

Assessment of the Treatment for Alcohol Withdrawal Syndrome With and Without Dexmedetomidine: A Retrospective Cohort Study

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Abstract

Background: Alcohol withdrawal syndrome (AWS) carries a high risk of morbidity and mortality that often requires critical care admission and monitoring. Dexmedetomidine is a known sedative that can help with patient symptoms and potentially reduce Clinical Institute Withdrawal Assessment of Alcohol Scale Revised (CIWA-Ar) scores. This study looks at two groups - the use of dexmedetomidine versus non-dexmedetomidine - in the management of AWS patients to assess the length of intensive care unit (ICU) stay, length of stay (LOS) in hospital, mortality and readmission rate.

Methods: This retrospective cohort study was conducted at Geisinger Health System, a tertiary care, academic health care system, between January 2016 and December 2022. This study did not require ethics approval according to the Institutional Review Board (IRB). The patients assessed included those aged 18 years or older with a documented CIWA-Ar score requiring admission to the ICU or ICU step-down unit. Exclusion criteria included ICU admission for alternative indications, a prior history of seizure disorder, or patients who left against medical advice.

Results: A total of 994 patients were identified to have met the inclusion criteria; 371 patients were in the non-dexmedetomidine group and 623 in the dexmedetomidine group. Primary outcomes assessed were hospital LOS (5.0 (3.0 - 9.0) vs. 10 (6.0 - 16.0), $P < 0.0001$) and days in the ICU (1.9 (1.0 - 3.4) vs. 4.9 (2.9 - 9.0), $P < 0.0001$), respectively. Secondary outcomes assessed were lorazepam equivalent usage, and readmission within 30, 60 and 90 days, which were not statistically significant when accounting for ICU LOS.

Conclusions: Among patients treated for AWS, dexmedetomidine use appeared to be associated with longer hospital and ICU stays, while benzodiazepine requirements remained unchanged. These associations may reflect differences in illness severity, emphasizing the need for future prospective evaluation.

Keywords: Alcohol withdrawal syndrome; Alcohol withdrawal; Dexmedetomidine; Benzodiazepine

Introduction

Alcohol withdrawal syndrome (AWS) is a major contributor to morbidity and mortality in the critical care setting in addition to being responsible for a disproportionate fraction of healthcare resource utilization [1]. The constellation of symptoms associated with AWS has historically been the target of gamma-aminobutyric acid agonists (GABA), chiefly benzodiazepines, with the use of phenobarbital increasing recently [2]. However, the search for alternative and adjuvant therapies has intensified in recent years, particularly given the risks of benzodiazepine therapy in critically ill patients, namely respiratory depression. Dexmedetomidine, a centrally acting alpha-2 receptor agonist, has been frequently used given that AWS is hallmarked by symptoms associated with adrenergic excess such as anxiety, diaphoresis, tremulousness, and tachycardia.

Investigations regarding the efficacy and safety of dexmedetomidine in the treatment of AWS in the critical care setting have thus far yielded mixed results. For example, some studies found that patients treated with adjunctive dexmedetomidine required less benzodiazepines while still achieving adequate

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control of hypertension and tachycardia [3, 4]. Others have concluded that continuous dexmedetomidine infusion resulted in decreased rates of intubation and decreased length of hospital stay when compared to patients receiving propofol or lorazepam infusions [5]. On the other hand, dexmedetomidine was found to be effective in reducing Clinical Institute Withdrawal Assessment of Alcohol Scale Revised (CIWA-Ar) scores, but its use was associated with an increased length of hospital stay indicating that the drug may not be a cost-effective option [6]. Similarly, a recent study found that dexmedetomidine prolonged intensive care unit (ICU) stays but had no effect on CIWA-Ar scores [7]. This study also reported trends of increased seizures with dexmedetomidine adjunctive therapy, which is hypothesized to be due to the drug's lack of activity on GABA receptors [7]. As such, there is no clear consensus on the efficacy of dexmedetomidine in this context, which is additionally highlighted by the current lack of clinical guidelines for its use.

