SUMMARY

Background

Acute cerebral injury is typified in acute stroke. Stroke is a major stressful event to the sufferers, physically emotionally and economically, the impact of which tends to endure beyond the acute phase of the event. The cerebral and systemic pathophysiological events triggered off as a result of acute stroke include ionic shifts, haemodynamic changes, endocrine and metabolic alterations as well as inflammatory responses. Some of these events, both local and systemic, may be adaptive, but recent evidence suggests that some of them may be damaging, and may potentiate the ischaemic damage to neurones. These effects could induce secondary brain damage in acute stroke.

This study set out to find the extent of acute stress response following stroke, measured by single cortisol estimate in the acute phase of the illness. It also sought to find out the benefits or otherwise of such responses in terms of stroke severity and outcome.

Method

A prospective study was carried out among patients with acute ischaemic stroke as shown by the Siriraj criteria at the Nnamdi Azikiwe University Teaching Hospital Nnewi. A total of fifty (50) patients, 22 males (44%) and 28 females (56%) who met
the diagnostic criteria were studied. Another group of fifty subjects matched for age and sex to the patients, were included in the study as controls, and made up of two subgroups of 25 each comprising 11 males (44%) and 14 females (56%). One subgroup C1 was made up of individuals who had hypertension and/or diabetes mellitus, while the other subgroup C2 was made up of healthy individuals. The age range of the study populations was 30-90 years. The mean ages of the males in the patient group, controls groups 1 and 2 were 59.30 ± 13.40 years, 59.00 ± 13.70 years and 58.10 ± 12.60 years respectively. The mean ages of the females were 66.60 ± 12.60 years, 66.60 ± 14.20 years and 65.90 ± 13.5 years respectively for the patients and the control groups 1 and 2. The patients and the controls were clinically assessed, and their morning serum cortisol, and random blood sugar estimations were done. The National Institutes of Health Stroke Score (NIHSS) (severity score) on admission, as well as their functional outcome at 30 and 90 days after stroke, using the modified Rankin scale (mRs) were determined. The mortality at 7, 30 and 90 days after stroke were also estimated.

**Results**

The results showed that serum cortisol levels were significantly higher in stroke patients when compared with controls (280.60 ± 101.30 ng/ml for the patients, 182.70 ± 39.20 ng/ml and 146.80 ± 21.20 ng/ml for C1 and C2 respectively), $F = 32.7$ p<0.001. Male patients had higher cortisol levels than male controls: $326.7 ± 93.9$ ng/ml for the
patients, 191.2 ± 20.4 ng/ml and 147.4 ± 26.2 ng/ml for control groups 1 and 2 respectively (f = 9.5, p< 0.025). Female patients also had higher cortisol level than controls: 244.3 ± 93.1 ng/ml for the patients 176.1 ± 48.5 ng/ml and 146.4 ± 17.4 ng/ml for the controls 1 and 2 respectively. (F=10.1, p<0.025)

Random blood sugar was significantly elevated among the patients 7.40 ± 4.86mmol/l, when compared with the controls 5.22 ± 2.03 mmol/l for C1 and 4.40 ± 0.47 mmol/l for C2, F = 4.92 p< 0.01.

Serum cortisol correlated with random blood sugar in the patient group (p<0.01, coefficient of correlation 0.90).

All patients had moderately severe stroke (NIHSS 5-20) on presentation. The mean admission cortisol levels showed no relationship with stroke severity at presentation: correlation coefficient r = 0, p>0.999 for mild stroke (NIHSS 1-4): r = 0.19 n=50, p>0.1 for moderate stroke (NIHSS 5-20): and r = 0 p>0.999 for the severe stroke.

Admission serum cortisol in the patients did not relate to functional outcome at 7, 30 and 90 days after the stroke event. Viz at 7th day: good outcome (mRs 0-2): – n = 0, r = p>0.999; moderate outcome (mRs 3-4): n= 41, mean cortisol 261.51 ± 124.1ng/ml, r = -0.36 p < 0.05; poor outcome (mRs 5-6): n=9, mean cortisol 378.0 ± 182.0, r = 0.48, p > 0.01. For the 30th day good outcome: n=9, cortisol 235.4 ± 79.7, r = 0, p>0.999; moderate outcome: n=21, cortisol 248.34 ± 87.10, r=-0.18, p>0.1;
poor outcome: \( n=12 \), cortisol 391.0± 58.9, r=0 p> 0.9999. For 90\(^{th}\) day, good outcome: \( n=36 \), cortisol 244.28 ± 82.3ng/ml, r = -0.009, p>0.8; moderate outcome: \( n=0 \); r=0, p>0.9999; poor outcome: \( n=14 \), cortisol 388.29 ± 31.1 ng/ml r = 0 p> 0.999.

The difference in the mean cortisol levels of the fatalities within 7 days of 417±117ng/ml \( n=2 \) compared with those of the survivors 275 ± 97.6 \( n=48 \) did not reach significance; (\( z = 1.69 \) p>0.05.). Though the mean admission serum cortisol level was not found predictive of 7\(^{th}\) day mortality after stroke. It however related significantly to the 30\(^{th}\) and 90\(^{th}\) day case fatalities. The admission cortisol level for those that died between 7 and 30 days, \( n=10 \), was 387.7 ± 50.6 ng/ml compared with that of the living within the period: 253.9 ± 92.6 ng/ml \( n=38 \); (\( z=6.17 \), p<0.001.)

At the end of the study ie 90 days, 28% fatality was recorded with mean cortisol level of 388.4 ± 53.1 ng/ml, compared to the survivors 72% with mean cortisol level of 238.6 ± 82.2 ng/ml. The finding showed predictability of admission cortisol to 90\(^{th}\) day fatality (\( z=7.59 \), p < 0.01).

**Conclusion**

Single admission serum cortisol levels in the acute phase of ishaemic stroke did not relate to stroke severity at presentation, neither did it predict functional outcome at 7, 30, 90 days but could predict mortality at 30 and 90 days after the event.

Serum cortisol did not correlate to the 7\(^{th}\) day mortality among patients (p > 0.05), but was found to be a predictor of death at 30 and 90 days after strok