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Cingulate infarction: A neuropsychological and neuroimaging study

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ABSTRACT

Background and purpose: Ischemic lesions rarely affect the cingulate cortex (CC) in isolation, restricting human lesion/behavioural change correlational analysis. The aim of this study was to determine clinical, neuropsychological and neuroimaging features of isolated cingulate infarcts.

Methods: We studied, 3800 patients with first-ever ischemic stroke included in our Stroke Registry between 2012 and 2018. Among them we studied 7 patients with an acute isolated cingulate infarct confirmed by MRI.

Results: Among all patients, 7 patients (0.01%) showed ischemic lesions in the territory of cingulate cortex territory, allowing us to delineate 2 substantial distributions; (1) Anterior cingulate cortex (ACC) infarction (4 patients [57%]) was presented low vigilance level with apathy, mutism, deficits in executive function, attention, and disturbances of working, episodic and verbal memory; (2) Posterior cingulate cortex (PCC) infarction (3 patients [43%]) developed topographic disorientation, visual memory deficit and affective-emotional behavioural changes.

Conclusions: According rarely seen CC infarction events, we suggest that anterior and posterior CC are functionally separated and differences in clinical presentation are explained by considering; ACC plays a role in executive functions, episodic and working memory, set maintenance, and PCC is focused on spatial and verbal attention, and memory system. We considered that different patterns of cingulate infarcts are the result of variation in cingulate arterial supply or suggest a source of embolism.

1. Introduction

The cingulate cortex (CC) may play a role in neurological and psychiatric disorders [1,2]. Therefore, a detailed understanding of the effects of isolated ischemic lesions on the CC is likely to be important. The cingulate cortex (CC) may be subdivided into two portions on the basis of cytoarchitectonics [3]. Anterior (genual part) and midcingulate cortex (ACC) has extensive bidirectional connections with dorsolateral, orbitofrontal, primary and secondary motor, and insular regions of the cerebral cortexes [4–6]. The posterior cingulate cortex (PCC) forms part of the posteromedial cortex. Previous studies suggest that the PCC plays a more prominent role in regulating the focus of attention and arousal state [7–9].

Pericallosal artery of the anterior cerebral artey courses posterosuperiorly above corpus callosum, below cingulate gyrus. It travels within the callosal sulcus and gives off many small cortical branches that supply the rostrum of the cingulate gyrus. The callomarginal artery runs within the cingulate sulcus and supplies the portion of cingulate gyrus underlying the paracentral lobule. Posteriorly, the precuneus and dorsal PCC receive vascular supply from the precuneal artery, a branch of the caudal branches of pericallosal artery. Moreover, the rostral branches of the posterior cerebral artery supply the ventral PCC (Fig. 1) [10].

There is unlikely a small number of neuropsychological research about the cognitive consequences of focal ischemic lesions on the cingulate cortex. Strokes around this area produced neuropsychological and motor syndromes, which may result in part from damage of the ACC and PCC [11,12]. We describe here a series of patients with isolated anterior and posterior cingulate infarcts associated with specific clinical features and discuss underlying pathophysiological mechanisms.

2. Subjects and methods

Between January 2012 and October 2018, 3800 patients with first-ever ischemic stroke were admitted to the Neurology Department of the Ege University Hospital and prospectively entered in the Ege Stroke Registry [13]. A total of 88 (2.3%) patients with MRI-proven ischemic lesions restricted to the anterior cerebral artery were identified. Patients with old infarcts, old hemorrhagic lesions on imaging or simultaneous acute unilateral or multiple lesions outside the CC were excluded.

