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Dear Doctor,

Greetings from Micro Labs. We are happy to bring you "Stroke Newsletter" which contains latest and interesting news in the field of Neuropsychiatry.

This issue contains:

1. **Effect of sleep duration on the relationship between anxiety and health-related quality of life in post-stroke patients**
2. **Association of anti-hypertensives with acute kidney injury among acute intracerebral haemorrhage patients**
3. **Ischaemic Stroke following Chronic Subdural Haematoma: A rare complication in a common disease**
4. **Case report on Anton-Babinski syndrome in a patient with recurrent ischemic stroke**

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Looking forward to your valuable feedback
Best Regards,
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Effect of sleep duration on the relationship between anxiety and health-related quality of life in post-stroke patients

A significant relationship among anxiety, sleep duration, and quality of life in post-stroke was found in this study

Introduction

Stroke is a defined as a syndrome of acute, focal neurological deficit attributed to vascular injury (infarction, haemorrhage) of the central nervous system. Deep or lobar intracerebral haemorrhage accounts for 15% of all strokes; cerebral small vessel disorders account for 80% of them.[1] The two post-stroke disorders that are most prevalent are depression and anxiety. With a lifetime incidence of 11%, anxiety disorders are the most common type of mental health problem worldwide. In addition to raising the risk of cognitive decline, stroke death and recurrence, post-stroke anxiety further delays functional recovery and reduces health-related quality of life (HRQOL). Without adequate sleep, patients with stroke may experience chronic fatigue, cognitive decline, emotional instability, a worsening of their neurological prognosis, and a decreased quality of life (HRQOL).[2] So, this study aimed to determine if sleep duration influences the association between anxiety and health-related quality of life in post-stroke patients.

Methods

In this cross-sectional study, participants were recruited using a multistage, stratified, probability proportional to size sampling technique. The study included 856 post-stroke patients in total. HRQOL was assessed using the WHOQOL-BREF, a questionnaire developed by the World Health Organisation. Participants' answers to a questionnaire regarding their night time sleep patterns were used to determine the participants' sleep duration. The Zung self-rating anxiety scale (SAS) developed was employed to assess anxiety. The National Health and Family Planning Commission's Stroke Prevention and Treatment Engineering Committee's risk assessment form for a high-risk stroke population was used to calculate stroke risks. An analysis using multiple linear regression was used to determine the relationship between quality of life, sleep quantity, and anxiety. Using the bootstrap approach and Model 4 (parallel

mediation) of the SPSS PROCESS macro, the direct and indirect effects of sleep duration on health-related quality of life were evaluated.

Results

Patients as an entire group exhibited a mild anxiety prevalence of 24.88%, a moderate anxiety prevalence of 7.36%, and a severe anxiety prevalence of 1.29%. After controlling for covariates, the multivariate linear regression analysis revealed a negative correlation between anxiety (mild, moderate, and severe) and all HRQOL categories, indicating that anxiety may have a negative effect on all HRQOL domains. Additionally, with the exception of the environment, statistically significant associations between the amount of sleep and HRQOL were found. With the exception of the domain of social relationships, mild-to-moderate anxiety had a significant association with HRQOL through sleep duration, with sleep duration acting as a mediating factor. The psychological domain was significantly associated with mild-to-moderate anxiety, with sleep duration of >6-7 h serving as a mediating factor. The association between mild-to-moderate anxiety and quality of life in the physical, psychological, and environmental domains has been influenced by sleep duration, with 7-8 hours of sleep becoming significant (Figure 1).

