Prognostic Value of Muscle Mass Measured via Brain Computed Tomography in Neurocritically Ill Patients

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Research

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Abstract

Background

To investigate whether skeletal muscle mass estimated via brain computed tomography (CT) can be used to predict neurological outcomes in neurocritically ill patients.

Methods

This is a retrospective, observational study. Adult patients who were admitted to the neurosurgical intensive care unit (ICU) in tertiary hospital from January 2010 to September 2019 were eligible. We included patients who were hospitalized in the neurosurgical ICU for more than 7 days. Cross-sectional areas of paravertebral muscle at the first cervical vertebra level (C1-CSA) and temporalis muscle thickness (TMT) on brain CT were measured to evaluate skeletal muscle mass. Primary outcome was Glasgow Outcome Scale score at 3 months.

Results

Among 189 patients, 167 (88.4%) survived until discharge from the hospital. Of these survivors, 81 (42.9%) patients had favorable neurologic outcomes. Initial TMT values and follow-up TMT values were higher in patients with favorable neurologic outcome compared to those with poor neurological outcome ($p = 0.003$ and $p = 0.001$, respectively). Initial the C1-CSA/body surface area was greater in patients with poor neurological outcome than in those with favorable outcome ($p = 0.029$). In multivariable analysis, age (adjusted odds ratio [OR]: 2.05, 95% confidence interval [CI]: 1.543–2.724), BMI (adjusted OR: 0.74, 95% CI: 0.638–0.849), use of mannitol (adjusted OR: 27.45, 95% CI: 4.833–155.860), change of C1-CSA (adjusted OR: 1.36, 95% CI: 1.054–1.761), and change of TMT (adjusted OR: 1.27, 95% CI: 1.028–1.576) were significantly associated with poor neurological outcome (Hosmer–Lemeshow test, Chi-square = 11.4, df = 8, $p = 0.178$) with the areas under curve of 0.803 (95% CI 0.740–0.866) using 10-fold cross validation method. Especially, the risk of poor neurologic outcome was proportional to changes of C1-CSA and TMT.

Conclusions

In this study, the follow-up skeletal muscle mass at first week from ICU admission, based on changes in C1-CSA and TMT, was associated with neurological prognosis in neurocritically ill patients. Eventually, brain CT-measured sarcopenia may be helpful in predicting poor neurological outcomes in these patients.

Key Messages

- Malnutrition affects neurological prognosis as well as mortality in neurocritically ill patients. Sarcopenia is associated with clinical prognosis in neurocritically ill patients.
- CSAs of skeletal muscle mass at C1 vertebral level and TMT are easily determined using brain CT scans. CSAs based on brain CT can be used as alternatives to estimate nutritional status.
- In this study, the follow-up skeletal muscle mass at first week from ICU admission, based on changes in C1-CSA and TMT, was associated with neurological prognosis in neurocritically ill patients.
- Adequate nutritional support and early mobilization to prevent skeletal muscle loss may facilitate recovery in neurocritically ill patients.

Background

Nutrition is an important factor in the management of critically ill patients [1–3]. Malnutrition is associated with prolonged hospitalization and duration of mechanical ventilation, infection, and mortality in the intensive care unit (ICU) [2, 4, 5]. Malnutrition is also associated with poor clinical outcomes in neurocritically ill patients [6–8]. Nutritional support can affect neurological prognosis as well as mortality in patients with stroke or traumatic brain injury [6–8]. Sarcopenia is characterized by the loss of skeletal muscle mass and its function [9]. Skeletal muscle mass is associated with physiologic functions [10, 11]. In critically ill patients, malnutrition and prolonged immobility due to severe illness increase the risk of sarcopenia during their ICU stay [2]. Eventually, sarcopenia is associated with poor clinical prognosis in these patients [12, 13]. Therefore, it is important to estimate the nutritional status based on skeletal muscle mass and to provide adequate nutrition.

Skeletal muscle mass can be measured via whole body or regional dual-energy X-ray absorptiometry scans and volumetric or cross-sectional area (CSA) measurements on magnetic resonance imaging or computed tomography (CT) scans at the arm, leg or third lumbar vertebral level [13]. However, muscle mass measurement using the CSA on imaging scans and dual-energy X-ray absorptiometry scans may not be routinely performed in neurocritically ill patients [14]. In neurocritically ill patients, brain CT scans are frequently performed. Although skeletal muscle mass is not routinely assessed in brain CT, it may rapidly decrease on follow-up brain CT scans in neurocrtically ill patients (Fig. 1). A limited number of studies evaluated the skeletal muscle mass via brain CT [14–16]. In addition, no study reported clinical prognosis according to the changes in skeletal muscle mass using brain CT. Therefore, the objective of this study was to investigate whether skeletal muscle mass estimated via brain CT can be used to predict neurological outcomes in neurocritically ill patients.

Methods

Study Population

This is a retrospective, single-center, observational study. Adult patients who were admitted to the neurosurgical ICU in our tertiary hospital (Samsung Medical Center, Seoul, Republic of Korea) from

January 2010 to September 2019 were eligible. This study was approved by the Institutional Review Board of Samsung Medical Center (approval number: SMC 2020-02-113). The requirement for informed consent was waived due to its retrospective nature. We included patients (1) who were hospitalized in the neurosurgical ICU for more than 7 days, (2) evaluated with brain CT on ICU admission, (3) with follow-up brain CT within the first 6 to 8 days after ICU admission. Of these patients, we excluded patients (1) aged below 18 years, (2) those who did not have a brain injury, (3) with insufficient medical records, (4) with a history of chronic neurological abnormality on admission, (5) who stayed in the ICU for more than 7 days due to the lack of a general ward, (6) on 'do not resuscitation' order, and (7) those who were admitted to departments other than neurosurgery.

Definitions and endpoints

In this study, baseline characteristics of comorbidities, causes of ICU admission, and initial clinical parameters on admission were retrospectively obtained through medical record review. Acute Physiology and Chronic Health Evaluation (APACHE) II score was calculated with worst values recorded during the initial 24 h after the ICU admission [17, 18]. If the patient was intubated, the verbal score of Glasgow Coma Scale was estimated using the eye and motor scores as described previously [19].

CSA of the paravertebral muscle at first cervical vertebral level (C1-CSA) was evaluated on brain CT (Fig. 3a). The skeletal muscles were identified at the transverse process level with Hounsfield unit thresholds ranging from − 29 to + 150. An investigator delineated all the muscles manually, and the C1-CSA was automatically retrieved as the total sum of pixels [14]. The difference between initial C1-CSA and follow-up C1-CSA (ΔC1-CSA) was defined as initial C1-CSA minus follow-up C1-CSA. The change of C1-CSA was defined as ΔC1-CSA divided by initial C1-CSA multiplied by 100. Temporalis muscle thickness (TMT) was also measured perpendicular to the long axis of the temporal muscle in the axial plane of the CT image (Fig. 3b). The Sylvian fissure was used as a reference point of TMT measurement at the level of the orbital roof [16, 20]. The maximum TMT was used as the TMT value, whichever was thicker than the other. If the patient underwent neurosurgery, including craniotomy or craniectomy, on one side within two weeks before the initial brain CT scan, the TMT of the other side alone was used for analysis. If the patient had neurosurgery bilaterally, their TMT values were not used in the analysis. The difference between initial TMT and follow-up TMT (ΔTMT) was defined as initial TMT minus follow-up TMT. The change of TMT was defined as ΔTMT divided by initial TMT multiplied by 100. All the CT studies were performed using 64-channel scanners (Light Speed VCT, GE Healthcare, Milwaukee, WI, USA) with a 5-mm slice width. Trained intensivists evaluated each of the patients' CT scans using commercial image-viewing software (Centricity RA1000 PACS Viewer, GE Healthcare) [21]. The images were changed to the "chest/abdomen" window (window width 300 & window level 10) and magnified threefold to fourfold on the particular image slice that demonstrated the largest diameter of TMT.

Primary outcome was performance on Glasgow Outcome Scale (GOS) at 3 months. Patients with GOS scores 4 to 5 indicated favorable neurological outcome, whereas GOS 1 to 3 suggested poor neurological outcome [22, 23].

