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Original Research Article

Correlation of serum cortisol levels with the severity of acute ischemic stroke

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Abstract

Background: There is an enormous requirement to identify a biomarker for forecasting the consequences of acute stroke. The studies finding the correlation between serum cortisol levels and the severity of acute ischemic stroke (AIS) are limited in the Indian population.
Aims & Objective: The current research was conducted to determine the correlation of serum cortisol levels with the severity of AIS.
Materials and Methods: One hundred and twenty patients of AIS confirmed on magnetic resonance imaging, admitted within 24 h of the symptoms, were included in this prospective observational study. The National Institute of Health Stroke Scale (NIHSS) score was noted within 24 h of onset, and the Modified Rankin's scale (mRS) was evaluated on the fifth day and 2-3 months after the onset. The primary outcome measures were the severity of AIS using the NIHSS scale and neurological functional outcome of AIS at the end of 5 days and after 2-3 months using mRS.
Results: A statistically significant positive correlation was observed between the serum cortisol levels with the NIHSS score (r = 0.898) and mRS score at day 5 (r = 0.875) and 2-3-month follow-up (r = 0.782). A statistically significant positive correlation was observed between the serum cortisol levels and the infarct size, total cholesterol, triglycerides, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, very low-density lipoprotein cholesterol and random blood sugar levels.
Conclusion: The serum cortisol at baseline can be used as a marker of the severity measured by NIHSS and short and long-term prognosis measured by mRS after AIS.

Keywords: Acute ischemic stroke, Correlation, Modified rankin's scale, National institute of health stroke scale, Serum cortisol

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1. Introduction

Acute ischemic stroke (ASI) is a sudden onset of neurological deficit because of the blocking of one of the vessels supplying the central nervous system. The Global Burden of Diseases study stated that stroke was the second important cause of mortality internationally.[1] Further updates stated that approximately 4.66 and 5.87 million international stroke deaths occurred in 1990 and 2010, respectively.[2,3] Hence, there was a 26 % rise in stroke mortality during the past two decades worldwide. According to the World Health Organization, nearly 15 million individuals are affected globally by stroke, out of which 5 million die and 5 million survive with significant disability.[4]

The stress response after the stroke activates the hypothalamic-pituitary-adrenal (HPA) axis and the sympathetic nervous system.[5] This change in the HPA axis makes measurable alterations in the endocrine system. Cortisol is one of the HPA axis-related hormones with a strong circadian rhythm. The levels of cortisol are highest in the early hours of the day and decline later.

It was reported that higher cortisol levels were noted in patients with AIS and subarachnoid haemorrhage. Similarly, it was reported that bigger infarct volume, greater stroke severity and poor outcomes, including death, were noted in patients with higher serum and urinary cortisol levels.[6-8]

The National Institute of Health Stroke Scale (NIHSS) can gauge the severity of a stroke at presentation. It was first used by Brott et al. to assess the severity of stroke.[9] Since then, the NIHSS scale has been widely used in clinical practice. The Modified Rankin's scale (mRS) allows for

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quantification of the disability caused by an AIS. Originally introduced by Dr. John Rankin in the year 1957, it was modified later in the year 1988 by Dr. Van Swieten.[10] It is a structured interview used to determine the functional neurological outcome of an AIS. Currently, measurement of serum cortisol levels requires sophisticated equipment and specific reagents, which can be difficult to procure in a resource-limited setting. The NIHSS score and the mRS are easy to carry out on patients as they are clinical tests.

There exists a lot of controversy regarding the serum cortisol levels and the severity and prognosis of AIS. There is an enormous requirement to identify a biomarker for forecasting the consequences of acute stroke. No biomarker exists that can accurately predict the severity of AIS, or predict the risk of developing severe ischemic stroke in patients with several risk factors such as hypertension, diabetes mellitus, obesity and metabolic syndrome. The studies finding the correlation between serum cortisol levels and the severity of AIS are limited in the Indian population. The current research was conducted to determine the correlation between serum cortisol levels and the severity of AIS.