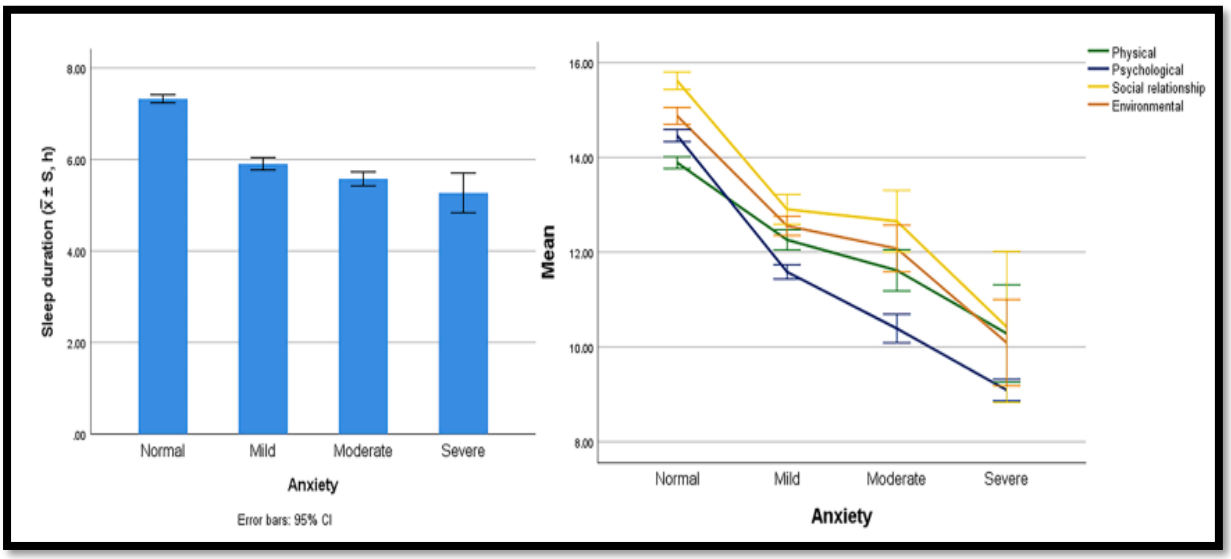

Figure 1. Graphical representation of Sleep duration and HRQOL of different anxiety levels

Discussion

In our study, we found that stroke patients had a considerable incidence of anxiety symptoms (32.48%). Our research indicates that getting sufficient rest can be beneficial in reducing anxiety, which raises HRQOL. This result is in accordance with earlier studies showing that patients who consistently get inadequate sleep are more likely to develop anxiety after a stroke. Our mediation studies show that individuals with mild to severe post stroke anxiety can benefit from altering their sleep duration, with 7-8 hours of sleep making the most difference in the physical, psychological, and environment components of HRQOL.[2] Almhdawi et al. showed that 74.5% of participants had depression, 52.9% had anxiety, and 68% had stress-related post-stroke symptoms. These levels are high and may be among the highest in the world. Grip strength and the mental component of HRQOL were significantly and adversely correlated with PSD, PSA, and PSS symptoms. Discontinuation of rehabilitation services and having comorbidities were significantly positively associated with developing PSD and PSS symptoms.[3]

Conclusion

This study revealed a significant incidence of anxiety in stroke patients and indicated that anxiety symptoms could have an impact on stroke patients' HRQOL both directly and indirectly through reducing their sleep. The results indicated that obtaining the recommended amount of sleep can help post-stroke patients' HRQOL by reducing their symptoms of anxiety.

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Association of anti-hypertensives with acute kidney injury among acute intracerebral haemorrhage patients

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Introduction

Spontaneous bleeding into cerebral tissue is known as intracerebral haemorrhage (ICH) usually results in disability or death. Majority of ICH patients have high blood pressure (BP), which increases the likelihood of intracranial haemorrhage expansion and its growth on subsequent CT scans. In order to decrease haematoma expansion, disability, and death in patients with acute ICH, acute BP reduction is regarded as standard of therapy.[1] The risk of acute kidney injury (AKI), which can occur in about 13% of patients during hospitalisation, is one factor to be taken into account in patients with intracerebral haemorrhage (ICH). Burgess et al. discovered that in patients with acute ICH, reducing systolic blood pressure (SBP) by >90 mm Hg was linked to a higher risk of AKI. In order to prevent AKI, the European Stroke Organization-Karolinska Stroke Update Conference-7 recommended maintaining SBP above 110 mm Hg while lowering it below 140 mm Hg and to avoid SBP reduction of >90 mm Hg.[2] This study aimed to investigate the possibility that patients with acute ICH might develop AKI when they are on anti-hypertensives.