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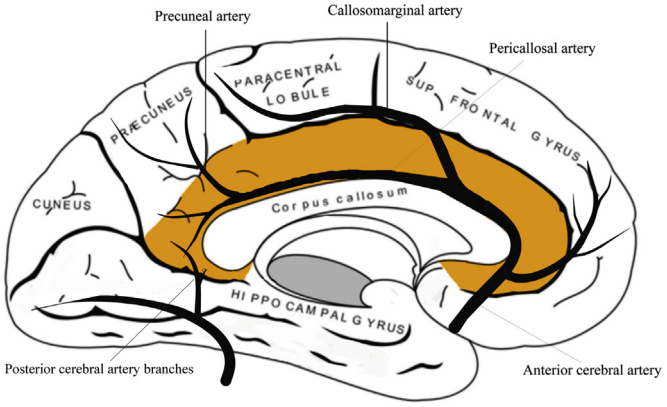

Fig. 1. Pericallosal artery of the anterior cerebral artey gives off many small cortical branches that supply the rostrum of the anterior cingulate cortex (ACC). The callomarginal artery supplies the portion of cingulate gyrus underlying the paracentral lobule. Posteriorly, dorsal posterior cingulate cortex (PCC) receive vascular supply from the precuneal artery and the rostral branches of the posterior cerebral artery supply the ventral PCC.

MRI was performed within 48 h of admission by 1.5 T or 3 T scanners (Siemens Sonata, Siemens Medical Solutions, Erlangen, Germany). Magnetic resonance or CT angiography, 2D-echocardiography, and 24-hour electrocardiography (Holter) monitoring were performed on selected patients, depending on the suspected etiology. The cause of stroke was assessed according to the criteria described previously [14]. The lesion territories were grouped according to their anatomic location within the cingulate cortex such as; anterior cingulate cortex territory and posterior cingulate cortex territory, as reported previously [4,9,15,16]. Our stroke cohort study was approved by the Medical Ethics Committees of the Ege University, and informed consent was obtained from all included patients.

3. Neuropsychological assessment

All patients had been examined clinically by at least 1 of us at the time of admission and had undergone a comprehensive neuropsychological examination by a battery of tests in the first week of stroke. The results of each test were analyzed using a normative cognitive data from a small sample of 100 healthy volunteers (males: 46%; mean ± SD age: 61 ± 12.1 years; educational level: 9.3 ± 4.1 mean years of education, primary school 31.3%, junior high 39.1%, high school 29.6%). Educational level of patients' cognitive data was 9.5.6 ± 2.9 years. Standardized neuropsychological tests were performed within the first week of stroke to assess cognitive functions including: orientation, attention, working memory (short term memory), episodic and semantic memory, verbal and visual memory, executive functions, goal-directed behaviour, reasoning, and spatial skills. For this purpose, we used the following tests to assess these cognitive and behavioural characteristics; Mini mental state examination (MMSE) [17] to measure the severity of cognitive impairment; Stroop test [18] to assess the ability to inhibit cognitive interference in the reaction time of a task, attention and executive function; Trail Making test [19] to assess visual attention and task switching; The Wisconsin Card Sorting test (WCST) [20] to test “set-shifting”, i.e. the ability to display flexibility, planning, reasoning, multitasking and goal-directed behaviour; Rey Auditory Verbal Learning Test (RAVLT) [21] to assess verbal learning, short delay recall, long delay recall, long delay cued recall; Wechsler Memory Scale-revised (WMS-R) [22] to determine episodic memory (information and orientation), semantic (logical) memory; Controlled Oral Word Association test (COWA) [23] to assess verbal fluency and executive functions; Benton Visual Retention test (BVRT) [24] to evalutae visuoperception and visuoconstructive abilities; Rivermead Behavioural Memory test (RBM) [25] to determine everyday memory performance ans social life assessment. A z-score (standard score) of each patients' cognitive test point was calculated and a z-score range out of ± 2 standard deviation was considered as falling to the far of the normal distribution and interpreted as impairment of related cognitive function (Table 2).

4. Results

Seven patients (8% of the anterior cerebral infarction in our registry) had isolated lesion inside the CC, and consisted of 4 men (57%) and 3 women (43%). Unilateral lesion was present in 5 patients (71%) and bilateral in 2 (29%) (Fig. 1). The mean age ± SD was 65.4 ± 4.2 years (range 59 to 71 years).