Statistical Analyses

All data are presented as medians and interquartile ranges (IQRs, Q1 ~ Q3) for continuous variables and as numbers (percentages) for categorical variables. Data were compared using the Mann-Whitney *U* test for continuous variables and the Chi-square test or Fisher's exact test for categorical variables. Variables with *p* value less than 0.2 in univariate analyses and clinically relevant variables, including age, sex, BMI, BSA, comorbidities, GCS and APACHE II score on ICU admission, initial level of serum albumin, and use of mannitol, were subjected to multiple logistic regression analysis to obtain statistically meaningful predictors. Stepwise variable selection was conducted to construct the final model. Adequacy of the prediction model was determined using the Hosmer-Lemeshow test, along with the areas under the curve (AUC). Split-sample analyses and 10-fold cross validation analysis were conducted to assess the internal validity. All tests were two-sided and $p < 0.05$ was considered statistically significant. All data were analyzed using R Statistical Software (version 4.0.2; R Foundation for Statistical Computing, Vienna, Austria).

Results

Baseline Characteristics and Clinical Outcomes

Finally, 189 patients were analyzed (Fig. 2). Median age of patients was 58.0 (IQR: 48.0–70.0) years. One hundred patients (52.9%) were males. Malignancy (56.1%) and hypertension (46.6%) were the most common comorbidities in the study population. Brain tumor (41.3%) and stroke (37.0%) were the most common causes of ICU admission. Age and APACHE II scores on ICU admission were greater in the poor outcome group than in the favorable outcome group. Body mass index (BMI) and body surface area (BSA) were higher in the favorable outcome group compared with poor outcome group. Baseline characteristics of the study population are presented in Table 1.

Table 1
Baseline characteristics

	Favorable neurologic outcome (*n* = 81)	Poor neurologic outcome (*n* = 108)	*P* value
Age (year)	53.0 (33–63.5)	63.5 (52.3–72.8)	< 0.001
Sex, male	41 (50.6)	59 (54.6)	0.689
BMI (kg/m^2)	24.1 (22.6–26.7)	22.8 (20.7–25.1)	< 0.001
Body surface area (m^2)	1.8 (1.6–1.8)	1.6 (1.5–1.8))	< 0.001
Comorbidities			
Malignancy	46 (56.8)	60 (55.6)	0.983
Hypertension	30 (37.0)	55 (50.9)	0.080
Diabetes mellitus	11 (13.6)	28 (25.9)	0.058
Current smoker	13 (16.0)	15 (13.9)	0.836
Ischemic heart disease	4(4.9)	8 (7.4)	0.698
Chronic kidney disease	4 (4.9)	8 (7.4)	0.698
Cause of ICU admission			0.027
Brain tumor	34 (42.0)	44 (40.7)	
Stroke*	29 (35.8)	41 (38.0)	
Traumatic brain injury	4 (4.9)	16 (14.8)	
Others	14 (17.3)	7 (6.5)	
GCS on ICU admission	7.0 (3.0–13.0)	6.0 (3.0–10.0)	0.030
APACHE II score on ICU admission	18.0 (14.0–23.0)	21.0 (17.3–26.0)	0.001
Use of mannitol†	70 (86.4)	104 (96.3)	0.027
Use of glycerin†	54 (66.7)	75 (69.4)	0.804

Data are numbers (%) or median (interquartile range).

*Stroke included intracerebral hemorrhage, subarachnoid hemorrhage, and cerebral infarction.

†Some patients received more than one hyperosmolar agents.

BMI, body mass index; ICU, intensive care unit; GCS, Glasgow Coma Scale; APACHE, Acute Physiology and Chronic Health Evaluation

	Favorable neurologic outcome (*n* = 81)	Poor neurologic outcome (*n* = 108)	*P* value
Use of dexamethasone	57 (70.4)	68 (63.0)	0.363
Initial albumin level (g/dl)	3.4 (3.1–3.9)	3.4 (3.0–3.9)	0.403
Data are numbers (%) or median (interquartile range).			
*Stroke included intracerebral hemorrhage, subarachnoid hemorrhage, and cerebral infarction.			
†Some patients received more than one hyperosmolar agents.			
BMI, body mass index; ICU, intensive care unit; GCS, Glasgow Coma Scale; APACHE, Acute Physiology and Chronic Health Evaluation			

Among 189 patients, 167 (88.4%) survived until discharge from the hospital. Of these survivors, 81 patients had favorable neurologic outcomes. The entire distribution of GOS is shown in Fig. 2.

Relationship between C1-CSAs, TMTs and neurological outcomes

Initial and follow-up TMT values were higher in patients with favorable neurological outcome compared to those with poor neurological outcome. However, the change of TMT and ΔTMT were not significantly different between the two groups. Although initial C1-CSA/BSA was greater in patients with poor neurological outcome than in favorable outcome ($p = 0.029$), other variables related to CSA were not significantly different between two groups (Table 2).

Table 2
The cross-sectional areas (CSAs) of first cervical vertebra level (C1) and temporalis muscle thicknesses (TMTs) according to neurological outcomes.

	Favorable neurologic outcome (*n* = 81)	Poor neurologic outcome (*n* = 108)	*P* value
Initial C1-CSA (mm^2)	1825.2 (1602.4–2165.3)	1853.9 (1605.1–2206.6)	0.495
Initial C1-CSA/BSA (mm^2/m^2)	1071.5 (952.0–1225.4)	1120.4 (1040.4 – 1299.0)	0.029
Follow-up C1-CSA (mm^2)	1850.0 (1598.3–2150.6)	1807.8 (1577.1 – 2089.1)	0.686
Follow-up C1-CSA/BSA (mm^2/m^2)	1072.6 (930.6–1201.8)	1099.4 (978.5 – 1231.8)	0.390
ΔC1-CSA (mm^2)	22.8 (-147.3–180.6)	78.1 (-86.3–225.7)	0.123
ΔC1-CSA/BSA (mm^2/m^2)	7.5 (-84.8–111.3)	60.0 (-42.4–137.7)	0.086
Change of C1-CSA	1.4 (-7.9–9.4)	4.4 (-4.4–11.6)	0.133
Initial TMT (mm)	7.2 (6.1–9.1)	6.4 (5.2–7.6)	0.003
Follow-up TMT (mm)	5.9 (4.9–7.6)	5.1 (4.0–6.6)	0.001
ΔTMT (mm)	1.0 (0.2–1.9)	1.3 (0.4–2.1)	0.496
Change of TMT	14.1 (-2.9–26.5)	18.1 (7.9–29.6)	0.110
Data are median (interquartile range).			
BSA, Body surface area; ΔC1-CSA,initial C1-CSA minus follow-up C1-CSA; ΔC1-CSA/BSA, initial C1-CSA/BSA minus follow-up C1-CSA/BSA; Change of C1-CSA, 100 × (ΔC1-CSA/initial C1-CSA); ΔTMT, initial TMT minus follow-up TMT; change of TMT, 100 × (initial TMT minus follow-up TMT)/initial TMT			

In multivariable analysis (Table 3), age (adjusted odds ratio [OR]: 2.05, 95% confidence interval [CI]: 1.543–2.724), BMI (adjusted OR: 0.74, 95% CI: 0.638–0.849), use of mannitol (adjusted OR: 27.45, 95% CI: 4.833–155.860), change of C1-CSA (adjusted OR: 1.36, 95% CI: 1.054–1.761), and change of TMT (adjusted OR: 1.27, 95% CI: 1.028–1.576) were significantly associated with poor neurological outcome (Hosmer–Lemeshow Chi-squared = 11.4, $df = 8$, $p = 0.178$) with the AUCs of 0.803 (95% CI 0.740–0.866) using 10-fold cross-validation method (Fig. 4). Especially, the risk of poor neurological outcome was proportional to changes of C1-CSA and TMT (Fig. 5).