More acute Blood pressure reduction is associated with acute kidney injury (AKI)

Methods

This is a large, prospective, randomised, clinical trial of patients with acute ICH that included 1000 patients with acute ICH and SBP >180mmHg to a goal of SBP of 120mm Hg or 140mm Hg in a ratio of 1:1. This study used the data from Antihypertensive Treatment of Acute Cerebral Haemorrhage (ATACH)-II clinical trial. The first-line antihypertensive drug of choice was nicardipine. Serum creatinine levels were used to categorize AKI in accordance with the Kidney Disease: Improving Global Outcomes (KDIGO) criteria. In the first 7 days in the ICU, incident AKI was defined as an increase in baseline creatinine levels of ≥0.3 mg/dL or an increase in baseline creatinine levels of at least 1.5 times within the 7-day period. Data were compared with analysis of variance, non-normally distributed values with a Mann-Whitney U test, and frequency of categorical variables with $\chi 2$ test as appropriate.

Results

Overall, AKI was present in 109 patients (10.9%). 90 patients had KDIGO stage 1, 15 had KDIGO stage 2, and 4 had KDIGO stage 3 (the most severe form of AKI). AKI and antihypertensive medicines were linked. Nicardipine was typically the only antihypertensive drug required in patients who were randomly assigned to aggressive BP treatment. Patients who had type 2 diabetes and received diltiazem as a secondary medication to nicardipine were

more likely to experience AKI. High doses of diltiazem were administered in patients with AKI as shown in Table 1.

Variable	No acute kidney injury	Acute kidney injury	P value
Antihypertensive after nicardipine			0.03
Labetalol	94 (56)	23 (56)	
Diltiazem	21 (12)	12 (29)	
Urapidil	13 (8)	1 (2)	
Other	41 (24)	5 (12)	
Atrial fibrillation	29 (3)	6 (6)	0.4
Diabetes type 2	7 (1)	4 (4)	0.002
Coronary artery disease	36 (4)	8 (7)	0.2
Cigarette use, current	224 (26)	30 (28)	0.7
Former	153 (18)	19 (17)	
Never	417 (48)	48 (44)	
Unknown	69 (8)	12 (11)	
Days to hospital discharge	15 (7 – 25)	18 (9 – 33)	0.03
Days in intensive care unit	4 (2 – 7)	6 (2 – 11)	0.003
modified Rankin Scale in survivors	3 (1 – 4)	3 (1.25–4)	0.04
EuroQOL-5D Utility Index for quality of life	0.77 (0.52–0.84)	0.69 (0.39–0.83)	0.05
Data are N(%) or median (Q1–Q3).			

Table.1- Stratified outcomes and antihypertensive drugs based on acute kidney damage

Discussion

These findings show that diltiazem shouldn't be used as a second-line treatment for acute BP lowering in patients with acute ICH when nicardipine is insufficient. Kidney blood flow is likely to be decreased by acute BP reduction and vasodilators. Patients with AKI were more likely to have type 2 diabetes; ATACH-II was the only trial to specify a BP goal below 140 mm Hg in patients with ICH; the incidence of AKI may be reduced with less stringent BP goals or standard medical care.[1] Tanaka et al. showed in the high-to-low SBP group, the chances of acute kidney damage, as well as renal and cardiac adverse events were high. The low-to-high SBP group showed the least hematoma expansion, but the most acute kidney injury, renal, and cardiac adverse events, which may have raised the patient group's mortality or disability risks.[3]

Conclusion

In patients with acute ICH, diltiazem was associated with AKI, particularly those randomised to intensive BP reduction. This study concludes that further research should be carried out to determine the effect of acute blood pressure reduction leading to AKI.