Given the extant gap in the literature regarding the use of dexmedetomidine in the critical care setting, this study seeks to further investigate the impact of its use in the treatment of AWS. We conducted a retrospective cohort study aimed at comparing patients with AWS who received dexmedetomidine in addition to standard therapies (e.g., benzodiazepines, phenobarbital) to those who received benzodiazepines or phenobarbital alone. The clinical outcomes considered included medication use, length of hospital and ICU stay, 30-day mortality, among others. In doing so, we hope to contribute to the growing body of literature aimed at characterizing the use of this novel agent in the treatment of AWS.

Materials and Methods

Study design

We conducted an organization wide, multi-hospital, retrospective cohort study of patients who were admitted to Geisinger Health System (GHS), Pennsylvania. This study was conducted in accordance with the principles of the Declaration of Helsinki. Due to its retrospective design and use of deidentified data, the requirement for informed consent and Institutional Review Board (IRB) approval was waived. Retrospective data collection was done for patients who were admitted to any of the hospitals within the system from January 2016 to December 2022.

Inclusion criteria were as follows: 1) all adults aged 18 years or older who were admitted to ICU or a progressive care unit with a primary diagnosis of AWS (ICD-10 F10.23, ICD-9 291.81); 2) a documented CIWA-Ar score on admission.

Exclusion criteria were as follows: 1) patients with an alternative indication for ICU admission other than alcohol withdrawal; 2) a CIWA-Ar score of 0 on admission or a score not recorded; 3) patients who were discharged against medical advice; 4) a history of seizure disorder.

Patients were divided into two groups: group 1 included those who received only standard-of-care treatment (benzodiazepines with or without barbiturates), and group 2 included those who received dexmedetomidine within 5 days of hospi-

tal admission with standard of care treatment (benzodiazepines with or without barbiturates). Dexmedetomidine initiation was at the discretion of the senior intensivist based on the patient's agitation or autonomic instability. Dexmedetomidine was typically initiated at 0.2 - 0.7 $\mu\text{g}/\text{kg}/\text{h}$ with a maximum of 1.5 $\mu\text{g}/\text{kg}/\text{h}$. Patients admitted multiple times for alcohol withdrawal were treated as separate subjects for statistical purposes.

Outcomes and statistical analysis

Baseline characteristics including demographics and comorbidities were captured (Table 1). Exposure was defined as initiation of dexmedetomidine infusion for any duration of time during the first 5 days of hospital admission. The primary outcome of the study was length of ICU stay, and length of hospital stay, which were both determined by automatic timestamps on the electronic medical record of each patient. Secondary outcomes included the number of lorazepam equivalent (total benzodiazepines administered were converted to lorazepam equivalent for standardization as characterized in Table 2), mortality, re-admission at 30 days, 60 days and 90 days. We also evaluated the factors that were related to dexmedetomidine administration. Baseline characteristic variables were expressed in numbers and percentages with age at the encounter expressed in mean with standard deviation (SD). Outcome variables were expressed in mean with SD or median with interquartile range (IQR) where appropriate. The Chi-square test and Kruskal-Wallis test were used for univariate analysis and baseline demographics, and a P value of 0.05 was considered significant. An independent sample *t*-test was used to compare means for hospital and ICU length of stay (LOS) between both groups.

A binary logistic regression to assess predictors of dexmedetomidine administration was conducted using dexmedetomidine use (yes/no) as the dependent variable. Independent variables included phenobarbital use, age, sex, race, and ethnicity. We also conducted two multivariable linear regression models to evaluate the association between dexmedetomidine use and 1) hospital LOS and 2) ICU LOS, for which we used log-transformed LOS values as dependent variables. Dexmedetomidine use was the main exposure of interest, while age, sex, race, ethnicity, and phenobarbital use were added as potential confounders based on clinical relevance.

All models were evaluated for statistical significance with a two-sided alpha of 0.05, and residual plots were checked in linear models to confirm approximate normality and model fit. Data analysis was done by SPSS v26.