Table 3
Multivariable analysis of factors associated with poor neurologic outcome at 3 months

	*Adjusted odds ratio (95% CI)	*P* value
Age (year)	2.05 (1.543–2.724)	< 0.001
BMI (kg/m^2)	0.74 (0.638– 0.849)	< 0.001
APACHE II score on ICU admission	1.84 (0.996–3.396)	0.052
Use of mannitol	27.4 (4.833– 155.860)	< 0.001
Change of C1-CSA	1.36 (1.054– 1.761)	0.018
Change of TMT	1.27 (1.028– 1.576)	0.027

CI, confidence interval; BMI, body mass index; APACHE, Acute Physiology and Chronic Health Evaluation; ICU, intensive care unit; GCS, Glasgow coma scale; C1-CSA, cross sectional area of first cervical vertebral level; TMT, temporalis muscle thickness; Change of C1-CSA, 100 × (initial C1-CSA minus follow-up C1-CSA)/initial C1-CSA; change of TMT, 100 × (initial TMT minus follow-up TMT)/initial TMT

Discussion

In this study, we investigated whether skeletal muscle mass estimated by brain CT could be used to predict neurological outcomes in neurocritically ill patients. Major findings of this study were as follows. First, a half of the surviving patients had a favorable neurological prognosis in this study. Second, during initial and follow-up CT, the TMT values of the poor neurological outcome group were significantly lower than those of the favorable neurological outcome group. However, during initial and follow-up CT, the C1-CSAs were not significantly different between the two groups except for initial C1-CSA/BSA. Second, in multivariable analysis, age, BMI, use of mannitol, and changes in C1-CSAand TMT were significantly associated with poor neurological outcomes in neurocritically ill patients. Especially, the risk of poor neurological outcome was proportional to changes of C1-CSA and TMT.

Nutritional support is an important issue in intensive care of critically ill patients [1–3]. Malnutrition is also associated with poor clinical prognosis of neurocritically ill patients [6, 7, 24]. Inadequate nutritional support increases susceptibility to infection, mortality, and neurological outcomes in these patients [6, 7, 24, 25]. Malnutrition has been estimated depending on various parameters that may include BMI, serum albumin and skeletal muscle mass [2]. However, BMI and serum albumin are poor parameters representing nutritional status in critically ill patients [1, 2]. Skeletal muscle mass is a more accurate parameter in assessing nutritional status and may reflect the clinical prognosis better than other nutritional measures in critically ill patients [2].

The CSA of skeletal muscle mass has been estimated via abdominal CT at third lumbar vertebral level, which correlates with the total body skeletal muscle mass and can be easily measured on an abdominal CT acquired during intensive care [12, 14, 26, 27]. Recent studies showed that CSAs of skeletal muscle

mass at the level of cervical vertebrae on a head and neck CT scan significantly correlate with those at third lumbar vertebral level on abdominal CT scan [14, 28]. In addition, TMT also correlates with CSAs of skeletal muscle mass at third lumbar vertebral level or total psoas muscle area on abdominal CT scan [15, 16]. Therefore, CSAs of skeletal muscle mass at the cervical vertebra levels and TMT on brain CT can be used as alternatives to estimate sarcopenia and nutritional status in neurocritically ill patients.

Sarcopenia generally occurs in critically ill patients and may progress after ICU admission [29]. Skeletal muscle mass begins to decrease remarkably within 3 days and gradually deteriorates [3, 29]. In addition, the muscle mass of the limbs can be reduced by one-fifth within 7 days after ICU admission due to malnutrition and prolonged immobility as a consequence of critical illness [29, 30]. Skeletal muscle mass plays an important role in physiological functions such as immune modulation, protein synthesis and glucose metabolism [2, 11]. Therefore, sarcopenia secondary to critical illness is associated with adverse clinical prognosis [12, 13]. Similarly, malnutrition during the first week could be associated with poor neurological outcomes in patients with stroke [6]. Therefore, sarcopenia in the first week may be associated with poor neurological outcomes in neurocritically ill patients as well. In this study, changed muscle mass at first week was also associated with prognosis in neurocritically ill patients.

This study has several limitations. First, this was a retrospective review. Thus, GOS was determined based on medical records. Any bias involving the scores was mitigated partially based on the consensus of two independent specialists. Second, the nonrandomized nature of registry data might have resulted in selection bias. Brain CT scans were not protocol-based in their performance. Third, TMT of the surgical direction was not available because of possible damage and mobilization of the temporalis muscle occurring during either dissection, transsection, or incision after temporal craniotomy [31]. Lastly, our study has limited statistical power due to its small sample size. Although it still provides a valuable insight, prospective large-scale studies are needed to confirm the role of brain CT-based muscle mass measurement in predicting the clinical prognosis of neurocritically ill patients to arrive at evidence-based conclusions.

Conclusions

In this study, follow-up skeletal muscle mass at first week from ICU admission based on changes in C1-CSA and TMT is associated with neurological prognosis in neurocritically ill patients. Eventually, sarcopenia measured via brain CT may suggest poor neurological outcomes in these patients. Therefore, adequate nutritional support and early mobilization to prevent sarcopenia may facilitate recovery in neurocritically ill patients.

Abbreviations

APACHE II, acute physiology and chronic health evaluation II; AUC, area under the curve; BSA, body surface area; CI, confidence interval; CSA, cross-sectional area; CT, computed tomography; GOS, Glasgow outcome scale; ICU, intensive care unit; OR, odd ratio; TMT, temporalis muscle thickness.

Declarations

Ethics approval and consent to participate

This study was approved by the Institutional Review Board of Samsung Medical Center (approval number: SMC 2020-02-113). Patients' records were reviewed and published according to the Declaration of Helsinki. Informed consent was waived because of the retrospective nature of this study.

Consent for publication

Not applicable. This study does not contain individual or personal data in any form (including individual details, images, or videos).

Availability of data and materials

Regarding data availability, our data are available on the Harvard Dataverse Network (http://dx.doi.org/10.7910/DVN/GF08RY) as recommended repositories of critical care.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

YIL contributed to the study design, data collection, drafting of the manuscript, and statistical analysis. REK contributed to the study design, data collection, drafting of the manuscript, and statistical analysis. KCC contributed to the study design, drafting of the manuscript, and statistical analysis. JA contributed to the study design and statistical analysis. JAR contributed to the study conception and design, data collection, and drafting of the manuscript. All authors read and approved the final manuscript.

References

1. Higgins PA, Daly BJ, Lipson AR, Guo S-E. Assessing nutritional status in chronically critically ill adult patients. American journal of critical care : an official publication, American Association of Critical-Care Nurses. 2006;15(2):166-77.

2. Moisey LL, Mourtzakis M, Cotton BA, Premji T, Heyland DK, Wade CE, et al. Skeletal muscle predicts ventilator-free days, ICU-free days, and mortality in elderly ICU patients. Crit Care. 2013;17(5):R206.
3. Puthucheary ZA, Rawal J, McPhail M, Connolly B, Ratnayake G, Chan P, et al. Acute skeletal muscle wasting in critical illness. Jama. 2013;310(15):1591-600.
4. Villet S, Chiolero RL, Bollmann MD, Revelly JP, Cayeux RNM, Delarue J, et al. Negative impact of hypocaloric feeding and energy balance on clinical outcome in ICU patients. Clin Nutr. 2005;24(4):502-9.
5. Dvir D, Cohen J, Singer P. Computerized energy balance and complications in critically ill patients: an observational study. Clin Nutr. 2006;25(1):37-44.
6. Yoo SH, Kim JS, Kwon SU, Yun SC, Koh JY, Kang DW. Undernutrition as a predictor of poor clinical outcomes in acute ischemic stroke patients. Arch Neurol. 2008;65(1):39-43.
7. Wang X, Dong Y, Han X, Qi XQ, Huang CG, Hou LJ. Nutritional support for patients sustaining traumatic brain injury: a systematic review and meta-analysis of prospective studies. PLoS One. 2013;8(3):e58838.
8. Sabbouh T, Torbey MT. Malnutrition in Stroke Patients: Risk Factors, Assessment, and Management. Neurocritical care. 2018;29(3):374-84.
9. Santilli V, Bernetti A, Mangone M, Paoloni M. Clinical definition of sarcopenia. Clinical cases in mineral and bone metabolism : the official journal of the Italian Society of Osteoporosis, Mineral Metabolism, and Skeletal Diseases. 2014;11(3):177-80.
10. Moisey LL, Mourtzakis M, Cotton BA, Premji T, Heyland DK, Wade CE, et al. Skeletal muscle predicts ventilator-free days, ICU-free days, and mortality in elderly ICU patients. Crit Care. 2013;17(5):R206.
11. Nelke C, Dziewas R, Minnerup J, Meuth SG, Ruck T. Skeletal muscle as potential central link between sarcopenia and immune senescence. EBioMedicine. 2019;49:381-8.
12. Zwart AT, van der Hoorn A, van Ooijen PMA, Steenbakkers R, de Bock GH, Halmos GB. CT-measured skeletal muscle mass used to assess frailty in patients with head and neck cancer. J Cachexia Sarcopenia Muscle. 2019;10(5):1060-9.
13. Kilgour AH, Subedi D, Gray CD, Deary IJ, Lawrie SM, Wardlaw JM, et al. Design and validation of a novel method to measure cross-sectional area of neck muscles included during routine MR brain volume imaging. PLoS One. 2012;7(4):e34444.
14. Swartz JE, Pothen AJ, Wegner I, Smid EJ, Swart KM, de Bree R, et al. Feasibility of using head and neck CT imaging to assess skeletal muscle mass in head and neck cancer patients. Oral Oncol. 2016;62:28-33.
15. Ranganathan K, Terjimanian M, Lisiecki J, Rinkinen J, Mukkamala A, Brownley C, et al. Temporalis muscle morphomics: the psoas of the craniofacial skeleton. J Surg Res. 2014;186(1):246-52.
16. Leitner J, Pelster S, Schopf V, Berghoff AS, Woitek R, Asenbaum U, et al. High correlation of temporal muscle thickness with lumbar skeletal muscle cross-sectional area in patients with brain metastases. PLoS One. 2018;13(11):e0207849.