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Ischaemic Stroke following Chronic Subdural Haematoma: A rare complication in a common disease

Introduction

Antiplatelets and Anticoagulants are predisposing factors for the development of chronic subdural haematoma

Accumulation of abnormal blood that lies beneath the Dural membrane is called chronic subdural haematoma (CSDH), which is a common neurosurgical problem. With considerable levels of comorbidity and polypharmacy among individuals referred for surgery, CSDH is often considered as an old-age condition.[1] Old age and the usage of antiplatelet/anticoagulant medications are risk factors. However, a rare effect after the evacuation of CSDH is ischemic stroke. After a diagnosis of non-traumatic SDH, there is a significantly elevated risk of ischemic stroke.[2] This study aimed to describe the condition of ischaemic stroke after evacuation of chronic SDH.

Case Description

This study included two case reports of CSDH in elderly patients who suffered from an ischaemic stroke after undergoing surgery. First case report was based on a patient aged 75 years presented with the complaints of history of progressive weakness of the right upper and lower limbs along with altered sensorium for one week without any definite history of significant trauma. Patient had a past medical history of percutaneous coronary angioplasty and was on clopidogrel, and also had hypothyroidism and hypertension. On examination patient had right hemiparesis and was disoriented. Non-contrast computed tomography (NCCT) scan showed the presence of a left-sided CSDH with a significant midline shift (Figure 1A). Clopidogrel was kept on hold and underwent burrhole and evacuation of CSDH. A post-

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operative NCCT scan of head showed evacuation of haematoma (Figure 1B). Infarct in the left anterior cerebral artery territory was seen in MRI (Figure 1C). Later, patient was started on aspirin and clopidogrel along with atomoxetine for akinetic mutism and was discharged with advise and follow up. Another case describes an 84 years old patient presented with complaints of right-sided weakness and altered sensorium for 3 days. Glasgow Coma Score (GCS) upon assessment was E1VTM4 with right hemiplegia. Patient had hypertension. On admission, head's NCCT scan revealed a left-sided chronic SDH with a sizable midline shift. Additionally, a hypodense region in the left thalamus that suggested an infarct was seen (Figure 1D). Patient underwent burrhole and CSDH was removed. A post-operative CT scan revealed that the CSDH had been evacuated and the bulk effect had diminished. On the fourth postoperative day, a repeat CT scan of the head revealed that the thalamic infarct had undergone hemorrhagic change (Figure 1E). On postoperative day 9, nevertheless, his GCS further decreased and changed to E1VTM3. A fresh infarct involving the inferior division of the right middle cerebral artery region was detected during a brain MRI (Figure 1F). Patient was started on aspirin. Slowly GCS improved to E2VTM4 and was discharged.