2. Materials and Methods

The present hospital-based prospective observational study was conducted from April 2023 to January 2024 in Western Maharashtra, India. An approval was obtained from the Institutional Review Board (Letter # ADM/ 2022-2023/418). All the participants gave a written informed consent before the commencement of the study. Participants of age ≥ 18 years, presenting within 24 h of new-onset focal neurological deficit, and confirmed to be an AIS on magnetic resonance imaging (MRI) were included. Patients of haemorrhagic stroke confirmed on computed tomography (CT) or MRI brain, malignancy, spontaneous intracranial haemorrhage (subdural hematomas, subarachnoid and epidural haemorrhages), acute or chronic inflammatory conditions, febrile illness, and receiving immunosuppressive agents, corticosteroids and psychotropic drugs were excluded.

A formula used to calculate the sample size was n[11] = $[(Z_\alpha+Z_\beta)/C]^2 + 3$. We have taken $Z_\alpha = 2.58$ (a standard normal variate at 1% type 1 error, p-value < 0.01), $Z_\beta = 0.84$ (the standard normal deviate for β power 80 % at type II error), and $C = 0.5 * \ln[(1+r)/(1-r)] = 0.3205$. Urra X et al. reported a correlation of = 0.31.[12] Hence, the calculated sample size was 120 patients. The detailed history and clinical examination were noted. The diagnosis of AIS was established using a GE SIGNA Pioneer 3 Tesla 96-channel wide-bore MRI machine (GE Healthcare, USA). The laboratory tests were conducted within the first 24 h of the onset of AIS. The NIHSS score was noted within 24 h of onset, and the mRS was assessed on the fifth day and 2-3 months after onset. Serum cortisol levels were measured using the enzyme-linked fluorescent assay method (VIDAS® Cortisol S kit, bioMérieux, France). According to the manufacturer's specifications, the normal reference range for serum cortisol is 5.5 to 28.7 µg/dL.

The primary outcome measures were to find the correlation between serum cortisol levels and severity of AIS using the NIHSS scale, whereas the secondary outcome measures were to find the correlation of serum cortisol levels with neurological functional outcome at the end of 5 days and after 2-3 months using mRS.

2.1. Statistical analysis

The data on discrete variables are shown as n (%), and the data on normally distributed quantitative variables are presented as mean and standard deviation (SD). The Chi-square test or Fisher's exact probability test was used to compare the discrete variables. The Pearson correlation analysis was used to find the correlation. A p-value less than 0.05 was considered to be statistically significant. The data was analyzed using Statistical Package for Social Sciences (SPSS ver 24.0, IBM Corporation, USA) for MS Windows.

3. Results

In the present hospital-based prospective observational study, 175 patients were assessed for eligibility. Fifty-five patients were excluded (42 had an acute haemorrhagic stroke, 7 had demyelinating lesions, 3 had space-occupying lesions, and 3 patients refused to enrol for the study). In all, 120 participants were enrolled in the research (**Figure 1**). The baseline characteristics of the study participants are evident from **Table 1**.

Table 1: Demographic and clinical profile of the study participants

Variables	**n (%)**
Age groups in years	
<40	12 (10.0)
40 < 50	12 (10.0)
50 < 60	29 (24.2)
60 < 70	27 (22.5)
70 < 80	27 (22.5)
≥80	13 (10.8)
Mean age in years ± SD	56.3 ± 14.3
Gender	
Males	86 (71.7)
Females	34 (28.3)
Comorbidity	
Hypertension	34 (28.3)
Diabetes mellitus	24 (20.0)
Ischemic heart disease	11 (9.2)
Dyslipidemia	5 (4.2)
Hypothyroidism	4 (3.3)
Chronic kidney disease	3 (2.5)
Chronic obstructive pulmonary disease	3 (2.5)
Other	2 (1.7)

Severity of stroke (NIHSS)	
Minor (1-4)	20 (16.7)
Moderate (5-15)	81 (67.5)
Moderate to severe (16-20)	11 (9.2)
Severe (21-42)	8 (6.7)
Serum cortisol in µg/dL)	
Normal (5.5-28.7)	74 (61.7)
Abnormal (>28.7)	46 (38.3)