17. Knaus WA, Draper EA, Wagner DP, Zimmerman JE. APACHE II: a severity of disease classification system. Crit Care Med. 1985;13(10):818-29.
18. Capuzzo M, Valpondi V, Sgarbi A, Bortolazzi S, Pavoni V, Gilli G, et al. Validation of severity scoring systems SAPS II and APACHE II in a single-center population. Intensive Care Med. 2000;26(12):1779-85.
19. Meredith W, Rutledge R, Fakhry SM, Emery S, Kromhout-Schiro S. The conundrum of the Glasgow Coma Scale in intubated patients: a linear regression prediction of the Glasgow verbal score from the Glasgow eye and motor scores. J Trauma. 1998;44(5):839-44; discussion 44-5.
20. Furtner J, Berghoff AS, Albtoush OM, Woitek R, Asenbaum U, Prayer D, et al. Survival prediction using temporal muscle thickness measurements on cranial magnetic resonance images in patients with newly diagnosed brain metastases. Eur Radiol. 2017;27(8):3167-73.
21. Ryu JA, Jung W, Jung YJ, Kwon DY, Kang K, Choi H, et al. Early prediction of neurological outcome after barbiturate coma therapy in patients undergoing brain tumor surgery. PLoS One. 2019;14(4):e0215280.
22. Jennett B, Bond M. Assessment of outcome after severe brain damage. Lancet. 1975;1(7905):480-4.
23. Jennett B, Snoek J, Bond MR, Brooks N. Disability after severe head injury: observations on the use of the Glasgow Outcome Scale. J Neurol Neurosurg Psychiatry. 1981;44(4):285-93.
24. Bouziana SD, Tziomalos K. Malnutrition in patients with acute stroke. Journal of nutrition and metabolism. 2011;2011:167898-.
25. Dávalos A, Ricart W, Gonzalez-Huix F, Soler S, Marrugat J, Molins A, et al. Effect of malnutrition after acute stroke on clinical outcome. Stroke. 1996;27(6):1028-32.
26. Shachar SS, Williams GR, Muss HB, Nishijima TF. Prognostic value of sarcopenia in adults with solid tumours: A meta-analysis and systematic review. Eur J Cancer. 2016;57:58-67.
27. Shen W, Punyanitya M, Wang Z, Gallagher D, St-Onge MP, Albu J, et al. Total body skeletal muscle and adipose tissue volumes: estimation from a single abdominal cross-sectional image. J Appl Physiol (1985). 2004;97(6):2333-8.
28. Ufuk F, Herek D, Yuksel D. Diagnosis of Sarcopenia in Head and Neck Computed Tomography: Cervical Muscle Mass as a Strong Indicator of Sarcopenia. Clin Exp Otorhinolaryngol. 2019;12(3):317-24.
29. Nakanishi N, Tsutsumi R, Okayama Y, Takashima T, Ueno Y, Itagaki T, et al. Monitoring of muscle mass in critically ill patients: comparison of ultrasound and two bioelectrical impedance analysis devices. J Intensive Care. 2019;7:61.
30. Nakanishi N, Oto J, Tsutsumi R, Iuchi M, Onodera M, Nishimura M. Upper and lower limb muscle atrophy in critically ill patients: an observational ultrasonography study. Intensive Care Med. 2018;44(2):263-4.
31. Abdulazim A, Filis A, Sadr-Eshkevari P, Schulte F, Sandu N, Schaller B. Postcraniotomy Function of the Temporal Muscle in Skull Base Surgery: Technical Note Based on a Preliminary Study. The Scientific World Journal. 2012;2012:427081.

Figures

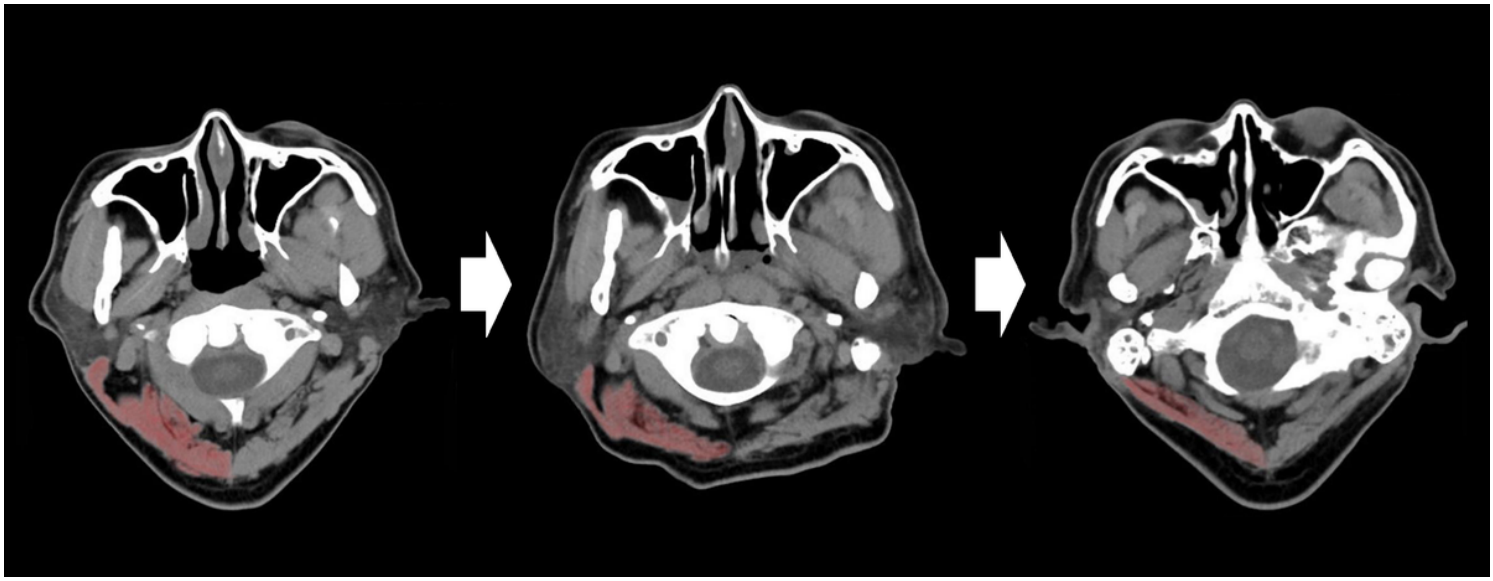
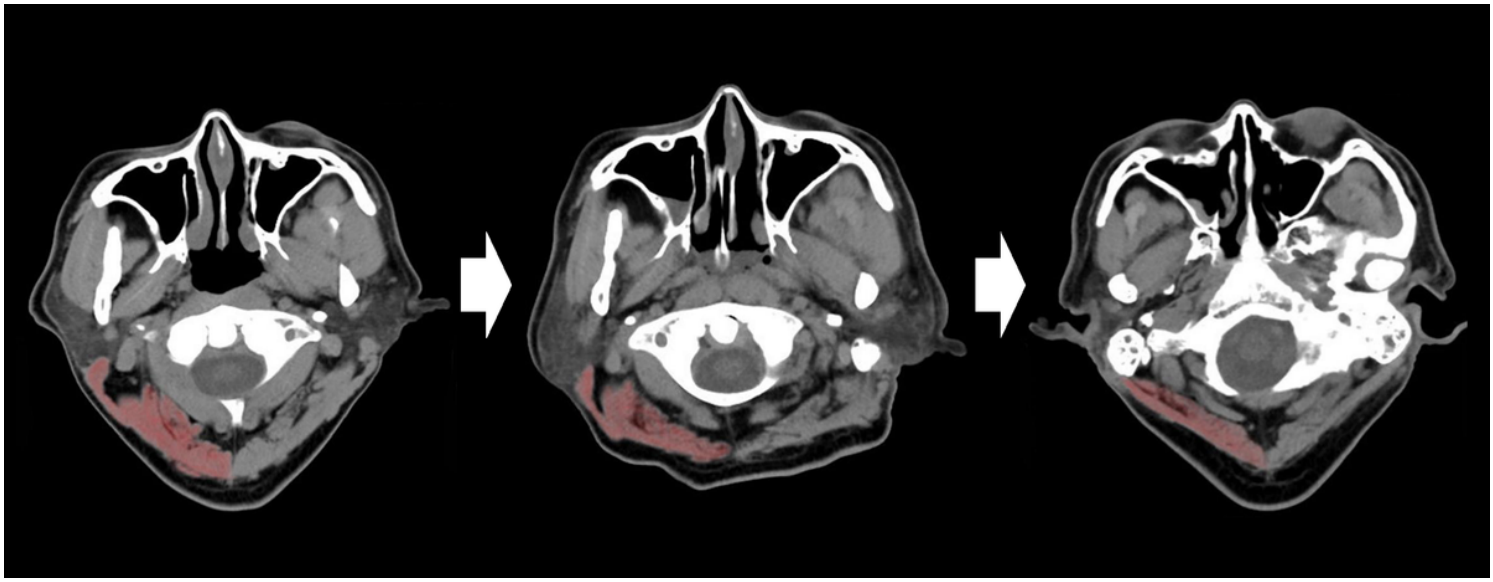