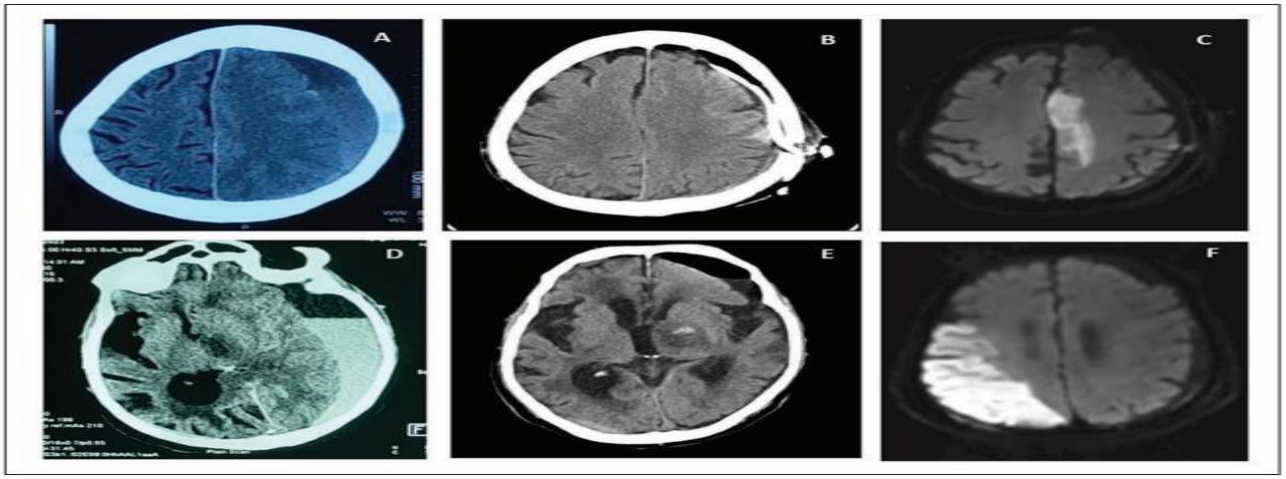

Figure 1. (A) First Case Non-Contrast CT Scan showing Left-sided CSDH and Midline Shift. (B) Post-operative Non-Contrast CT Scan Demonstrating the Removal of the Haematoma with an Installed Subdural Drain. (C) DWI Sequence Demonstrating Diffusion Restriction in Left Anterior Cerebral Artery Territory Indicative of an Infarct. (D) The second case's non-contrast CT scan revealed left-sided CSDH and a hypodense area in the left thalamus. (E) Post-operative Non-Contrast CT Scan Displaying Haematoma Evacuation and Hemorrhagic Thalamic Infarct. (F) A DWI Sequence Showing Diffusion Restriction Along the Right Middle Cerebral Artery's Inferior Division Suggestion of Infarct

Discussion

A history of anticoagulant or antiplatelet use was found in 45% of patients with CSDH. The use of these drugs increases the risk of recurrence following CSDH evacuation as well as the incidence of CSDH. From these 2 cases, it was found that stopping the antiplatelet medication during the perioperative phase may have triggered the first patient's ischemic stroke. Given the second patient's age and history of hypertension, they assume that atherosclerosis was the underlying cause of condition. However, discontinuing antiplatelet medication increases the risk of transient ischemic attacks or ischemia stroke by 40%.[2] Hamou et al. stated initiating antithrombotic medication as soon as possible after surgical treatment for CSDH, or even, in certain situations, initiating treatment under continuous antithrombotic therapy in patients who are particularly at risk. Early antithrombotic drug resumption appears to have low risk, especially when it comes to recurrent CSDH.[3]

Conclusion

Although it's uncommon, ischaemic stroke after CSDH can have serious side effects and put the patient's recovery at risk. To achieve the best results, it is important to assess the risk factors that put CSDH patients at risk for ischemic strokes and make case-by-case decisions whether to start anticoagulants or antiplatelets.

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Case report on Anton-Babinski syndrome in a patient with recurrent ischemic stroke

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Introduction

Anton-Babinski syndrome should be considered for patients who deny visual loss, exhibit unrealistic confabulation, and display evidence of bilateral occipital lobe injuries.

The Anton-Babinski syndrome, often referred to as ABS or Anton syndrome (AS), is cortical blindness accompanied by confabulation and visual anosognosia, or the denial of vision loss. It is an important neurological visual impairment resulting from abnormality or damage in the brain rather than due to eye abnormalities.[1] One of the various causes is cerebrovascular stroke involving bilateral occipital lobes with various levels of cortical blindness. Typically, ABS can cause damage to the temporal and parietal lobes, as well as the primary visual cortex (bilateral occipital lobes). A gap between the parietal lobe's white matter and functional areas like the speech and language areas may cause visual anosognosia.[2] This report presents a case of woman who experienced repeated ischemic stroke and is believed to have ABS.