5. Anterior cingulate infarcts (patients 1–4)

Four patients (57% of the cingulate infarcts) had infarction in the anterior cingulate cortex (Fig. 2). Mean age ± SD was 66.5 ± 3.8 years (range 62 to 71). At stroke onset 2 patients had changes in vigilance level with apathy and loss of time orientation (Table 1). Transient akinesia, and astasia with instability and marked right-sided pulsion was observed in 1 patient each. Executive dysfunction, attentional deficits, disruption of multi-tasking, including learning task rules, remembering to implement new rules, and the ability to follow distinct plans in different parts of a task, affective-emotional behavioural changes were present in 3 patients (Table 2). Disturbances of working, episodic and verbal memory were found in 2 patients and oral word association in 3. Affective blunting and emotional slowdown with facial emotional expression loss was found in 3 patients, prominently in patient no.4. Three patients had perseveration, social withdrawal and excessive self-concern during follow-up. The most frequent stroke cause was large-artery disease in 2 patients (nos. 1 and 2).

6. Posterior cingulate infarcts (patients 5–7)

There was alteration in arousal and awareness state in one patient with bilateral PCC lesion (no.7). Topographic disorientation and visual memory deficit was observed in 2 patients. Early clinical assessment revealed some changes that might suggest behavioural and emotional changes, goal-directed behaviour loss, attentional deficits that were noted in unilateral and bilateral PCC territory involvement. Working, episodic memory and word association were also affected in these patients. Emotional responses to stimuli were slow and in inappropriate in 2 patients. Two patients (nos. 6 and 7) had cardioembolic source of embolism and one had artery-to-artery embolism (Table 1).

7. Discussion

This study provides neuroimaging and cognitive findings on the function of CC through a series of ischemic stroke patients. The most general conclusion from the anatomical-cognitive analysis is that anterior and posterior CC involved appear to be functionally different in part. At the general level the results here support recent evidence for the separation of the “ACC” and ``PCC” syndrome. Regarding to arousal and awareness state, mutism and abulia with attention deficit has been associated with ACC lesions in our patients which imply a deficit in focused or controlled attention, referring to a specific system that guides complex, starting voluntary actions [26].

Involvement of the AAC was associated with impairments of retrograde (plannig and reasoning) and anterograde (working and episodic memories) parts of cognitive functions. Involvement of the ACC gave rise to problems in memory and recognition which is supported by a large of recent evidence of its involvement in memory functions [27,28]. It is known that this region is also involved for retrieval of data from memory storage. Interestingly, studies support the fact that the