SD – Standard deviation
NIHSS - National Institute of Health Stroke Scale

The neurological functional outcomes at day 5 and 2-3-month follow-up are evident in **Table 2**. The participants with a high serum cortisol level (>28.7 µg/dL) at the time of admission were likely to have moderate to severe and severe stroke (**Table 3**). A substantially higher proportion of cases with raised serum cortisol levels (>28.7 µg/dL**)** had a higher incidence of severe neurological functional outcome at day 5- and 2- 3-month follow-up compared to the group of cases with normal serum cortisol levels at the time of admission (**Table 4**, **Table 5**). A significantly higher proportion of patients with raised serum cortisol levels had a higher incidence of larger infarct size (≥50 cm^3) than those with normal serum cortisol levels. A significantly higher proportion of cases with raised serum cortisol levels (>28.7 µg/dL) had higher proportions of abnormal levels of lipid profile parameters such as triglycerides, high-density lipoprotein (HDL) cholesterol, low-density lipoprotein (LDL) cholesterol, very low-density lipoprotein (VLDL) cholesterol and random blood sugar level (RBSL) compared to the group of cases with normal serum cortisol levels (**Table 6**).

A statistically significant positive correlation was observed between the serum cortisol and the NIHSS score (r = 0.898, p-value = 0.001, **Figure 2**), and mRS score at day 5 (r = 0.875, p-value = 0.001, **Table 3**). A statistically significant positive correlation was observed between the serum cortisol and mRS score at 2-3-month follow-up, the infarct size, total cholesterol, triglycerides, LDL cholesterol, VLDL cholesterol and RBSL, but a significant negative correlation with HDL cholesterol (**Table 7**).

Table 2: Neurological functional outcome at day 5 and 2-3-month follow-up

Functional outcome	**mRS Score**	**mRS at day 5 n (%)**	**2-3 months follow-up n (%)**
No symptom	0	0 (0.0)	33 (30.0)
No significant disability	1	8 (6.7)	41 (37.3)
Slight disability	2	42 (35.0)	23 (20.9)
Moderate disability	3	37 (30.8)	7 (6.4)
Moderate to severe disability	4	13 (10.8)	3 (2.7)
Severe disability	5	10 (8.3)	0 (0.0)
Dead	6	10 (8.3)	3 (2.7)
Total		120 (100.0)	110 (100.0)

mRS - Modified Rankin's scale

Table 3: Association between serum cortisol levels and severity of acute ischemic stroke (NIHSS score).

	Serum cortisol Level			
Severity of stroke (NIHSS score)	**Normal (5.5-28.7 µg/dL)**	**High (>28.7 µg/dL)**	**Total**	**p-value**
	n (%)	n (%)	n (%)	
Minor (1-4)	20 (27.0)	0 (0.0)	20 (16.7)	0.001
Moderate (5-15)	54 (73.0)	27 (58.7)	81 (67.5)	
Moderate to severe (16-20)	0 (0.0)	11 (23.9)	11 (9.2)	
Severe (21-42)	0 (0.0)	8 (17.4)	8 (6.7)	
Total	74 (100.0)	46 (100.0)	120 (100.0)	

Fisher's exact test was used
NIHSS – National Institute of Health Science Score

Table 4: Association between serum cortisol levels and neurological functional outcome at day 5 (mRS score).

	Serum cortisol Level			
Neurological functional outcome (mRS score)	**Normal (5.5-28.7 µg/dL) n (%)**	**High (>28.7 µg/dL) n (%)**	**Total n (%)**	**p-value**
No symptom (0)	0 (0.0)	0 (0.0)	0 (0.0)	0.001
No significant disability (1)	8 (10.8)	0 (0.0)	8 (6.7)	
Slight disability (2)	42 (56.8)	0 (0.0)	42 (35.0)	
Moderate disability (3)	24 (32.4)	13 (28.3)	37 (30.8)	
Moderate to severe disability (4)	0 (0.0)	13 (28.3)	13 (10.8)	
Severe disability (5)	0 (0.0)	10 (21.7)	10 (8.3)	
Dead (6)	0 (0.0)	10 (21.7)	10 (8.3)	
Total	74 (100.0)	46 (100.0)	120 (100.0)	

Fisher's exact test was used

mRS – Modified Rankin"s Score

Table 5: Association between serum cortisol levels and neurological functional outcome at the end of 2-3 months (mRS score).

