Summary of patients with subarachnoid haemorrhage: clinical profiles, and amino acid neurotransmitter (AANT) concentrations in CSF

Case	Age /sex	H and K grade	GCS	Day of Operation	Day of Deterioration	Day of sampling	Vasospasm	GOS	AANT concentrations (μM) ASP	GLU	TAU	ALA
1	53/M	2	15	1		4	+	GR	5·26	9·28	7·68	60·3
2	56/M	2	14	1		3	+	GR	0·22	2·57	5·74	64·43
3	62/F	3	11	1		8	+	GR	2·13	9·32	15·6	129·1
4	52/M	3	13	0		3	+ +	MD	1·02	0·88	4·65	155·4
5	64/M	3	14	1	3	5	+ +	SD	2·02	3·11	8·94	132·4
6	48/M	3	13	1		6	+	GR	0·54	3·76	5·98	60·94
7	41/F	4	7	1		5	+	GR	1·48	0·77	4·35	110·0
8	79/F	4	6	1	5	5	+ + +	D	0·24	9·45	5·33	40·3
9	55/M	4	5	0	3	2	+ + +	D	2·00	3·24	12·9	145·4
10	65/F	4	11	1	4	5	+ + +	SD	4·29	7·85	14·8	151·0
							Mean (SD)		1·9(1·7)*	5·0(3·6)*	8·6(4·3)	105·3(44)
							Control (n = 16)		0·6(0·5)	1·6(0·7)	5·8(1·8)	31·9(6·2)

*$p < 0.05$ v controls.
H and K = Hunt and Kosnik grade; GOS = Glasgow outcome scale; GCS = Glasgow coma scale; ASP = aspartate; GLU = glutamate; TAU = taurine; ALA = alanine; Day = postsubarachnoid haemorrhage day; + = mild (100–149 cm/s); ++ = moderate (150–199 cm/s); +++ = severe (≥200 cm/s); GR = good recovery; MD = moderately disabled; SD = severely disabled; D = dead.

outcome requires study of a larger population of patients but our preliminary results provide a rational basis for such studies and for trials of excitotoxic amino acid antagonists.

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Incidence of internal carotid artery dissection in the community of Dijon

Dissection of the cervical internal carotid artery is one of the major causes of ischaemic stroke in patients under 50 years old.[1] Reliable epidemiological data are not numerous. Only one defined population study on internal carotid artery dissection has been published.[2]

We examined the occurrence of spontaneous internal carotid artery dissection in the population of the city of Dijon from 1985 to 1993, by means of the stroke Registry of Dijon[3] which records all first strokes, and also asymptomatic stenosis, occlusion, and dissection of internal carotid artery occurring in the population of the city of Dijon. The diagnosis was ascertained by an ultrasonic Doppler examination of the cervical carotid arteries, and a carotid angiography. Patients with a history of cervical trauma were not included. For the calculation of incidence, the whole population of Dijon was considered to be at risk, with 135 000 inhabitants in 1985 and 150 000 inhabitants in 1992, and was used as the denominator for the study.

From 1985 to 1993, 36 internal carotid artery dissections were diagnosed. The average annual incidence for all age groups was 2·9 per 100 000 population (95% confidence interval 1·9–3·9). There were no statistically significant annual variations (three cases in 1985, four in 1986, three in 1987, five in 1988, four in 1989, five in 1990, three in 1991, four in 1992, five in 1993). The mean age was 39·9 (7·5) years for the 21 women, 43·7 (5·7) years for the 15 men. All had headache or neck pain; 19 patients presented with cerebral ischaemic symptoms, four with retinal ischaemic symptoms; one patient had had a subarachnoid haemorrhage. Oculosympathetic palsy was noted in 12 patients, and 12th cranial nerve palsy in one. Isolated headache was the single symptom in three cases (8·3%). The diagnosis of dissection was suspected with ultrasound in 34 cases (with demonstration of a double lumen in eight cases), and proved with angiography in all cases, with MRI in only the last four patients. No necropsy cases of spontaneous cervical internal carotid artery dissection were found during this period. No bilateral cervical internal carotid artery dissections, or associated vertebral artery dissection were found. Arterial hypertension was seen in 19 cases. Dysplastic cervical or renal arteries were found in 15 cases. Association with tobacco and pill consumption was present in 15 out of 21 female cases. Evolution was marked by slight neurological sequelae in four cases. Headache resolved in all patients. Recurrent arterial dissections were not found at follow up ranging from three months to nine years.

These 36 cases represent 2% of 1784 patients with stroke collected in this population of Dijon and 10·1% of the 356 stroke patients under 50 years old. This confirms that internal carotid artery dissection is a major cause of cerebral infarction in those under 50 years old.[1]

The average annual incidence of spontaneous internal carotid artery dissection for all age groups was 2·9 per 100 000 inhabitants, similar to that in the city of Rochester.[2] This compares with 2·2 per 100 000 for aneurysmal subarachnoid haemorrhage in Dijon.[3]

Before ultrasound was introduced many cases remained undiagnosed. Biller *et al*[4] reported a spontaneous cervical internal carotid artery dissection in 0·13% of 4531 patients with acute stroke and Bogousslavsky *et al*[5] reported dissection in 2·5% of 1200 patients with acute stroke.

A community based study in Rochester, Minnesota, 1987–92 gave a similar incidence of 2·6 per 100 000.[2]

Headache, ischaemic cerebrovascular disease, and oculosympathetic palsy were the most common manifestations of spontaneous internal carotid artery dissection. Some internal carotid artery dissections do not present with ischaemic stroke (10% in the study of Shievinck *et al*,[2] 8·3% in the present study).

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