Figure 1

Serial changes of cervical muscle mass in a patient with traumatic brain injury. The brain CT images with 7 days interval showed progressive decrease of cervical muscle mass. CT, computed tomography.



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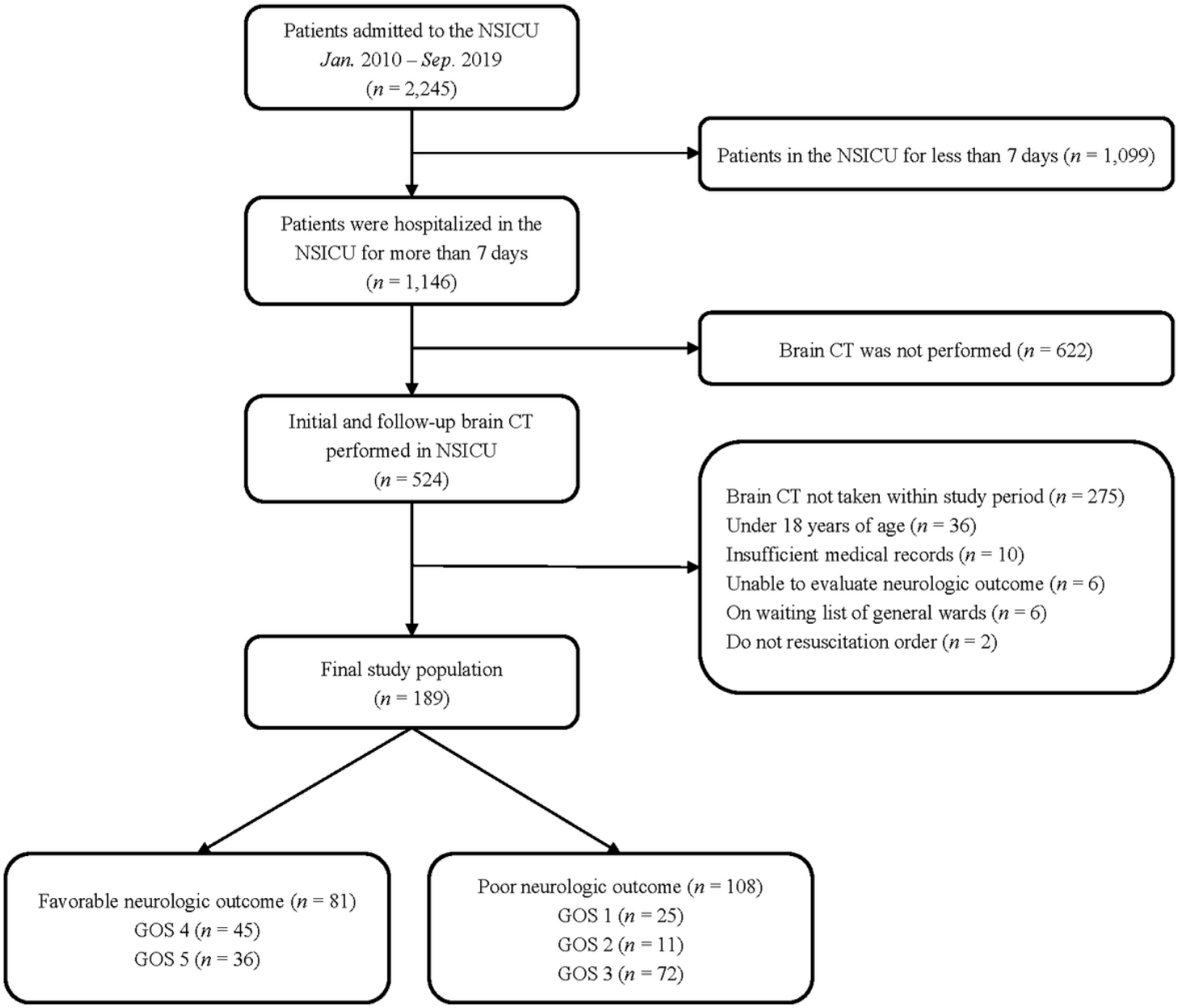
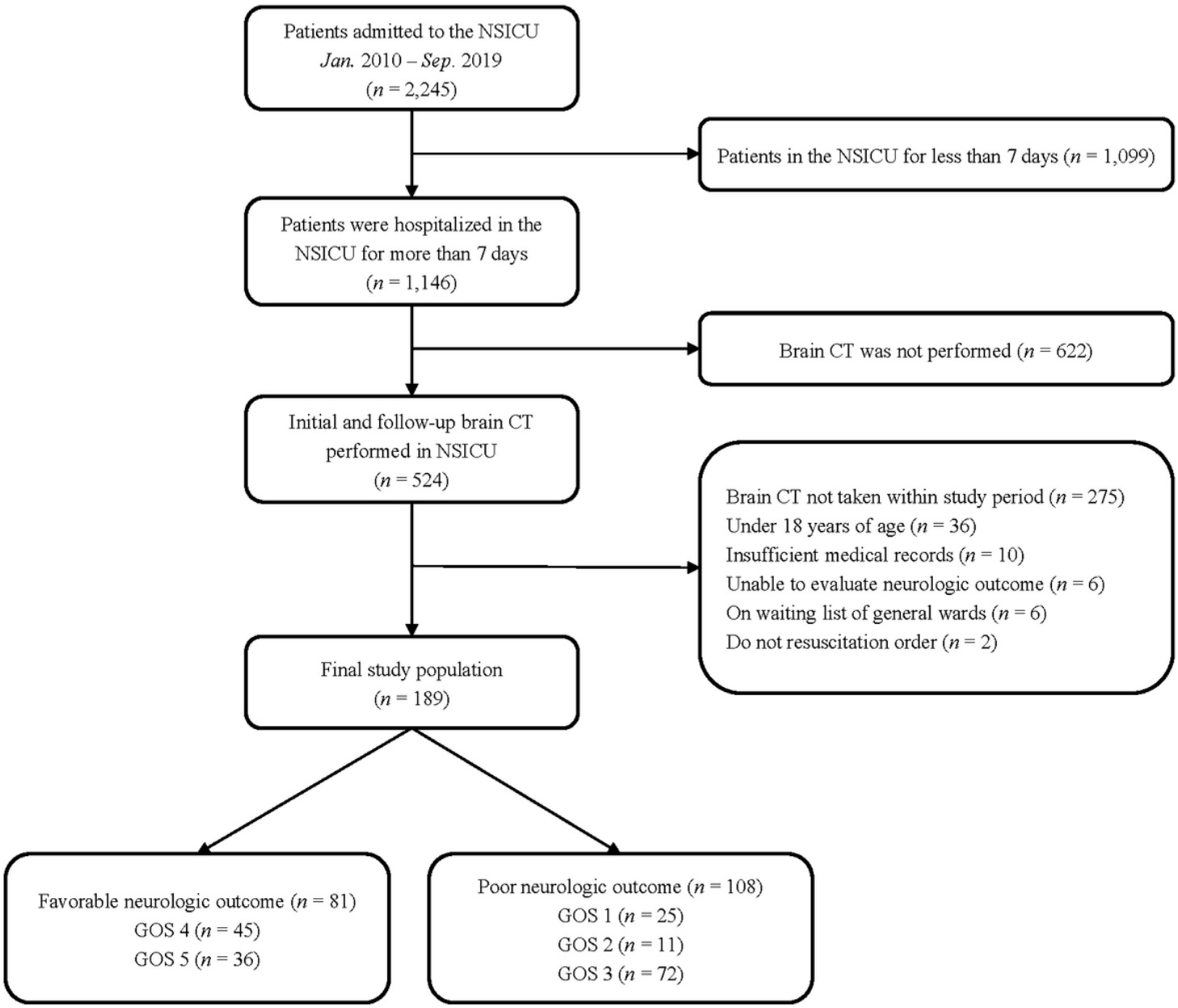


