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Original Article

Evaluating the Prevalence of Cardiac Surgery–associated Acute Kidney Injury After Septal Myectomy Combined With Concomitant Procedures in Obstructive Hypertrophic Cardiomyopathy

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Objectives: Hypertrophic obstructive cardiomyopathy (HOCM) may be treated by septal myectomy. Cardiac surgery–associated acute kidney injury (CSA-AKI) is a common complication, but little is known about its incidence after septal myectomy. The objectives of this work were to evaluate the prevalence of CSA-AKI after septal myectomy and identify potential perioperative and phenotype-related factors contributing to CSA-AKI.
Design: This was a retrospective database analysis with new data analysis.
Setting: The study occurred in a single university academic expertise center for septal myectomy HOCM patients.
Participants: Data from 238 HOCM patients with septal myectomy operated on between 2005 and 2022 were collected.
Interventions: CSA-AKI was stratified according to the Kidney Disease: Improving Global Outcomes (KDIGO) guidelines using measurement of creatinine and urine production. Important HOCM phenotype-related and perioperative factors were analyzed for their possible associations with CSA-AKI.
Measurements and Main Results: CSA-AKI occurred in 45% of patients; of these, 55% were classified as KDIGO stage I and the remaining 45% as stage II, with no chronic kidney damage observed. Moreover, there were no phenotypical or perioperative characteristics that were more prevalent in the CSA-AKI cohort. However, the use of beta-blockers and coronary artery disease were more prevalent in the CSA-AKI cohort.
Conclusions: CSA-AKI is a common complication after septal myectomy but was transient, and kidney function recovered in all patients.

Key Words: hypertrophic cardiomyopathy; myectomy; acute kidney injury; perioperative risk; cardiac surgery; beta-blockers

Hypertrophic cardiomyopathy (HCM) is one of the most common inherited cardiac diseases, affecting approximately 1 in 200 to 500 people worldwide.[1,2] It is characterized by left ventricular hypertrophy in the absence or in excess of other abnormal loading conditions such as hypertension or aortic valve disease. Approximately two-thirds of HCM patients develop obstructive HCM (HOCM).[1,3] HOCM patients with a

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persistent left ventricular outflow tract obstruction of ≥50 mmHg, in combination with symptoms defined by a New York Heart Association (NYHA) class III-IV (although in specific circumstances, patients with NYHA class II or those with recurrent syncope may be operated upon) despite medical therapy, may be considered for septal myectomy.[1,4] Septal myectomies necessitate the administration of cardioplegia and the use of cardiopulmonary bypass (CPB). Given the previously established association between HCM and end-stage kidney disease,[5] it is critical to examine whether there is also a correlation with cardiac surgery–associated acute kidney injury (CSA-AKI). There is a notable lack of data on the incidence of CSA-AKI following surgical myectomy, which underscores the need for additional research on its prevalence after this surgery.

CSA-AKI is a common complication after cardiac surgery and has an incidence that varies between 5% and 42%.[6] Following cardiac surgery, CSA-AKI is independently associated with increased morbidity and mortality, and prolonged hospital admissions.[7,8] Severe CSA-AKI may increase the duration of intensive care unit admissions, which results in increased mortality and costs of care.[9] Even a small increase in serum creatinine (sCr) after cardiac surgery may be independently associated with a significant increase in 30-day mortality.[10] Regardless of whether there is complete renal recovery or not, the 10-year mortality associated with AKI after cardiac surgery in general remains increased.[11] The pathophysiological pathways that result in CSA-AKI remain incompletely understood and complex, potentially involving multiple pathways at once.[6] Notably, during the intraoperative phase, the predominant contributing factors include hemodynamic instability, hypo- or hypervolemia, nephrotoxic medications, anemia, CPB, reperfusion injury, and inflammation[12,13]. These injury mechanisms can occur to varying degrees in any patient during the intraoperative period.[14]

Moreover, the longer the duration of CSA-AKI, the worse the prognosis. Studies show that CSA-AKI lasting more than 1 week has higher mortality rates, and early renal recovery significantly boosts 1-year survival chances, especially with rapid sCr decrease.[15,16] Therefore, evaluating whether CSA-AKI is prevalent after septal myectomy could lay the groundwork for future studies evaluating possible septal myectomy-specific CSA-AKI-predictive strategies. These can include personalized blood pressure management, the use of balanced fluids, biocompatible circuit coatings, and lung-protective ventilation.[9] Therefore, predicting CSA-AKI in a timely manner could enable the implementation of effective protective measures, which, in turn, may lead to quicker recognition and more efficient prevention and rapid treatment of CSA-AKI.