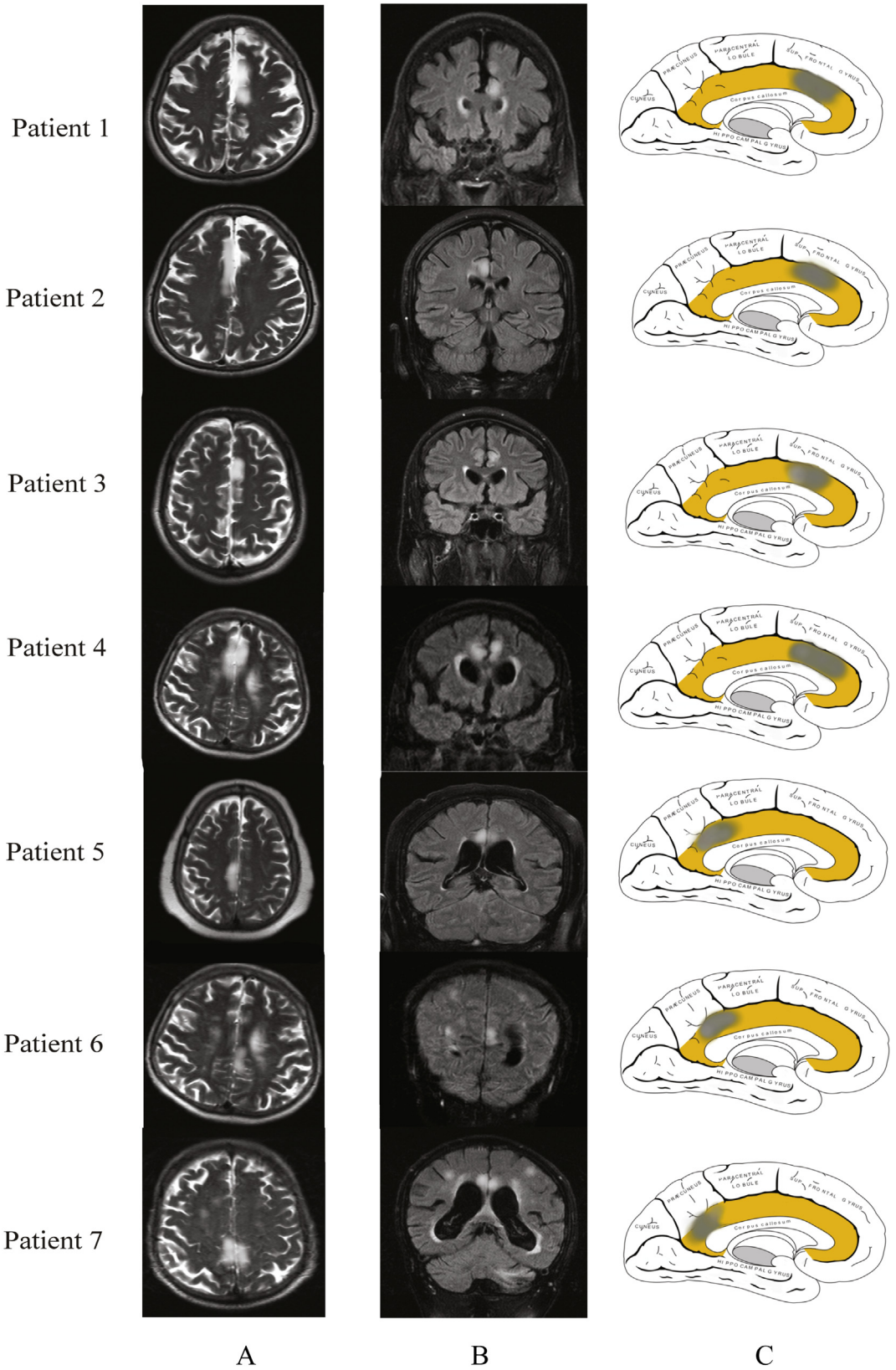

Fig. 2. Axial (A) and coronal MRI images (B) of the brain, showing high intensity in anterior and posterior parts of the cingulate cortex. (C) Schematic representation of ischemic lesions mapped onto sagittal sections showing the extent of the ACC (nos.1–4) and PCC (nos.5–7) damage.

ACC and PCC operate different functions although they are all anatomically interconnected [29,30]. Both site of the ACC lesions gave deficits in attention, executive fonctions and affective-emotional behaviour. The role for ACC dysfunction in emotional behavioural disorders is consistent with previous studies which they appeared as disinhibition, impulsivity, poor decision making, disturbance of the

Table 1
Clinical features of patients with cingulate infarct.