Results

Baseline characteristics

In this study, the baseline characteristics (Table 1) of 994 patients with AWS were assessed, with 371 patients in the group without dexmedetomidine and 623 patients in the group with dexmedetomidine. On univariate analysis, there were no significant differences in sex ($P = 0.79361$), race ($P = 0.35591$),

Table 1. Patients Characteristics

	No dexmedetomidine (n = 371)	With dexmedetomidine (n = 623)	Total (n = 994)	P value
Sex, n (%)				
Female	77 (20.8%)	125 (20.1%)	202 (20.3%)	0.7936 ^a
Male	294 (79.2%)	498 (79.9%)	792 (79.7%)	
Race, n (%)				
American Indian or Alaska Native	0 (0.0%)	1 (0.2%)	1 (0.1%)	
Asian	4 (1.1%)	2 (0.3%)	6 (0.6%)	
Black or African American	7 (1.9%)	18 (2.9%)	25 (2.5%)	
Unknown	1 (0.3%)	4 (0.6%)	5 (0.5%)	0.3559 ^a
White	359 (96.8%)	598 (96.0%)	957 (96.3%)	
Ethnicity, n (%)				
Hispanic or Latino	2 (0.5%)	15 (2.4%)	17 (1.7%)	
Non-Hispanic or Latino	361 (97.3%)	597 (95.8%)	958 (96.4%)	
Unknown	8 (2.2%)	11 (1.8%)	19 (1.9%)	0.0827 ^a
Age at encounter, years				
Mean (SD)	52.8 (12.98)	54.0 (12.24)	53.6 (12.53)	
Median (IQR)	54.0 (45.0, 61.0)	55.0 (47.0, 62.0)	55.0 (46.0, 62.0)	0.0833 ^b
Range	18.0, 85.0	18.0, 88.0	18.0, 88.0	
Phenobarbital (yes = 1, no = 0), n (%)				
No	313 (84.4%)	476 (76.4%)	789 (79.4%)	0.0027 ^a
Yes	58 (15.6%)	147 (23.6%)	205 (20.6%)	
Benzodiazepines (yes = 1, no = 0), n (%)				
Yes	371 (100.0%)	623 (100.0%)	994 (100.0%)	Not applicable
No	0 (0%)	0 (0%)	0 (0%)	
COPD (yes = 1, no = 0), n (%)				
No	285 (76.8%)	484 (77.7%)	769 (77.4%)	
Yes	86 (23.2%)	139 (22.3%)	225 (22.6%)	0.7514 ^a
CAD (yes = 1, no = 0), n (%)				
No	314 (84.6%)	527 (84.6%)	841 (84.6%)	0.9847 ^a
Yes	57 (15.4%)	96 (15.4%)	153 (15.4%)	
Asthma (yes = 1, no = 0), n (%)				
No	344 (92.7%)	572 (91.8%)	916 (92.2%)	0.6064 ^a
Yes	27 (7.3%)	51 (8.2%)	78 (7.8%)	

^aChi-square P value. ^bKruskal-Wallis P value. SD: standard deviation; IQR: interquartile range; COPD: chronic obstructive pulmonary disease; CAD: coronary artery disease.

and chronic obstructive pulmonary disease (COPD) status ($P = 0.75141$) between the two groups. The distribution of sex in both groups was similar, with 79.2% male patients in the non-dexmedetomidine group and 79.9% in the dexmedetomidine group. The majority of patients in both groups were White (96.8% in the non-dexmedetomidine group and 96.0% in the dexmedetomidine group). The prevalence of COPD was also similar between the groups, with 23.2% in the non-dexmedetomidine group and 22.3% in the dexmedetomidine group. There was a marginally significant difference in ethnicity distribution

between the two groups ($P = 0.08271$), with a slightly higher percentage of Hispanic or Latino patients in the dexmedetomidine group (2.4%) compared to the non-dexmedetomidine group (0.5%). The mean age at enrollment was similar between the two groups, with a mean age of 52.8 years (SD = 12.98) in the non-dexmedetomidine group and 54.0 years (SD = 12.24) in the dexmedetomidine group ($P = 0.08332$).

On univariate analysis, phenobarbital use appeared to be associated with increased dexmedetomidine use (71.7% vs. 60.3%, $P = 0.0027$). However, after adjusting for age, race,