Moreover, the underlying pathophysiology of CSA-AKI remains incompletely understood and, thus, HOCM-specific factors may play a role in the development of CSA-AKI. Specifically, the significant phenotypical variability in patients with HOCM revealed in previous studies might play a role.[17-19] The aims of this study were to analyze the incidence of CSA-AKI in patients receiving septal myectomy, and evaluate which phenotype-specific and perioperative factors may contribute to the development of CSA-AKI.

Materials and Methods

Study Design and Primary Outcome

A retrospective analysis was performed utilizing previously acquired data from the existing inherited cardiomyopathy database of a single HCM expertise center (Erasmus Medical Center, Rotterdam, the Netherlands). All analyses were conducted with previous written informed consent and approval by the local institutional review board (MEC-2021-0781). For the primary outcome, electronic patient dossier analysis was performed to identify and quantify CSA-AKI, which was stratified using the validated Kidney Disease: Improving Global Outcomes (KDIGO) guideline scoring system.[16] These criteria define AKI as either a urine output of <0.5 mL/kg/h for 6 hours, and/or a rise in sCr by ≥26.5 μmol/L within 48 hours, or an increase of sCr values to 1.5 times the baseline. The KDIGO criteria categorize AKI into three stages based on these changes in sCr levels or urine production (KDIGO 1-3).[20] In this study, the preoperative sCr level from blood draws the day prior, or otherwise within 1 month prior to operation; and the highest postoperative sCr level within 72 hours were recorded. Additionally, urine production (in mL, per 1- to 4-hour intervals, as measured and reported with indwelling catheter measurement on the intensive and high care unit) within 24 hours postoperation was measured.

Study Population

All identified individuals in the database between the ages of 16 and 80 years diagnosed with HCM were eligible for inclusion in the study. Patients with HCM phenocopies (eg, patients with left ventricular hypertrophy secondary to an aortic stenosis or syndromal phenocopies such as Noonan syndrome or Fabry disease) who underwent septal myectomy were excluded from the analysis, as were those undergoing an emergency operation (where severe acute illness would confound the findings). Any patients with missing data necessary for the quantification of CSA-AKI were excluded. Other missing data was considered missing and not imputed. As perioperative data prior to 2005 was unavailable, only patients operated on between 2005 and 2022 were included.

Secondary Outcomes

Secondary analyses were conducted to identify perioperative and patient-related factors associated with the prediction of CSA-AKI. Long-term outcomes, including the development of chronic kidney disease and 90-day and 1-year mortality rates, were described. Perioperative parameters, such as blood loss, time of asystole, CPB and aortic clamping times, were measured. The duration of admission on the intensive care unit and time until stable hospital discharge were noted. Baseline characteristics were recorded, including history of atrial

fibrillation (irrespective of the rhythm just prior to operation), HCM-related medication use (beta-blockers, nondihydropyridine calcium channel blockers, and disopyramide), and echocardiographic parameters prior to operation (intraventricular septal thickness, maximal left ventricular outflow tract [LVOT] gradient measured in mmHg, left atrial diameter, left ventricular ejection fraction stratified in 4 stages: ≥60%, 50-59%, 36-49%, and ≤35%; diastolic dysfunction graded from grades 1 to 3). Mitral regurgitation was measured semiquantitatively (grades 0 to 4) according to the standard guidelines.[21,22] Patient genotype (defined using the 2015 American College of Medical Genetics and Genomics/Association for Molecular Pathology [ACMG/AMP] guidelines)[23] was also analyzed for possible differences in CSA-AKI incidence.

