.

More than half of all individuals who reported having headaches prior to surgery continued to experience headaches after their SSCD treatment. Of the 5 patients in our study who reported preoperative headaches, surgical treatment of SSCD was associated with headache resolution in 2 of them. Of the 3 patients with continuing headaches, 2 had a history of head trauma that resulted in chronic headaches. The remaining patient, who did not have a history of head trauma, was treated with sequential bilateral SSCD surgery 4 months apart. This patient's mild headaches developed into severe, painful headaches following the second surgery. The multifactorial nature of headaches may be responsible for these observations. Furthermore, the length of time it takes for headaches to resolve after surgery may not have been adequately captured within the follow-up period for these patients. As a result, increased care and attention should be given to patients who present with headaches prior to surgery so that a realistic understanding may be reached for expectations in symptom resolution following surgery for SSCD repair.

Patients With Bilateral SSCD

In our cohort of 18 patients, 9 patients (50%) presented with bilateral SSCD. Such a high percentage of bilateral SSCD suggests a possible predisposition to developing SSCD in these patients. Although the etiology remains

TABLE 6. Summary of results from published studies of SSCD treated with surgery

Authors & Year	Approach	No. of Pts	Age, Yrs*	Follow-Up, Mos*	Outcomes
Agrawal et al., 2009	MF	42	NA	NA	16/42 (38%) pts w/ postop vestibular hypofunction at 1 wk; 11% at 6+ wks
Goddard & Wilkinson, 2014	MF	23	52.2	19.9	20/24 (83%) pts w/ partial or complete resolution in symptoms
Mikulec et al., 2005	MF	11	NA	NA	10/11 (91%) pts w/ partial or complete resolution in symptoms
Niesten et al., 2012	MF	33	43.0	28.7	33/33 (100%) pts w/ improvement in chief complaint
Beyea et al., 2012	TM	16	NA	NA	15/16 (94%) pts w/ partial or complete resolution in symptoms
Crane et al., 2010	MF	19	48.0	3.0	13/18 (72%) pts w/ complete resolution in autophony
Ward et al., 2012	MF	40	43.9	3.0 (median)	8/32 (25%) pts w/ mild high-frequency sensorineural hearing loss after 1+ mo
Limb et al., 2006	MF	29	44.0 (median)	NA	No significant differences in hearing before & after surgery
Present study	MF	18	56.2	5.0	18/18 (100%) pts w/ complete resolution in ≥1 symptom

MF = middle fossa craniotomy; NA = data not reported; pts = patients; TM = transmastoid.
* Values are expressed as the mean unless otherwise indicated.

unknown, possible developmental or congenital variation could result in the formation of an abnormally thin bony layer over the semicircular canals, which would predispose an individual to developing bilateral SSCD.[16] A precipitating event, such as a head injury or sudden increase in intracranial pressure, could then rupture this abnormally thin layer and form the dehiscence.[5] In a review of 850 patients with symptoms associated with canal dehiscence, Elmali et al. found that only 22% of individuals had bilateral dehiscence.[8] However, the study's inclusion criteria encompassed patients who presented with nonspecific vestibular and auditory symptoms associated with any canal dehiscence, including both the lateral and posterior forms of the syndrome.

Study Limitations

Limitations of this study include those inherent to a retrospective analysis of the experiences at a single institution. Additionally, reporting of symptoms may have varied from patient to patient. To reduce the impact of this variation, we focused solely on the resolution of the symptom in our review of outcomes. The relatively short length of follow-up assessment may have also influenced outcomes, because the improvement or development of symptoms may take months to fully manifest and stabilize following surgery.

Conclusions

This study investigated 21 cases of SSCD repair in 18 patients who received surgical treatment from the same otolaryngologist and neurosurgeon at a single institution. In reviewing the pre- and postoperative symptoms of the cohort, all patients demonstrated resolution of ≥ 1 of their symptoms following surgery, with autophony, aural fullness, imbalance, and tinnitus demonstrating the highest rates of resolution. Imbalance, dizziness, tinnitus, and aural fullness may also develop in previously asymptomatic individuals, and care should be taken by the physician and patient in understanding these risks. We found that preexisting headaches are less likely than other symptoms of SSCD to be resolved with surgery. In conclusion, we found that surgical repair of SSCD using a middle fossa craniotomy is associated with a high rate of symptom resolution. Continued investigation using a larger patient cohort and longer-term follow-up could further demonstrate the effectiveness of SSCD repair in symptomatic patients.

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Disclosures

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author Contributions

Conception and design: Yang, Gopen. Acquisition of data: Chung, Ung, Thill. Analysis and interpretation of data: Chung, Ung. Drafting the article: Chung. Critically revising the article: Chung, Ung, Spasic, Gopen. Reviewed submitted version of manuscript: Yang, Ung, Pelargos. Statistical analysis: Chung, Spasic. Study supervision: Yang, Nagasawa, Thill, Voth, Hirt, Gopen.

Supplemental Information

Previous Presentations

Portions of this work were presented in abstract form at the Western Student Medical Research Forum, Carmel, California, January 31, 2015.

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