Table 2. Primary and Secondary Outcomes

	No dexmedetomidine (n = 371)	With dexmedetomidine (n = 623)	Total (n = 994)	P value
Primary outcomes - hospital LOS (days)				
Mean (SD)	7.7 (8.32)	12.3 (11.29)	10.6 (10.51)	
Median (IQR)	5.0 (3.0, 9.0)	10.0 (6.0, 16.0)	8.0 (5.0, 14.0)	
Range	0.0, 73.0	1.0, 203.0	0.0, 203.0	< 0.0001 ^a
Primary outcomes - ICU LOS (days)				
Mean (SD)	3.1 (3.77)	6.9 (7.44)	5.5 (6.59)	
Median (IQR)	1.9 (1.0, 3.4)	4.9 (2.9, 9.0)	3.6 (1.8, 7.1)	
Range	0.0, 31.1	0.1, 122.3	0.0, 122.3	< 0.0001 ^a
Secondary outcomes - lorazepam equivalent (mg)				
Mean (SD)	41.19 (82.45)	40.8 (59.17)	40.95 (68.60)	
Median (IQR)	24.8 (42.2)	24.8 (42.2)	24 (42.87)	
Range			0.5, 1410	0.5692
Secondary outcomes - re-admission in 30 days (n)				
Yes	262 (70.6%)	475 (76.2%)	737 (74.1%)	0.0990 ^{b, c}
No	109 (29.4%)	148 (23.8%)	257 (25.9%)	
Secondary outcomes - re-admission in 60 days (n)				
Yes	308 (83.0%)	541 (86.8%)	849 (85.4%)	0.0334 ^{b, c}
No	63 (17.0%)	82 (13.2%)	145 (14.6%)	
Secondary outcomes - re-admission in 90 days (n)				
Yes	314 (84.6%)	556 (89.2%)	870 (87.5%)	0.0426 ^{b, c}
No	57 (15.4%)	67 (10.8%)	124 (12.5%)	

^aIndependent sample t-test. ^bChi-square P value. ^cUnadjusted for ICU LOS. SD: standard deviation; IQR: interquartile range; ICU: intensive care unit; LOS: length of stay.

sex, and ethnicity in a multivariable logistic regression model, phenobarbital use was independently associated with 41% lower odds of receiving dexmedetomidine (adjusted odds ratio (aOR) = 0.59, 95% confidence interval (CI): 0.42 - 0.83, P = 0.002). In addition, the adjusted logistic regression model showed that Hispanic or Latino ethnicity patients had 6.8 times higher odds of being administered dexmedetomidine compared to non-Hispanic or Latino patients (aOR = 6.800, 95% CI: 1.126 - 41.088; P = 0.037).

Each additional day in the ICU was associated with a 26.7% increase in the odds of receiving dexmedetomidine, after adjusting for sex, race, ethnicity, and phenobarbital use (Exp(B) = 1.267, 95% CI: 1.209 - 1.329, P < 0.001). This indicates that longer ICU stays are independently associated with higher likelihood of dexmedetomidine administration, possibly reflecting greater clinical complexity or sedation needs in these patients.

Primary outcomes

Our study revealed significant differences in the primary outcomes between patients who received dexmedetomidine (n = 623) and those who did not (n = 371) as described in Table

2. Patients in the dexmedetomidine group had a longer mean hospital LOS (12.3 ± 11.29 days) compared to the non-dexmedetomidine group (7.7 ± 8.32 days), with a median LOS of 10.0 days (IQR: 6.0 - 16.0) versus 5.0 days (IQR: 3.0 - 9.0), respectively (P < 0.0001). Additionally, the dexmedetomidine group experienced a greater mean number of ICU days (6.9 ± 7.44 days) compared to the non-dexmedetomidine group (3.1 ± 3.77 days), with a median of 4.9 days (IQR: 2.9 - 9.0) versus 1.9 days (IQR: 1.0 - 3.4), respectively (P < 0.0001).

In the multivariable linear regression model evaluating log-transformed hospital LOS as described in Table 3, the use of dexmedetomidine was independently associated with 58% increase in hospital LOS (CI: 0.380 - 0.542, P < 0.001, Exp(B) = 1.586). In addition, increasing age was also significantly associated with longer hospital stays (CI: 0.012 - 0.018, P < 0.001) which was a 1.5% increase in LOS per year of age. Phenobarbital use showed a positive association with LOS but did not reach statistical significance (CI: -0.012 - 0.182, P = 0.085). Sex, race, and ethnicity were not significantly associated with hospital LOS.