Cardiopulmonary Bypass

The CPB circuit used by the Erasmus Medical Center, for the last decade is the Quadrox HMOD 71000 with an integrated Quart HBF140 arterial filter, a venous hard-shell cardiotomy reservoir, and the Revolution pump for the heart-lung machine, Stöckert S5 (Sorin Group, Saluggia, Italy). All surfaces were coated with Safeline coating (Maquet Cardiopulmonary, Rastatt, Germany) or PHYSIO coating (Sorin Group). The priming solution consisted of Gelofusine (B.Braun, Melsungen, Germany) and mannitol 200 g/L (Baxter Healthcare, Utrecht, the Netherlands). The initial heparin dosage (Leo Pharmaceuticals, Weesp, the Netherlands) was 300 IU/kg of body weight, with an additional 7,500 IU in the CPB circuit prime solution. Anticoagulation through active clotting time (ACT) was monitored using the Hemochron® Jr. (J.T.C. Europe, Rodano, Italy). Values >440 seconds were considered safe for CPB. Oxygenation was regulated using in-line blood gas monitoring (CDI-500, Terumo Corporation, Tokyo, Japan) in the α-stat method, combined with a mass flow meter (Brooks Instruments, Hatfield, PA, USA). Intraoperative hemodynamic management targeted an arterial nonpulsatile flow of 2.4 L/min/m^2 and mean arterial pressure >60 mmHg. The targeted value for PaO_2 during the CPB time was 150 mmHg. Cardioplegia was induced by the administration of St. Thomas Hospital solution. There were no adjustments based on age and flow.

Statistical Analysis

Statistical analysis was conducted using IBM SPSS Statistics version 28 (SPSS, IBM, Armonk, NY, USA). Baseline characteristics and secondary outcomes were analyzed using the chi-square (χ^2) test for categorical data, the independent-samples *t*-test or analysis of variance for normally distributed continuous data, and the Mann-Whitney U or Kruskal-Wallis H test for non-normally distributed continuous data. The normality of the data was assessed using the Kolmogorov-Smirnov test. Data displaying a normal distribution are reported using means and standard deviations, and medians with interquartile ranges are used for data not conforming to the normal distribution. Statistical significance was assumed for $p < 0.05$.

Results

Between 2005 and 2022, 330 patients who had provided written informed consent for inclusion in the database and undergone an elective septal myectomy were retrospectively identified. Of these, 92 patients were excluded (Fig 1): 77 due to missing data, 7 due to being under 16 years of age, 4 due to emergency operation, and 4 phenocopies. Table 1 displays demographic characteristics of this septal myectomy cohort. The analyzed cohort of 238 patients was predominantly male, and BMI was significantly higher in the CSA-AKI cohort ($p < 0.001$). Males were significantly younger than females at the time of myectomy (median age 54.0 years *v* 61.5 years, $p < 0.001$). Most patients (85%) were in NYHA class III, and 32% carried a pathogenic HCM-related DNA variant. The most prevalent comorbidity was arterial hypertension (37%), followed by atrial fibrillation (21%), coronary artery disease (CAD; 9%), and diabetes mellitus (7%). Prior to surgery, 77% of patients used beta-blockers, 30% used verapamil or diltiazem, and 3% used disopyramide. Isolated septal myectomy was infrequent (10%), with a majority of patients (80%) receiving a concomitant mitral valve repair via autologous pericardial patch due to preoperative mitral valve morphologies, 2% of patients receiving a mitral valve replacement, and 8% undergoing an additional procedure (eg, single-graft coronary artery bypass graft, Cox maze procedure). No patients died within 90 days after surgery and the overall 1-year postoperative mortality rate was less than 1% (2 patients).

Echocardiographic analysis showed a mean intraventricular septal diameter of 20 ± 6 mm, a mean LVOT gradient of 89 ± 33 mmHg, and a mean left atrial diameter of 46 ± 8 mm. Most patients had mild mitral regurgitation (50%), with 26% having moderate and 8% having severe mitral regurgitation. Normal left ventricular function was determined in 89% of patients, and 11% had mild-to-moderate left ventricular dysfunction. Furthermore, assessment of diastolic function showed that the majority (60%) had stage 2 or 3 diastolic dysfunction. Notably, none of the echocardiographic measurements differed significantly between the patients who developed CSA-AKI and those who did not. Furthermore, the incidence of CAD was higher in the CSA-AKI group (14% *v* 5%, $p = 0.011$). The use of beta-blockers was significantly higher in the CSA-AKI patient cohort (87% *v* 69%, $p = 0.001$). No other phenotype-

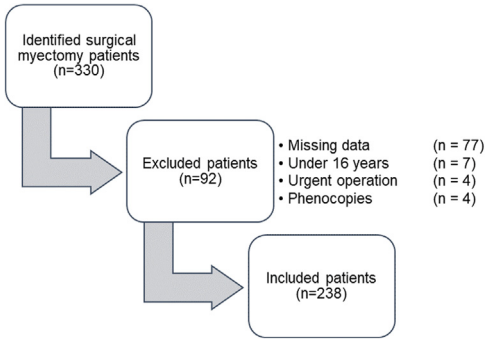

Fig. 1. Patient inclusion flowchart.