	Serum cortisol level			
Neurological functional outcome (mRS score)	**Normal (5.5-28.7 µg/dL) n (%)**	**High (>28.7 µg/dL) n (%)**	**Total n (%)**	**p-value**
No symptom (0)	33 (44.6)	0 (0.0)	33 (30.0)	0.001
No significant disability (1)	33 (44.6)	8 (22.2)	41 (37.3)	
Slight disability (2)	8 (10.8)	15 (41.7)	23 (20.9(	
Moderate disability (3)	0 (0.0)	7 (19.4)	7 (6.4)	
Moderate to severe disability (4)	0 (0.0)	3 (8.3)	3 (2.7)	
Severe disability (5)	0 (0.0)	0 (0.0)	0 (0.0)	
Dead (6)	0 (0.0)	3 (8.3)	3 (2.7)	
Total	74 (100.0)	36 (100.0)	120 (100.0)	

Fisher's exact test was used

mRS – Modified Rankin's Scale

Table 6: Association of the size of the infarct, lipid profile and RBSL according to serum cortisol levels.

		Serum cortisol level			
Parameters		**Normal (5.5-28.7 µg/dL) n (%)**	**High (> 28.7 µg/dL) n (%)**	**Total n (%)**	**p-value**
Size of infarct	<50 cm^3	19 (25.7)	0 (0.0)	19 (15.8)	0.001*
	≥50 cm^3	55 (74.3)	46 (100.0)	101 (84.2)	
Total cholesterol	Normal (<200)	72 (97.3)	41 (89.1)	113 (94.2)	0.105*
	Abnormal (≥200)	2 (2.7)	5 (10.9)	7 (5.8)	
Triglycerides	Normal (<150)	71 (95.9)	31 (67.4)	102 (85.0)	0.001*
	Abnormal (≥150)	3 (4.1)	15 (32.6)	18 (15.0)	
HDL Cholesterol	Normal (>40)	64 (86.5)	24 (52.2)	88 (73.3)	0.001**
	Abnormal (≤40)	10 (13.5)	22 (47.8)	32 (26.7)	
LDL Cholesterol	Normal (<130)	71 (95.9)	35 (76.1)	106 (88.3)	0.002*
	Abnormal (≥130)	3 (4.1)	11 (23.9)	14 (11.7)	
VLDL Cholesterol	Normal (<30)	68 (91.9)	29 (63.0)	97 (80.8)	0.001**
	Abnormal (≥30)	6 (8.1)	17 (37.0)	23 (19.2)	
RBSL	Normal (≤140)	70 (94.6)	16 (34.8)	86 (71.7)	0.001*
	Abnormal (>140)	4 (5.4)	30 (65.2)	34 (28.3)	

*Fisher's exact test was used, **The Chi-Square test was used

HDL – High-density lipoprotein, **LDL**- Low-density lipoprotein,

VLDL – Very low-density lipoprotein, **RBSL** – Random blood sugar level

Table 7: Spearman's correlation of serum cortisol levels with mRS score at 2-3 months, the size of the infarct, lipid profile and RBSL.

Correlation between serum cortisol	R-value	p-value
mRS score at 2-3 months	0.782	0.001
The size of the infarct	0.792	0.001
Total cholesterol	0.367	0.001
Triglycerides	0.432	0.001
LDL cholesterol	0.530	0.001
HDL cholesterol	-0.378	0.001
VLDL cholesterol	0.497	0.001
RBSL	0.634	0.001

Spearman's correlation was used

mRS - Modified Rankin's scale; **RBSL** – Random blood sugar level; **LDL** – Low-density lipoprotein; **HDL** - High-density lipoprotein; **VLDL** – Very-low-density lipoprotein