Characteristics	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5	Patient 6	Patient7
Age	65	71	62	68	59	64	69
Sex	M	M	F	M	F	M	F
Lesion site	Left	Right	Left	Bilateral	Right	Left	Bilateral
Cause of stroke	LAD	LAD	CE	UN	LAD	CE	CE
Altered consciousness	+	−	−	+	−	−	+
Confusion	−	−	−	+	−	−	+
Mutism	+	−	−	+	−	−	−
Abulia	−	+	−	−	−	−	+
Seizure	−	−	−	+	−	−	−
Hallucination	+	−	−	−	−	+	−
Transient akinesia	+	−	−	−	−	−	−
Spontaneous motor response	−	+	+	−	+	+	+
Astasia	−	−	−	+	−	−	+
Executive dysfunction	+	+	−	+	−	−	+
Impairment of multi-tasking	+	+	−	+	+	−	+
Topographic disorientation	−	−	−	−	+	−	+
Affective-emotional behavioural activities impairment	+	+	−	+	+	−	+
Attentional deficit	+	+	−	+	+	+	+
Goal-directed behaviour loss	+	+	−	+	+	−	+
Impaired autobiographical memory	−	−	−	+	−	−	+
Impaired working memory	+	−	−	+	+	−	+
Impaired social function	+	+	−	+	−	−	−

CE: cardioembolism, LAD: large-artery disease; UN: unknown; +, present; −, absent.

social self, or emotional dysregulation [31,32]. We observed that social behavioural change and loss of external interest was present only in patients with ACC lesions suggesting its role in the regulation of emotional behavior and executive functioning [16,33,34].

One important hypothesis is that the PCC has a central role in supporting memory functions such as observed in our patients with PCC lesion. Bilateral infarction of ACC and PCC is associated with difficulty of retrieval of autobiographical memories, set maintenance and goal-directed planning for the future. More specifically, it has been proposed that at least one of the roles of the posterior cingulate is in the transfer and accentuation of memory-related information passing between the hippocampal system and neocortical association areas. It has been

Table 2
Raw and z-scores of neuropsychological tests of patients with cingulate infarct.

Neuropsychological tests (Mean ± 1SD of normal healthy subjects)	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5	Patient 6	Patient 7
Education level (years)	6	9	8	15	8	9	7
Mini-mentale state Examination (27 ± 1.6)	23(−2.50)	24(−1.88)	22(−3.13)	23(−2.50[a])	25(−1.25)	24(−1.88)	23(−2.50[a])
Stroop test	49(3.13[a])	46(2.75[a])	39(1.88)	65(5.13[a])	34(1.25)	33(1.13)	38(1.75)r
Name color print of non-color words (24 ± 8 s)	79(3.00[a])	68(2.21[a])	55(1.29)	76(2.79[a])	62(1.79)	52(1.07)	66(2.07)
Name color print of color words (37 ± 14 s)							
Trail making test	78(3.33[a])	96(4.83[a])	48(0.83)	65(2.25[a])	52(1.17)	54(1.33)	60(1.83)
Trails A (38 ± 12 s)	210(2.16[a])	280(3.44[a])	120(0.53)	220(2.35[a])	160(1.25)	185(1.71)	195(1.89)
Trails B (91 ± 55 s)							
Wisconsin Card Sorting test	0,5(−1,80)	1,0(−1,55)	3(−0,55)	0,5(−0,55)	2,5(0,80)	3,0(−0,55)	2,5(−0,80)
Categories achieved (4.1 ± 2)	34(−4.00)	48(−2.25[a])	45(−2.63[a])	50(−2.00)	55(−1.38)	54(−1.50)	48(−2.25)
Correct responses (66 ± 8)	88(2.30)	75(1.65)	68(1.30)	90(2.40[a])	65(1.15)	78(1.80)	70(1.40)
Errors (42 ± 20)	55(2.69)	49(2.23[a])	48(2.15[a])	63(3.31[a])	36(1.23)	34(1.08)	42(1.69)
Perseverative responses (20 ± 13)	2.4(2.50)	2.2(2.17[a])	1.9(1.67)	2.6(2.83[a])	1.8(1.50)	1.4(1.00)	2.3(2.33[a])
Failure to maintain (0.9 ± 0.6)							
Working memory (short term memory)	2.0(−3.58[a])	4.5(−1.50)	4.0(−1.92)	3.5(−2.33[a])	4.0(−1.92)	4.5(−1.50)	3.0(−2.75[a])
Trial 1 List A[b] (6.3 ± 1.2)							
Episodic memory Information/orientation[c] (13.9 ± 1)	10.0(−3.90[a])	11.0(−2.90[a])	12.0(−1.90)	10.0(−2.90[a])	12.0(−1.90)	12.0(−1.90)	11.0(−2.90[a])
Verbal memory	5.0(−2.48[a])	7.0(−1.52)	8.0(−1.05)	4.0(−2.95[a])	7.0(−1.52)	8.0(−1.05)	5.0(−2.48[a])
Delayed recall[b] (10.2 ± 2.1)	5.0(−2.77[a])	6.0(−2.32)	8.0(−1.41)	5.0(−2.77[a])	9.0(−0.95)	7.0(−1.86)	5.0(−2.77[a])
Recognition[b] (11.1 ± 2.2)							
Controlled oral word association (COWA)	7.0(−2.50[a])	8.0(−2.25[a])	13.0(−1.00)	8.0(−2.25[a])	14.0(−0.75)	11.0(−1.50)	11(−1.75)
Mean numbers of animal named (17 ± 4)							
Visual memory	13.0(−1.64)	14.0(−1.29)	13.0(−1.64)	11.0(−2.36[a])	14.0(−1.29)	15(−0.93)	10(−2.71[a])
Visual memory span[d] (17.6 ± 2.8)							
Rivermead behavioural	5.0(−3.33[a])	7.0(−2.00)	6.0(−2.67[a])	5.0(−3.33[a])	8.0(−1.3)	8.0(−1.33)	6.0(−2.67)
Memory test (10 ± 1.5)							