Table 1
Clinical, Demographic, and Laboratory Characteristics of Patient Cohort Stratified by Incidence of Cardiac Surgery–associated Acute Kidney Injury

Characteristic	All Patients (n = 238)	Non-AKI (n = 131)	CSA-AKI (n = 107)	p-Value
Demographics				
Male, %	61	63	60	0.770
Age at operation, years (IQR)	57 (46-66)	56 (46-65)	58 (47-68)	0.163
BMI, kg/m^2 ± SD	28.0 ± 3.8	27.3 ± 3.5	29.0 ± 4.1	<.001
90-day mortality rate, %	0	0	0	-
365-day mortality rate, n (%)	2 (0.8)	0	2 (1.9)	0.038
ICD, n (%)	17 (7)	8 (6)	9 (8)	0.492
Genotype positive, %	32	34	30	0.545
Comorbidities, %				
Hypertension	37	34	40	0.435
Diabetes mellitus	7	6	8	0.675
Coronary artery disease	9	5	14	0.011
Atrial fibrillation	21	17	25	0.109
Medications, %				
Beta-blockers	77	69	87	0.001
Verapamil/diltiazem	30	32	28	0.501
Disopyramide	3	2	5	0.153

NOTE. Values expressed as frequency (%), mean ± standard deviation, or median and interquartile range.
Abbreviations: BMI, body mass index; CSA-AKI, cardiac surgery–associated acute kidney injury; ICD, implantable cardioverter defibrillator; IQR, interquartile range; SD, standard deviation.

related factors were associated with CSA-AKI. This data is portrayed in further detailed in Tables 1 and 2.

In total, 107 patients (45%) developed CSA-AKI, of whom 59 developed KDIGO stage 1 CSA-AKI (55%), while 48 developed KDIGO stage 2 CSA-AKI (45%). No patients developed KDIGO stage 3 CSA-AKI. The mean urine production for the non-CSA-AKI group was 117 ± 207 mL per hour. For KDIGO stage 1 and 2, it was 80 ± 25 and 59 ± 19 mL per hour, respectively (p < 0.001). Patients who underwent a concomitant procedure along with septal myectomy did not have a significantly higher risk of developing CSA-AKI compared with those who only underwent an isolated septal myectomy (56% *v* 48%, p = 0.464). Patients that were operated on in recent years showed a higher prevalence of CSA-AKI (31% during 2005-2010 *v* 42% during 2011-2016 *v* 54% during 2017-2022, p = 0.018). The median age at the time of operation increased in the later time periods (during 2005-2010: 52 years [(45-60], during 2011-2016: 58 years [46-66] and during 2017-2022: 58 years [49-67], p = 0.042). Furthermore, no differences were found in perioperative characteristics, including CPB time, operation time, blood loss, and nonautologous blood transfusion, between the AKI and non-AKI groups. Further details are provided in Table 3.

Table 2
Echocardiographic Characteristics of Patient Cohort Stratified by Incidence of Cardiac Surgery-associated Acute Kidney Injury

Characteristic	All Patients (n = 238)	Non-AKI (n = 131)	CSA-AKI (n = 107)	p-Value
Intraventricular septum diameter, mm	19.9 ± 5.8	19.9 ± 6.5	19.9 ± 4.8	0.974
LVOT gradient, mmHg	89 ± 33	86 ± 32	92 ± 34	0.267
Left atrial diameter, mm	46.4 ± 8.1	45.8 ± 8.7	47.2 ± 7.4	0.202
Mitral regurgitation, %				0.580
Grade 0	15	16	14	
Grades 1-2	50	49	52	
Grade 3	26	25	28	
Grade 4	9	10	6	
Left ventricular function, %				0.392
≥60%	89	92	88	
50-59%	10	8	11	
36-49%	1	0	1	
≤35%	0	0	0	
Diastolic dysfunction*, %				0.881
Stage 1	39	40	37	
Stage 2	57	56	59	
Stage 3	4	4	4	

NOTE. Values expressed frequency (%) or mean ± standard deviation.
Abbreviations: CSA-AKI, cardiac surgery–associated acute kidney injury; LVOT, left ventricular outflow tract.
* Diastolic function parameters were only available in 208 (87%) of the cohort (114 [87%] in non-AKI group, 94 [88%] in CSA-AKI group).