Case presentation

A 50 years old female patient with the past medical history of hypertension, type-2 diabetes mellitus, and dyslipidemia had a recurrent ischemic stroke characterized by symptoms of severe headache, vomiting, and sudden onset of blurred vision. During the first ischemic stroke episode patient suffered from left hemiparesis and left homonymous hemianopia. Patient underwent thorough treatment and recovered completely without any residual disability. However, patient still had vision impairment that limited her ability to do her work and subsequently patient stopped taking medications for secondary stroke prevention. Patient had a repeat ischemic stroke, but this time there was no sensory loss or limb weakening, unlike her previous stroke. The patient denied having any vision loss despite having obvious visual impairments as shown by her frequent hitting with objects when walking or utilising furniture for ambulation. Patients visual acuity was bilateral hand movement when examined and subjected to an objective refraction test. Using the confrontation method, her visual field was lost, but tracking and light perception were restored and produced erratic results. Fundoscopic examination and anterior portion revealed normal results. The cerebral arteries were imaged using computed tomography angiography (CTA), which showed subacute hypodensities in the left occipital lobes and a well-defined hypodensity in the right parieto-occipital-temporal lobe, along with ex vacuo dilatation of temporal horn of the right lateral ventricle, in keeping with encephalomalacia changes (Figure 1). Bilateral posterior cerebral arteries (PCA) had mild-to-moderate stenoses, extensive calcification of the right vertebral artery, and diminished opacification across the P4 segment of the left PCA. Severe stenosis at the central part of the basilar artery was observed using both cerebral CTA in oblique coronal view and volume

rendering approach with maximal intensity projection (Figure 2). The radiological and clinical results supported the diagnosis of ABS. Before the subacute left PCA infarct, the patient had a right PCA infarct with left homonymous hemianopia. Notably, her visual anosognosia was only evident after the second stroke episode. Patients functional status following the recurrent ischemic stroke significantly decreased, and a thorough inpatient recovery plan was suggested.

Figure 1. Computed tomography of the brain

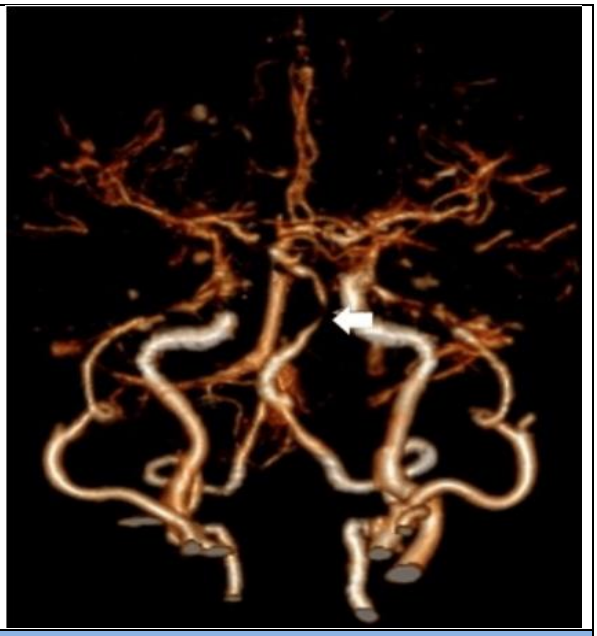

Figure 2. Cerebral computed tomography angiography

Discussion

Setting up of individualised rehabilitation goals is difficult due to uncommon and various clinical manifestations of ABS. Two methods are typically used in stroke rehabilitation: compensatory and restorative. Being difficult, ABS rehabilitation focuses mostly on compensating techniques using sensory rehabilitation, environmental modifications, and adaptive technologies to improve ADL performance. In essence, depending on the stroke patient's chance of recovery, a combination of compensatory and restorative treatments may be used.[2] In individuals with atypical vision loss and indications of occipital lobe injury, suspicion of cortical blindness and AS should be taken into consideration. Cerebrovascular disease is the most common cause of AS, as in our patient.[3] Noor et al. described an Anton-Babinski syndrome case in which the patient had visual anosognosia, as evidenced by the completely unawareness of her eyesight loss during the medical interview and cranial nerve examination. Bilateral periventricular microangiopathic changes and a filling deficiency in the right vertebral

artery's V4 segment were seen on magnetic resonance angiography (MRA). However, patient was diagnosed as having AB syndrome and continued to have cortical blindness.[4]

Conclusion

ABS should be taken into consideration for individuals who deny visual loss, display unrealistic confabulation, and show evidence of bilateral occipital lobe damage when a reference ophthalmological examination reveals no relevant results. Establishing a proper diagnosis of stroke is crucial for modifying rehabilitation goals to ensure improved functional outcomes.

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