onset GBS patients, two of whom were positive for *C jejuni* serology and antiganglioside IgG. Another patient who developed acute pure motor neuropathy following *C jejuni* enteritis was reported to have localised weakness in his hands and anti-GM1 IgG.[4] Although that patient had preserved tendon reflexes in the four limbs, a serial electrophysiological study confirmed the diagnosis of an axonal variant of GBS, indicating that anti-GM1 IgG and *C jejuni* infection are related to hand-predominant weakness in GBS. It also is noteworthy that the six patients who had hand onset GBS had an initial diagnosis of cervical spondylosis (n = 4), lacunae infarction (n = 1), or brachial plexus neuritis (n = 1) on hospital admission. Frequent hand function problems have been reported even in mildly affected GBS patients who could walk unaided at nadir.[5] Early treatment has been suggested in such cases. Recognition of the clinical characteristics of hand onset GBS may lead to a good prognosis because individuals can be given specific treatment as early as possible.

Acknowledgements

This research was supported in part by a grant-in-aid from the Uehara Memorial Foundation, a grant for scientific research (B) (KAKENHI 14370210 to NY) from the Ministry of Education, Culture, Sports, Science and Technology of Japan, and a research grant for neuroimmunological diseases from the Ministry of Health, Labour and Welfare of Japan.

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Lack of association between interleukin-1β polymorphism (–511) and ischaemic stroke

A growing body of evidence suggests an important role for interleukin 1 (IL-1) in the pathogenesis of brain damage following cerebral ischaemia. Central administration of IL-1 exacerbates brain damage, and overexpression of the IL-1 receptor antagonist (IL-1Ra) or blockade of IL-1 converting enzyme activity reduces infarct size dramatically (reviewed by Touzani *et al*[1]). Clinical studies suggest there is intrathecal IL-1 production early during stroke.[2]

Table 1 Distribution of risk factors and IL-1β genotypes in patients and controls

	Stroke patients (n = 183)	Controls (n = 180)	p Value
Age (years) (mean (SD))	65.2 (14.7)	64.8 (14.8)	0.80
Male	81 (44.3)	69 (38.3)	0.25
Hypertension	139 (76)	94 (52.2)	<0.01
Diabetes mellitus	33 (18)	32(17.8)	0.59
History of myocardial infarction	15 (8.21)	9 (5)	0.22
Current smoker	47 (25.7)	21 (11.7)	<0.01
IL-1β genotypes			0.37
CC	94 (51.4)	87 (48.3)	
CT	69 (37.7)	79 (43.9)	
TT	20 (10.9)	14 (7.8)	
T allele frequency (%)	29.8	29.7	0.97

Values are n (%) unless stated otherwise.

A single nucleotide polymorphism in the promoter region of IL-1β at position −511 resulting in C–T transition influences the protein production, and IL-1β-511T carriers are reported to be higher producers of IL-1β than IL-1β-511C carriers.[3]

In the study described here we investigated whether IL-1β polymorphism (−511) can be involved in the genetic susceptibility to ischaemic stroke. We studied 183 consecutive patients with ischaemic stroke presenting to our stroke unit and 180 control subjects without a history of stroke. Control subjects were recruited from spouses of the patients, from individuals admitted to the university hospital for any reason other than neurological diseases, and from persons randomly selected from the community of our town. All patients, controls, and their parents had to be of white extraction.

Cerebral infarction was defined as a focal neurological deficit of sudden onset that persisted beyond 24 hours, documented by brain computed tomography or magnetic resonance imaging, indicating the presence of infarction or the absence of haemorrhage.

Stroke aetiology was defined according to the TOAST criteria[4]: 66 patients had large vessel disease, 50 had small vessel disease, 49 had cardioembolic stroke, and 18 had stroke of undetermined aetiology.

Arterial hypertension was diagnosed when its presence was documented in the medical records or if two or more readings of blood pressure were ⩾160 mm Hg (systolic) or ⩾95 mm Hg (diastolic) before the onset of stroke or three months later. Diabetes mellitus was diagnosed if the patient gave a history of diabetes that was confirmed by their medical records or was taking insulin or an oral hypoglycaemic agent. A patient was defined as a current smoker if there was a history of cigarette smoking during the last five years.

Genomic DNA was extracted from peripheral blood using a commercially available kit from Qiagen. Interleukin-1β polymorphism (−511) was detected using the polymerase chain reaction and restriction enzyme digestion as described elsewhere.[5]

All subjects gave informed consent and the local ethics committee approved the study protocol.

The sample size was calculated with a power of 80% at the 0.05 significance level. The sample size would allow detection of a relative risk by allele of 2.2. Differences between groups were examined using the χ^2 test or the unpaired Student *t* test as appropriate. Probability (p) values of less than 0.05 were considered statistically significant.

The characteristics of study subjects and distribution of IL-1 genotype are shown in table 1.

There was no significant difference between stroke patients and controls in age and sex.

Allele frequency in both controls and patients was in Hardy-Weinberg equilibrium (p = 0.32 for controls, p = 0.40 for stroke patients).

There was no significant difference between stroke patients and controls in IL-1β genotype distribution. There was also no relation between IL-1β polymorphism and any particular stroke subtype: large vessel disease, for TT, 7/66 (10.6%); small vessel disease, 6/50 (12.0%); cardioembolic stroke, 7/49 (14.3%) (p = 0.24, χ^2 test).

Comment

We failed to find a relation between IL-1β polymorphism (−511) and ischaemic stroke in this Polish population. Recently Seripa *et al* investigated the same polymorphism in an Italian population of 110 stroke survivors and 101 healthy controls[6] and also did not find any significant association between IL-1β polymorphism (−511) and stroke, although they showed a significantly higher frequency of the IL-1Ra 1/1 genotype in stroke survivors than in controls.

Several issues should be taken in account in interpreting the results of our study.

First, cytokines do not work alone, but in a network. Therefore a genetic predisposition to produce anti-inflammatory cytokines (for example, IL-10 or IL-1Ra) could interfere with the biological effects of IL-1.

Second, we did not examine another IL-1β polymorphism in exon 5 at position +3953 which could determine IL-1β synthesis.

Third, we cannot exclude the possibility that IL-1β polymorphism (−511) is associated with one particular stroke subtype; however, in our study we found no relation between IL-1 polymorphism and large vessel disease, small vessel disease, or cardioembolic stroke. From our point of view, there is currently a lack of strong evidence indicating a functional association between IL-1 and any particular stroke subtype.

Fourth, IL-1 may be linked to inflammatory mechanisms of atherogenesis. Hypertension and smoking play an important role in the pathogenesis of atherosclerosis. In our study the incidence of hypertension and smoking was higher in stroke patients than in controls, and the frequency of the TT allele was higher in smokers than in non-smokers (15.4% *v* 7.9%, p = 0.18) and in subjects with hypertension than in those without (10.3% *v* 7.7%, p = 0.48). Atherosclerosis is related to

large vessel disease; however, in our study the IL-1 genotype distribution did not differ significantly between patients with large vessel disease and controls.

Fifth, further studies are needed that are focused on achieving sufficient power to detect a possible smaller allelic relative risk (<2).

In conclusion, our results do not support the hypothesis that IL-1β polymorphism (−511) is associated with ischaemic stroke.

Acknowledgement

This study was supported by a grant from Medical Committee of Collegium Medicum, Jagiellonian University.

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