Figure 2

Study flow chart. NSICU, neurosurgical intensive care unit; CT, computed tomography; GOS, Glasgow Outcome scale.



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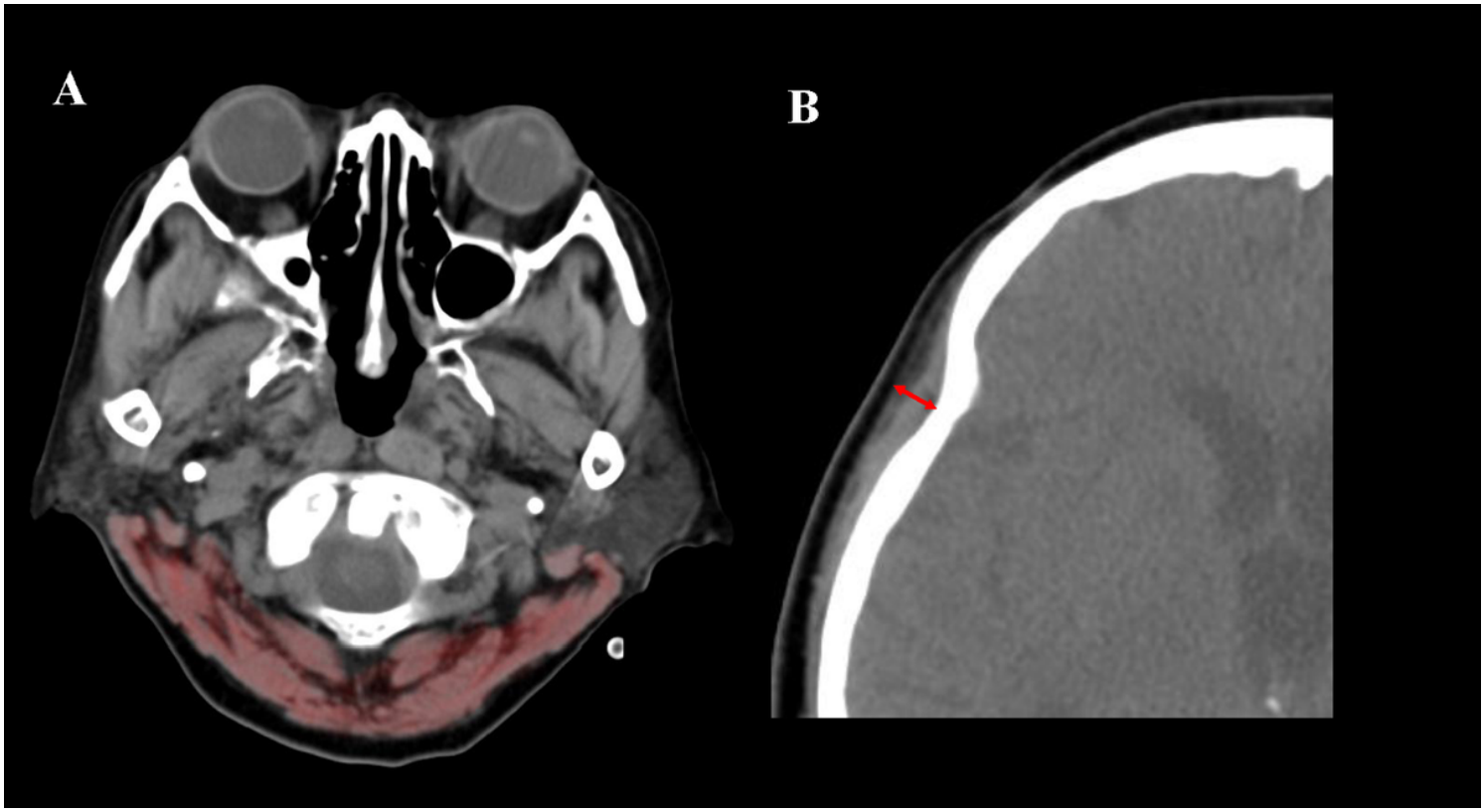
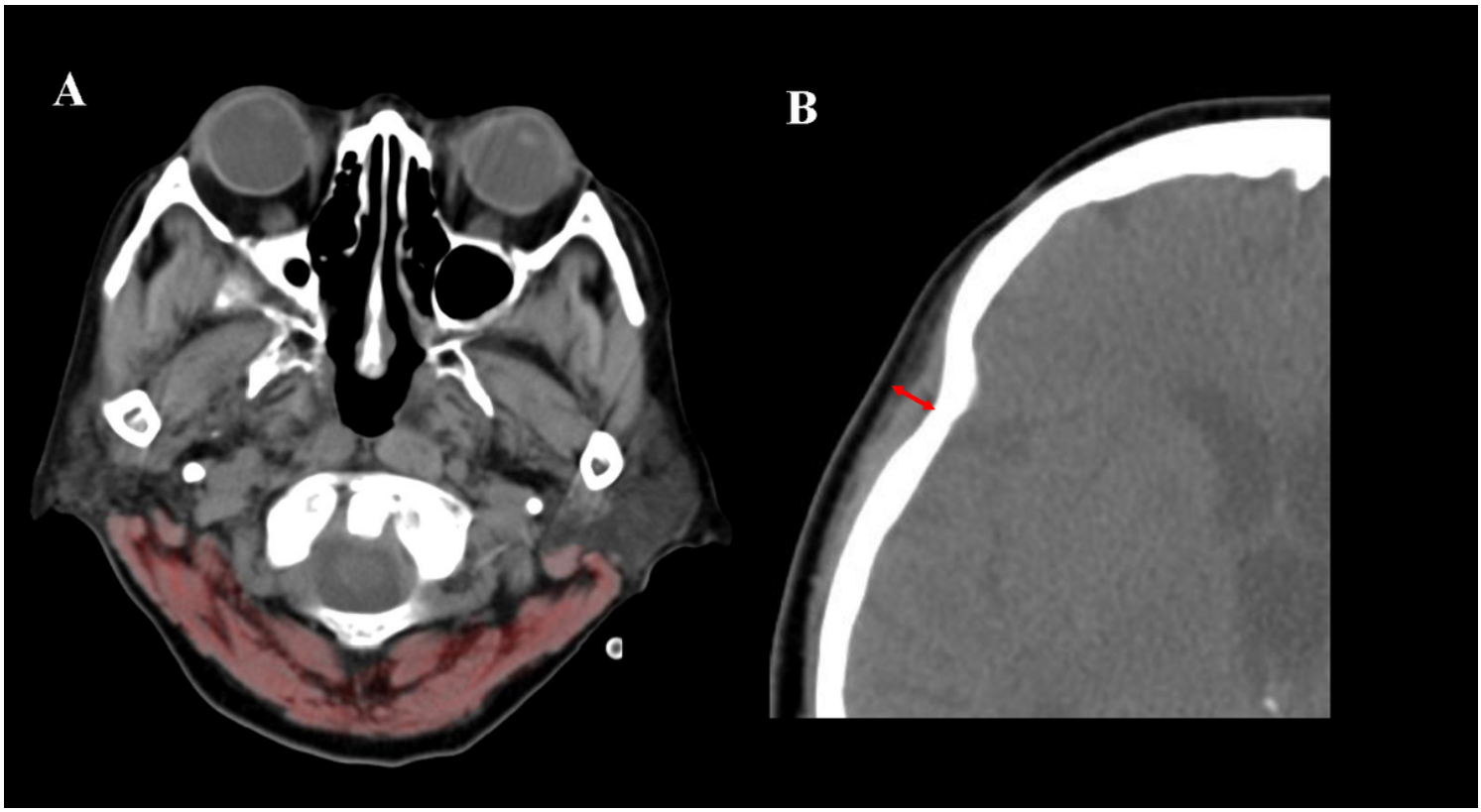