The CSA-AKI cohort had a significantly higher 1-year mortality rate compared with the non-CSA-AKI group (2% *v* 0%, p = 0.038), however, highlighting that only two patients died within 1 year in the CSA-AKI group. Lastly, the occurrence of CSA-AKI was not associated with a longer median duration of intensive care unit admission (1 *v* 1 days, p = 0.150), however, it was associated with a significantly longer median time until discharge from the hospital (7 [range 3-43] *v* 6 [range 3-39] days, p = 0.012).

Discussion

The prevalence of CSA-AKI utilizing the KDIGO score[20] in the examined cohort of septal myectomy patients was 45%. Phenotype-specific cardiac and perioperative parameters did not significantly differ between groups. Because this is one of the only studies to primarily examine the prevalence of

Table 3
Perioperative Characteristics of Patient Cohort in Relation to Cardiac Surgery–associated Acute Kidney Injury

Characteristic	All Patients (n = 238)	Non-AKI (n = 131)	CSA-AKI (n = 107)	p-Value
Laboratory values				
Pre-op creatinine, μmol/L, mg/dL	85 ± 20 0.96 ± 0.22	84 ± 20 0.95 ± 0.22	87 ± 20 0.98 ± 0.23	0.238
Post-op creatinine, μmol/L, mg/dL	90 ± 26 1.02 ± 0.29	86 ± 22 0.97 ± 0.25	95 ± 30 1.08 ± 0.34	0.007
Delta creatinine, μmol/L, mg/dL	8.7 ± 12.4 0.11 ± 0.14	8.0 ± 5.9 0.09 ± 0.14	12.1 ± 17.2 0.14 ± 0.19	0.012
Pre-op urea, mmol/L	5.9 ± 1.6	5.9 ± 1.6	5.9 ± 1.7	0.762
Post-op urea, mmol/L	7.1 ± 2.5	6.8 ± 2.2	7.3 ± 2.9	0.202
Delta urea, mmol/L	1.8 ± 1.8	1.6 ± 1.5	1.9 ± 2.0	0.229
Pre-op glucose, mmol/L	6.0 ± 1.3	5.9 ± 1.2	6.1 ± 1.4	0.277
Perioperative characteristics				
Operation time, min	235 ± 56	235 ± 45	236 ± 67	0.434
CPB time, min	149 ± 46	148 ± 35	150 ± 57	0.355
Aortic clamping time, min	110 ± 37	109 ± 28	111 ± 46	0.424
CVP, mmHg	11.6 ± 3.6	11.5 ± 3.4	11.6 ± 3.9	0.981
≥3 Operative vasopressors, %	23 (10)	9 (7)	14 (13)	0.107
Intraoperative blood loss, mL	1,078 ± 664	1,113 ± 671	1,035 ± 657	0.458
Nonautologous blood transfusion, %	29	31	26	0.458
Heparin use, IU	32,500 (26,000-37,500)	30,000 (25,000-37,250)	32,500 (27,500-37,500)	0.005
Pre-op ACT, s	114 (18)	113 (16)	115 (20)	0.544
Maximal operative ACT, s	665 (160)	660 (160)	672 (160)	0.554
Post-op ACT, s	111 (19)	112 (14)	110 (25)	0.372
Mean urine production 24 h post-op, mL/h	96 ± 156	117 ± 207	71 ± 25	0.023

Values expressed frequency (%), mean ± standard deviation, or median (interquartile range).
Abbreviations: ACT, activated clotting times; CPB, cardiopulmonary bypass; CSA-AKI, cardiac surgery–associated acute kidney injury; CVP, central venous pressure; Post-op, postoperative; Pre-op, preoperative.