In the multivariable linear regression model for ICU LOS as described in Table 3, dexmedetomidine use was independently associated with a 91.2% increase in ICU LOS (95% CI: 0.564 - 0.733, P < 0.001, Exp(B) = 1.912). Age at encounter

Table 3. Primary Outcomes Adjusted for Age and Dexmedetomidine Use

Outcome	Predictor	B	95% CI (B)	P value	Exp(B)	% Change in LOS
Hospital LOS	Age (per year)	0.015	0.012 to 0.018	< 0.001	1.015	+1.5%
	Precedex use	0.461	0.380 to 0.542	< 0.001	1.586	+58.6%
ICU LOS	Age (per year)	0.011	0.008 to 0.014	< 0.001	1.011	+1.1%
	Precedex use	0.648	0.564 to 0.733	< 0.001	1.912	+91.2%

ICU: intensive care unit; LOS: length of stay; CI: confidence interval.

was also a significant predictor (95% CI: 0.008 - 0.014, $P < 0.001$), with each additional year associated with a 1.1% increase in ICU LOS. Phenobarbital use also showed a positive association with ICU stay duration (95% CI: -0.006 - 0.196) but was not statistically significant ($P = 0.065$).

Secondary outcomes

In terms of secondary outcomes, there was no significant difference in lorazepam equivalent doses between the two groups ($P = 0.569$). On univariate analysis, the rate of 30-day readmission did not significantly differ between the dexmedetomidine group (23.8%) and the non-dexmedetomidine group (29.4%), with a P value of 0.0990. After adjusting for baseline covariates including age, sex, race, ethnicity, and phenobarbital use, dexmedetomidine administration was significantly associated with higher odds of 30-day readmission (OR: 1.39, 95% CI: 1.03 - 1.87, $P = 0.031$). However, after adjusting for ICU LOS, this association was no longer statistically significant (OR: 1.25, 95% CI: 0.91 - 1.71, $P = 0.170$), which suggests that dexmedetomidine use and early readmission could be mediated by severity of illness and longer ICU stays.

Similarly, the observed association between dexmedetomidine use and 90-day readmission appears to be confounded by ICU LOS as the effect of dexmedetomidine loses statistical significance once length of ICU stay is accounted for. In summary, our retrospective cohort study found that the use of dexmedetomidine in the treatment of AWS was associated with a significantly longer hospital and ICU LOS compared to patients not receiving dexmedetomidine. Dexmedetomidine administration was independently influenced by increased length of ICU stay, and there was no influence of dexmedetomidine on readmission rates when controlled for ICU LOS.

Discussion

We conducted a retrospective cohort study aimed at characterizing the use of dexmedetomidine in the treatment of AWS in the critical care setting. Our study revealed that the use of dexmedetomidine in addition to standard therapy can possibly lead to longer lengths of hospital and ICU stay as compared to standard therapy alone. There were no significant differences in lorazepam equivalent doses and 30-day readmission rates or 90-day readmission rates.

Previous studies examining dexmedetomidine in the management of AWS have generally been limited by small sam-

ple sizes and single-center designs, often including fewer than 200 patients. These studies lacked sufficient power to control for key confounders such as adjunct phenobarbital use, demographic variability, and illness severity. In contrast, our investigation includes nearly 1,000 encounters across multiple hospitals within a large integrated health system, providing one of the largest real-world cohorts to date and allowing for more robust multivariable analysis. Previous studies have conveyed similar results, showing increased length of hospital stay and longer duration of ICU stay [6, 8, 9]. This is notable given the myriad detrimental associations of extended lengths of hospital and ICU stays. Although causation cannot be established with the observational study of this nature, there may be an association between patients who receive dexmedetomidine for the treatment of AWS and an increased LOS in the hospital and critical care setting, which is corroborated by existing literature.

However, logistic regression suggests that longer ICU stays also predict the likelihood of receiving dexmedetomidine, raising the possibility of confounding by clinical severity and a possible reverse association. Therefore, while dexmedetomidine use and ICU LOS are closely linked, we cannot infer a causal direction without accounting for illness acuity.

We found that re-admission rates at 30 days were increased among dexmedetomidine-treated patients, though this difference only trended toward significance. The rate of readmission at 60 and 90 days post-discharge was significantly higher in patients who received dexmedetomidine (Table 2). However, when accounting for ICU LOS, these results were also not statistically significant. In sum, these findings may point toward a pattern of increased healthcare utilization and re-utilization among AWS patients treated with dexmedetomidine.