Figure 3

Methods for the measurement of cross-sectional area at the level of first cervical vertebra (C1-CSA) (A) and temporalis muscle thickness (TMT) (B) on brain computed tomography images.



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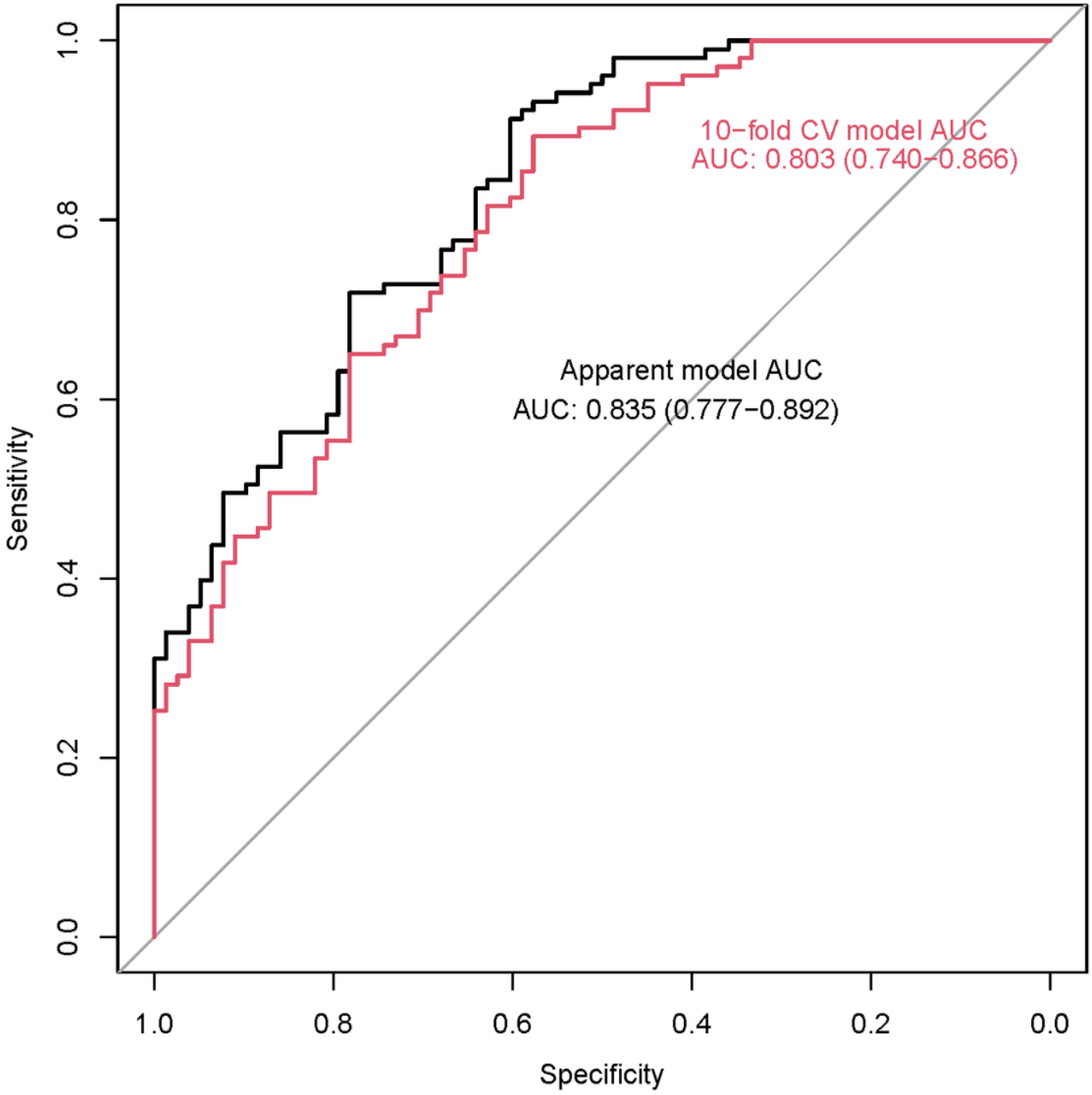
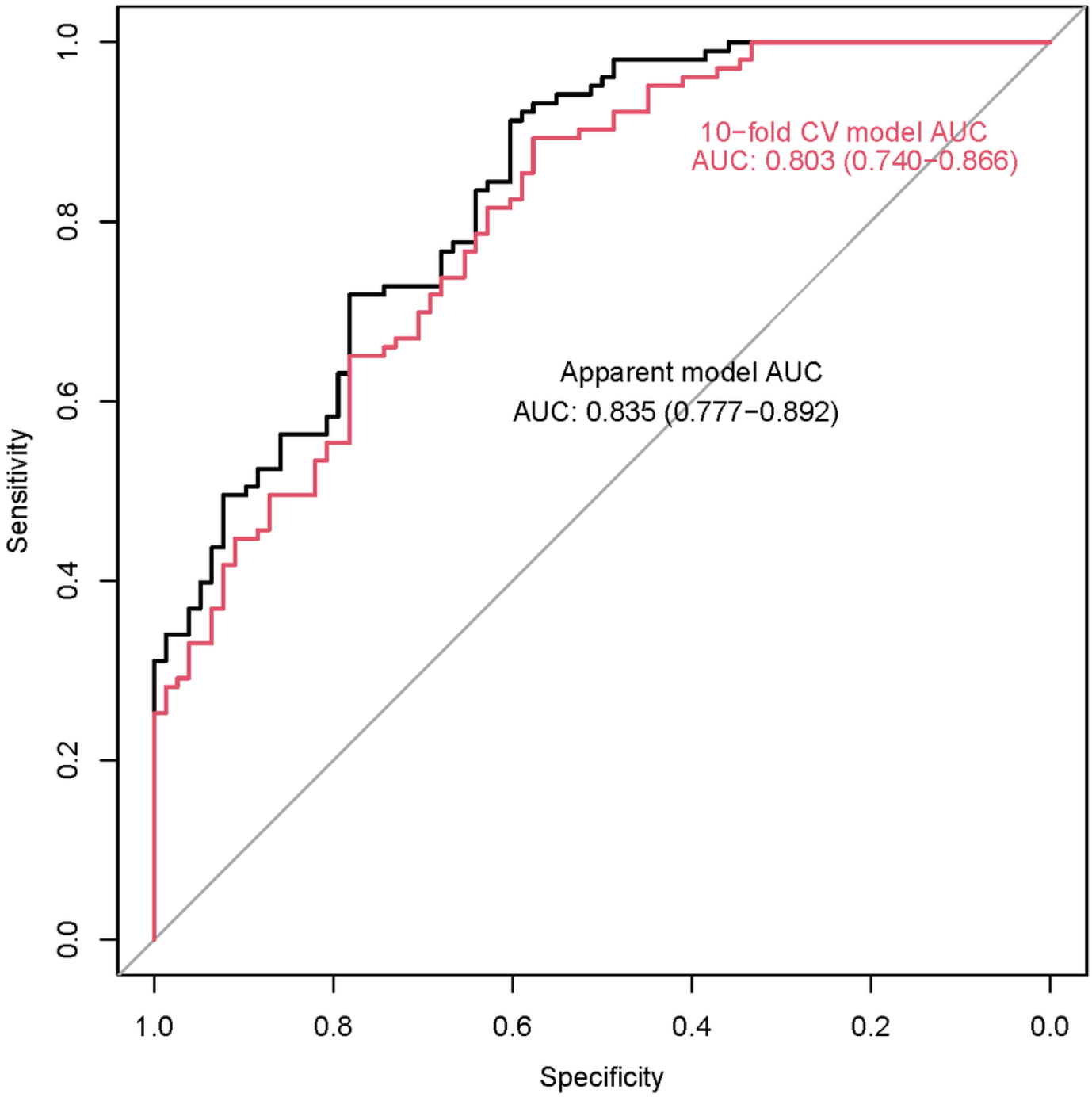