CSA-AKI in septal myectomy patients,[24] it is notable that this prevalence is at the upper end of the overall prevalence of CSA-AKI reported in literature.[6,25,26] However, only mild and moderate cases of CSA-AKI (corresponding to KDIGO stages 1 and 2) were observed, with no instances of severe AKI (KDIGO stage 3). Furthermore, none of the patients had renal failure or required (chronic) renal replacement therapy or intermittent dialysis. The addition of a concomitant procedure during septal myectomy was not associated with a higher incidence of CSA-AKI. However, whether HOCM itself was a risk factor for CSA-AKI could not be determined, and no evident explanation for the high incidence of CSA-AKI in this cohort was identified.

The use of beta-blockers was significantly higher in the CSA-AKI cohort. This finding was also observed in a prior study by O'Neal et al. examining a coronary artery bypass graft cohort.[27] They also found that the preoperative use of beta-blockers was associated with an increased chance of developing stage 2 CSA-AKI, even when accounting for potential confounders.[27] Beta-blockers are a mainstay in the treatment of HOCM and are primarily given for symptom relief.[1] However, there is no evidence in the literature that beta-blocker use has a causal relationship with CSA-AKI, and no explanation could be found in this study for the discrepancy in beta-blocker use between groups. Future research is warranted to investigate whether preoperative beta-blocker use is associated with an increased risk of CSA-AKI in this population. During the analysis of CSA-AKI prevalence, operation periods were stratified into three 6-year periods, with each successive period having a higher incidence of CSA-AKI. This may have multiple explanations. First, it may reflect better and more standardized reporting methods of perioperative data. This could suggest under-reporting of CSA-AKI during earlier years of operation. However, the age at the time of operation was older in each successive operation period. As CSA-AKI is more prevalent with increased age,[6] this may also explain the increasing incidence of CSA-AKI.

Future Perspectives

It is important to continue investigating the incidence of CSA-AKI and its associated risk factors, to understand their role in the pathophysiology of CSA-AKI.[28-30] Given that a new class of medications, cardiac myosin inhibitors such as mavacamten,[1,31] now exist for the treatment of HOCM, a better understanding of which patients may develop perioperative complications such as CSA-AKI may allow for more individualized decision-making when evaluating the best therapy option in a single HOCM patient.

Furthermore, additional studies are needed to identify the causes of the high incidence of CSA-AKI found in this cohort. Future research should determine whether there are

HOCM-specific factors that contribute to this considerable, although transient, amount of CSA-AKI. If this finding can be replicated, it highlights the need to better predict risk factors that may contribute to CSA-AKI development in septal myectomy patients. A potential perspective might be the development of a septal myectomy-specific CSA-AKI predictive risk model, which could enhance patient care and assist in predicting CSA-AKI.

Limitations

This study was conducted utilizing a retrospective dataset consisting of data collected during a 17-year collection period. One major limitation when analyzing CSA-AKI in a retrospective cohort is that urine production intervals were not standardized, which could have altered urine production measurements. This is especially worth mentioning since prior research has shown that the prevalence of CSA-AKI, as stratified by KDIGO score, can vary depending on the reporting of urine output and creatinine values.[32] However, the tight adherence to the KDIGO definition in this study should have reduced erroneous classification of CSA-AKI.

This retrospective single-center study showed that CSA-AKI was frequently observed following septal myectomy but was transient. Future research is essential to further understand the factors contributing to CSA-AKI in septal myectomy patients.

Data availability

The data underlying this article will be shared upon reasonable request to the corresponding author.

Declaration of competing interest

M. Michels reports speaker and consultant honoraria supported by BMS/Myokardia, Cytokinetics, and Pfizer, outside the submitted work. All other authors declare no relevant conflict of interests.

CRediT authorship contribution statement

Calvin J. de Wijs: Writing – review & editing, Writing – original draft, Supervision, Resources, Conceptualization. **Stephan A.C. Schoonvelde:** Writing – review & editing, Writing – original draft, Supervision, Resources, Conceptualization. **Egbert G. Mik:** Writing – review & editing, Supervision, Resources, Conceptualization. **Peter L. de Jong:** Writing – review & editing, Resources, Conceptualization. **Michelle Michels:** Writing – review & editing, Writing – original draft, Supervision, Resources, Conceptualization. **Floor A. Harms:** Writing – review & editing, Writing – original draft, Supervision, Resources, Conceptualization.

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