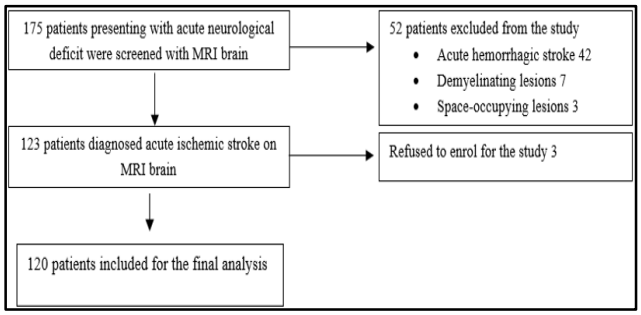
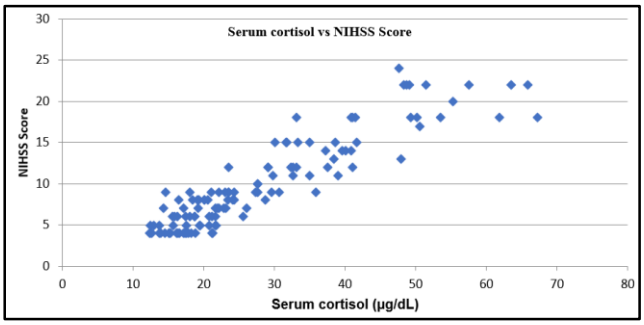
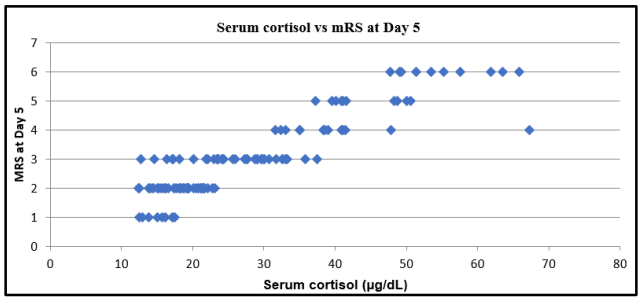


Figure 1: Flow chart



NIHSS - National Institute of Health Stroke Scale

Figure 2: Scatter diagram showing a positive correlation between serum cortisol levels and NIHSS score.



mRS - Modified Rankin's scale

Figure 3: Scatter diagram showing a positive correlation between serum cortisol levels and mRS at day 5.

4. Discussion

AIS remains a significant cause of death and illness in the world, but fatalities and illnesses are higher in developing countries than in developed nations. This is due to a lack of required infrastructure, early diagnosis, and treatment. Also, there is a lack of a definitive plasma biomarker which can precisely forecast the severity of ischemic strokes and their short/long-term prognosis regarding neurological outcome and residual morbidity.

The acute, severe illness activates the HPA axis, leading to an increase in cortisol levels. The advantages of elevated cortisol levels are the mobilization of glucose from the liver and adipose tissue and the potentiation of cardiovascular output.[13,14] It was reported that the serum cortisol levels become high if the illness is more severe.[15,16] Following an acute stroke, prolonged HPA axis activation may also occur for reasons specific to stroke. These reasons include cytokine release following neuronal injury.[17]

In the present study, the serum cortisol levels showed a markedly positive correlation with the NIHSS score (r = 0.898, p-value = 0.001) and mRS score at day 5 (r = 0.875, p-value = 0.001) and 2-3-month follow-up (r = 0.872, p-value = 0.001). Also, there was a marked positive correlation of serum cortisol levels with the infarct size, total cholesterol, triglycerides, LDL cholesterol, VLDL cholesterol and RBSL levels and a negative correlation with HDL cholesterol.

Saini G et al.[18] reported that there was a positive correlation between stroke severity measured by NIHSS and serum cortisol (r = 0.785, p-value < 0.001); stroke outcome measured by mRS at 1, 4, and 24 weeks; and serum cortisol (p-value < 0.001 and r = 0.676, 0.654, 0.650, respectively). The study further stated that the severity of stroke, measured by NIHSS, and outcomes measured by mRS were significantly associated with mean baseline serum cortisol level. The severity was higher and the outcome was poor in patients with a high mean baseline serum cortisol level. In the present study, we measured the serum cortisol levels only

once at admission and mRS was measured at day 5 and 2-3-month follow-up.