Figure 4

Adequacy of the prediction model for poor neurological outcome in this study. It was determined using the Hosmer-Lemeshow test (Chi-squared = 11.4, df = 8, p = 0.178), along with the areas under the curve (AUC: 0.803, 95% confidence interval: 0.740–0.866). In this study, 10-fold cross validation (CV) analysis was conducted to assess the internal validity.



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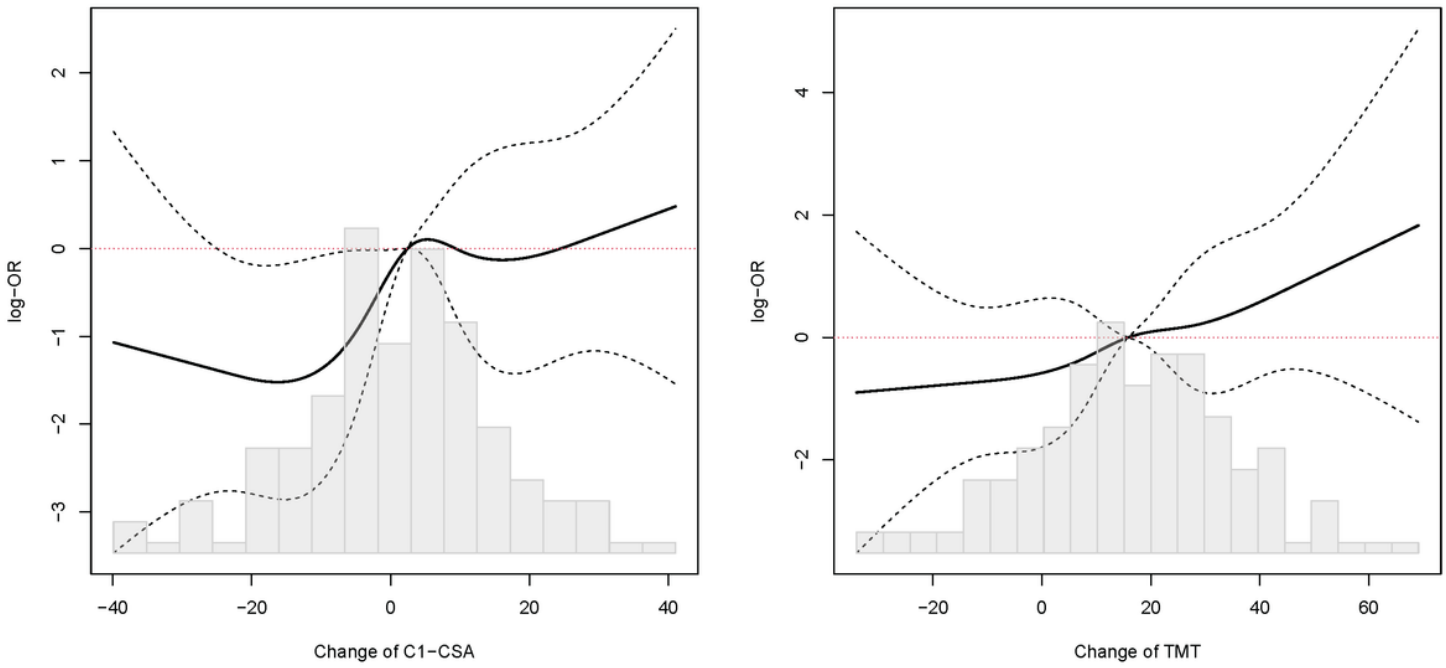
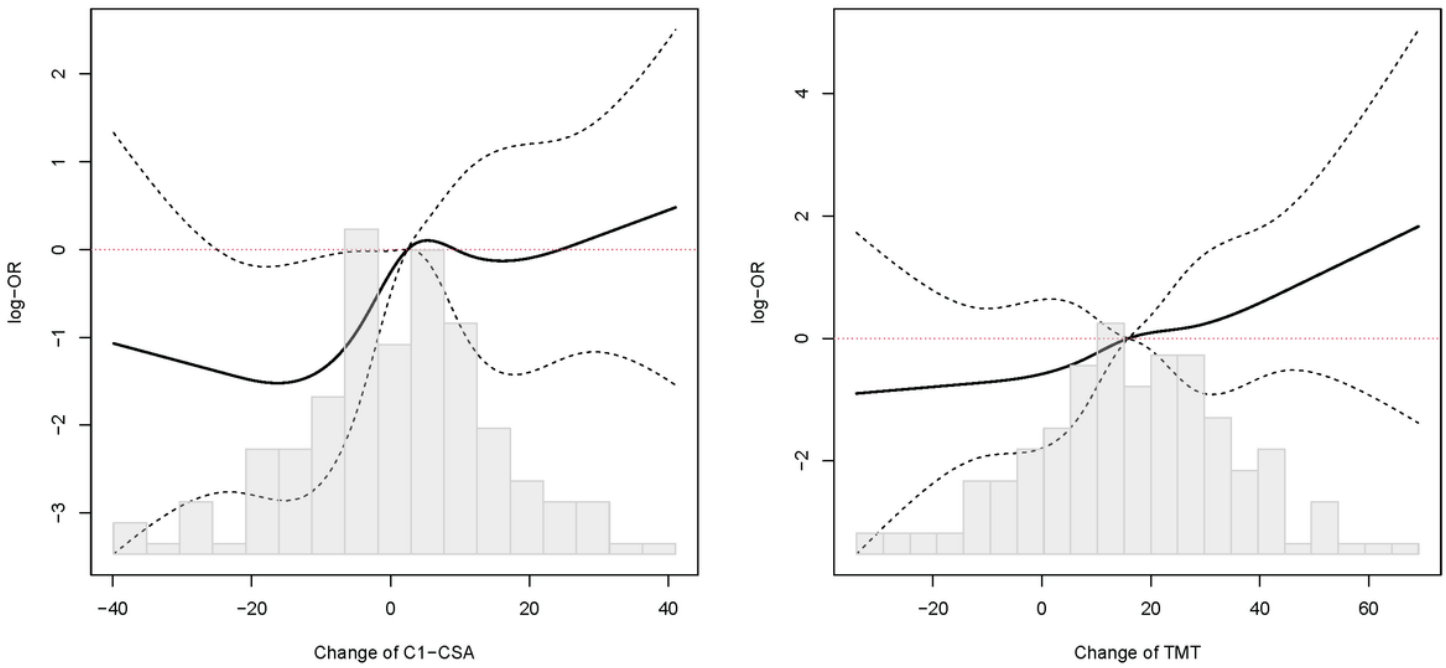


Figure 5

Association between changes in C1-CSA (A) or TMT (B) and poor neurologic outcome.OR, odds ratio; C1-CSA, cross-sectional area at the level of first cervical vertebra; TMT, temporalis muscle thickness; Change of C1-CSA, 100 × (initial C1-CSA minus follow-up C1-CSA)/initial C1-CSA; change of TMT, 100 × (initial TMT minus follow-up TMT)/initial TMT



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