Values are as raw points and (z-score).
Values in parentheses are mean ± 1 SD of normative data after adjustment for age and education.
[a] Indicates z-scores out of standard 2.0 z-score (0.01 percentile).
[b] Rey-Auditory Verbal Learning Test (RAVLT).
[c] Wechsler Memory Scale-Revised (WMS-R).
[d] Benton Visual Retention test (BVRT).

suggested that the ACC and PCC together play some part in the memory requirements necessary for working and episodic memory [35,36]. Moreover, our patients with attentional and goal directed behavioural deficits suggest that the PCC plays a more direct role in regulating the focus of attention, perhaps controlling the balance between internally and externally focused thought [37–40]. It has been proposed that the PCC is involved in 'tuning' the focus of attention [8]. Another findings of PCC lesion is the affective-emotional disturbances which are consistent with the suggestion that this region may mediate interactions of emotional and memory-related processes such as the enhancement of memory for emotional information.

Astasia and dysequilibrium has been observed in our patients with large bilateral CC lesion probably by damage of connection between cingulate motor area and vestibulocerebellar system through the thalamic nuclei [41]. We observed transient akinesia and mutism in one patient with ACC lesion as such as previously reported in one patient with slight encroachment of lesion on adjacent cortical regions [42].

As summary, PCC and ACC contributes to behaviour and memory retrospectively and prospectively by linking the processing of information derived from internal and external world. It monitors the affective-emotional of stimuli in conjunction with orbitofrontal cortex, exerts control over arousal and awareness with prefrontal regions, and modulates planning, executive functions in dorsolateral frontal cortex. Ischemic lesions of CC are the result of difference in cingulate arterial supply or suggest a source of embolism.

Authors contribution

Principal author Emre Kumral M.D.
Study concept or design Emre Kumral M.D., Fatma Ece Bayam M.D.
Acquisition of data Can Emre Erdoğan M.D., Hasan Arslan PhD.
Analysis or interpretation of data Fatma Ece Bayam M.D.
Statistical analysis Emre Kumral M.D. PhD.
Study supervision or coordination Emre Kumral M.D., Can Emre Erdoğan M.D.

Conflict of interest

We have no actual or potential conflicts of interest for all authors involved in this paper.

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Ethical committee approval

Ege University Medical Ethical Committee was approved this study following the principles outlined in the Helsinki Declaration before starting the study (2010).

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