A prospective observational study conducted in Hyderabad, India, by Vempaty S and Reddy SV[19] reported that high serum cortisol levels were associated with lower Scandinavian Stroke Scale (SSS) (r = - 0.990, p-value < 0.001). However, we have used NIHSS in the present study to assess the severity of stroke. A study conducted in Kerman, Iran, by Iranmanesh F et al. concluded that ischemic stroke is linked with a change in serum cortisol level, but it has no predictive value. The study further stated that logistic regression analysis showed that there was no significant correlation between cortisol level and death.[20] A study conducted in Tumakuru, Karnataka, India by Jnanendrappa KH reported that in patients with AIS, elevated serum cortisol levels at the time of admission correlated with the NIHSS and mRS at 15 days follow-up.[21] In the above-mentioned study, they did a CT scan, whereas we did an MRI of all the patients. A study conducted in Bhubaneswar, Odisha, India by Konduru A et al. observed that the serum cortisol level had a significant positive correlation with the deterioration of stroke and is a prognostic indicator of the severity of stroke. The study further observed that the serum cortisol level test is easy to conduct and may be used as a biomarker for stroke severity forecast.[22]

A study conducted in India by Murugaraj R et al. reported that as the serum cortisol increased, the SSS score decreased and hence showed more severe stroke and worse prognosis. The study further stated that as cortisol value increased in stroke patients, the mRS also increased, indicating poor prognosis. A markedly positive correlation was observed between serum cortisol and mRS at the end of three months follow-up (r = 0.819).[23] In the present research, we used the NIHSS score.

A study conducted in Barcelona, Spain by Urra X et al. observed that the NIHSS score was correlated with cortisol (r=.31, p-value = 0.05). [12] Zi WJ and Shua J conducted a study in the People's Republic of China stated that there was a positive correlation between serum cortisol and the NIHSS (r = 0.298, p-value = 0.0001), glucose levels (r = 0.324, p-value = 0.0001) and infarct volume (r = 0.328, p-value = 0.0001).[24] In the afore-mentioned study, the blood was collected for analysis of serum cortisol on the next morning (6.00 a.m.) of the day of admission. We collected the blood samples within 24 hours of admission. A study by Allwyn Yabesh TA in Chennai, Tamil Nadu, India, observed that the elevated serum cortisol at the time of admission correlated with the NIHSS and the mRS at 15 days follow-up.[25] In the present study, we assessed the functional outcome at 2-3 months of follow-up. A study conducted in Chengalpattu, Tamil Nadu, India by Ilanchetchenni K et al. reported that the SSS and serum cortisol were significantly inversely correlated (r = - 0.984). High serum cortisol levels were associated with lower SSS scores (p-value < 0.001).[26] In the present study, we used the NIHSS score. The study described above used a CT scan, whereas we used an MRI scan. Singhal K et al. observed that the serum cortisol levels increased significantly with stroke severity, with mean levels of 360.2 ± 50.5 nmol/L for mild, 478.3 ± 65.7 nmol/L for moderate, and 612.5 ± 80.3 nmol/L for severe strokes (p < 0.001). The study further stated that a strong positive correlation (r = 0.78) was observed between cortisol levels and NIHSS scores.[27]

5. Limitations

The correlation of serum cortisol levels with other markers such as C-reactive protein, HbA1C, and N-terminal pro-B-type natriuretic peptide was not considered. This study only measured the free serum cortisol level and not cortisol bound to cortisol-binding globulin/albumin, which may be affected by multiple factors, hence becoming difficult to correlate with other factors. The serum cortisol was tested within 24 h of admission, but the exact time of the collection was not noted. Repeated measurement of the serum cortisol may influence the results. The adrenocorticotropic hormone, noradrenaline, adrenaline and other hormones involved in the stress response were not measured. We have not included the sites of infarction. Multi-centric prospective observational studies involving adjustment for co-morbidities with a larger population, repeated cortisol measurements and longer follow-up periods are needed to effectively gauge the correlation of serum cortisol with the severity of AIS. Interventional studies targeting cortisol modulation may also offer new therapeutic avenues for improving stroke care.

6. Conclusions

The serum cortisol levels were markedly positively correlated with the NIHSS score and mRS score at day 5 and the 2-3-month follow-up. The serum cortisol levels were markedly positively correlated with the size of the infarct, total cholesterol, triglycerides, LDL cholesterol, VLDL cholesterol and RBSL levels, but a negative correlation with HDL cholesterol. A significantly higher proportion of cases with abnormal or raised serum cortisol levels had a higher incidence of larger infarct size (≥50 cm^3) and had higher proportions of abnormal levels of lipid profile parameters such as triglycerides, HDL cholesterol, LDL cholesterol, VLDL cholesterol and RBSL.

7. Ethical No.

ADM/2022-2023/418

8. Source of Funding

None.

9. Conflict of Interest

None.

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