This suggests that dexmedetomidine may provide some long-term benefit in preventing AWS recurrence by allowing for a thorough withdrawal management, symptom control and stabilization prior to discharge. However, this should be weighed against the possibility of a significantly longer ICU and hospital LOS that has been associated with the dexmedetomidine group, which raises concerns about the increased healthcare resource utilization and costs. Hence, it might be a better option to tailor its use to high-risk populations with a history of multiple hospitalizations for AWS or severe withdrawal symptoms that require prolonged medical management. Future studies should focus on identifying subpopulations of AWS patients who may benefit the most from dexmedetomidine use and evaluating strategies to limit its routine use in cases where benzodiazepines alone may suffice, thereby helping avoid excessive ICU stays.

Our study did not find any significant reduction in lorazepam equivalents, which is similar to the findings reported by Yavarovich et al [8] and Beg et al [6]. However, conflicting evidence was noted in the work done by Collier et al [7], who reported an increase in the requirement of benzodiazepines in patients who were on dexmedetomidine. Beg et al [6] reported a statistically significant improvement in CIWA-Ar scores, indicating that dexmedetomidine may still help in symptom control despite not reducing benzodiazepine requirements.

Patients who received dexmedetomidine in our study were more likely to have also received phenobarbital, which shows that these individuals might have presented with more severe AWS and hence required additional pharmacologic management and had worse outcomes. This is a critical consideration, as phenobarbital has been shown to be effective in AWS treatment by potentiating GABAergic inhibition, thereby reducing withdrawal symptoms and the need for prolonged ICU stays [10, 11]. While phenobarbital alone has been associated with shorter ICU stays and reduced hospitalizations in AWS management, its combination with dexmedetomidine may reflect a more severe baseline disease state rather than a direct effect of the drug combination itself. Additionally, the severity of AWS based on CIWA-Ar scores was not taken into consideration in the analysis due to limited data from our chart review, hence this could be another confounding variable that could independently be contributing to prolonged LOS in hospital and ICU. This highlights the need for further prospective studies to isolate the independent effects of dexmedetomidine, phenobarbital and CIWA-Ar scores on AWS outcomes.

Our study's limitations include its adoption of a retrospective cohort design, as we are unable to make causal inferences regarding the use of dexmedetomidine and the outcomes described above. As such, the influence of unmeasured factors on our study's findings cannot be discounted. However, given the concordance of our findings with other investigations of dexmedetomidine in the treatment of AWS, we believe that our study nonetheless adds valuable information to the literature. Our sample was composed almost entirely of non-Hispanic White individuals; this is significant insofar as it limits the generalizability of these findings to the population of individuals with AWS at large, especially given that the study of novel agents to treat AWS should be made to include individuals of diverse ethnic and racial backgrounds.

We feel confident in our study given the degree to which the findings above were significant, the sample size, and our study's concordance with others [6, 7]. Much of the previous research regarding the use of dexmedetomidine in the treatment of AWS has understandably featured smaller samples of patients. As such, our study's large sample provided us with sufficient power to investigate the effects of dexmedetomidine on outcomes for patients with AWS. Additionally, while the current study is certainly limited by its predominantly Caucasian sample, it is worth noting that the patients included in this study come from a largely rural catchment area. Furthermore, the distribution of significant cardiac and respiratory comorbidities (i.e., COPD, asthma, coronary artery disease) present in the sample make it unlikely that disease burden from these illnesses played a significant role in influencing study outcomes (e.g., lengths of hospital and ICU stay, readmission rates).

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Financial Disclosure

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Conflict of Interest

The authors declare that they have no conflict of interest related to this study.

Informed Consent

Informed consent was waived by the IRB as the study was retrospective in nature with the potential of causing minimal or no harm to the subjects.

Author Contributions

RS and WM contributed to the conceptualization of the study. AA, RS, SA, EC, JW, DZ contributed to the data curation and writing of the manuscript. AA and RS provided edits for the final manuscript. SN, TN, GS, SH, CD and RSP contributed to the interpretation of data and editing of the initial drafts of the publication, as well as performing the initial literature review. BI provided expert opinion on the statistical analysis of the data and validation of the results. WM and RS were senior authors who were supervisors and mentors for all authors. All authors agreed to the submitted version of the manuscript.

Data Availability

All data generated or analyzed during this study are included in this article. Further enquiries can be directed to the corresponding author.

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