Cervicocephalic arterial dissections related to skiing.

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Capillaroscopic Findings in Lacunar Infarction and Control Groups

Finding	LI, %	CI, %	Control, %	χ^2
Tortuous loops	25	20	25	NS
Loss of capillaries	0	0	0	NS
Enlarged loops	0	0	0	NS
SVP visibility	90	15	10	$P<.001$

LI indicates lacunar infarction patients; CI, cerebral infarction patients; and SVP, subpapillar venous plexus.

the data reported in the literature are generally contrasting and not conclusive.[2] The aim of our study was to investigate the presence of capillaroscopic alterations in patients with lacunar infarctions (LIs). The LIs are small infarcts that occur in the subcortical regions of the brain (ie, deep white matter, basal ganglia, internal capsule, thalamus, and brain stem), with consequent variable clinical syndromes.[4] Pathological lesions underlying the lacunes are small-vessel lipohyalinosis or microatheroma, while arterial hypertension and diabetes mellitus are the most frequent risk factors.[4,5] Twenty consecutive patients followed up at the Neurology Institute of the University of Pisa (14 men and 6 women, mean±SD age 68±7 [range, 55 to 78] years) with LI and without other neuropsychiatric disorders were studied. In addition, two other age- and sex-matched groups were included in the study as controls: 20 patients with cortical or subcortical cerebral infarction and 20 healthy control subjects. Patients with LI had motor, sensory, or sensorimotor stroke; in all cases, diagnosis of LI was based on computed tomographic scan results. A clinical assessment was carried out in all LI and control groups and included epidemiological, medical, and neurological history; physical examination; and laboratory results. In addition, carotid flow was evaluated by Doppler analysis. Various risk factors (arterial hypertension, diabetes mellitus, carotid stenosis, coronary insufficiency, smoking, altered lipid metabolism) were recorded with variable but not significantly different frequency in both LI patients and control groups. Capillaroscopic examinations were performed by the same investigator (C.F.), blinded to the protocol, using a Leitz stereomicroscope at 12× magnification, as previously described.[1-3] All digits of both hands, excluding the thumbs, were examined. The Table summarizes the capillaroscopic alterations found in LI patients and control groups.

Tortuous capillary loops and other minor morphological abnormalities were recorded with comparable frequency in both LI patients and control subjects. On the contrary, in the large majority of LI patients the subpapillar venous plexus was clearly evident in at least half of the digits examined; the prevalence of this finding resulted in statistical significance when compared with cerebral infarction patients and healthy control subjects ($P<.001$).

In our present series of patients with LI, the almost-constant evidence of subpapillar venous plexus seems to suggest the presence of a systemic microangiopathy in this disease. Because the incidence of various risk factors is comparable in both LI and cerebral infarction series, it is possible to hypothesize a different pathogenetic mechanism of stroke in these two patient populations. Further studies are certainly necessary to confirm and explain these preliminary data by means of morphofunctional and laboratory investigations.

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References

1. Maricq HR. Widefield capillary microscopy: technique and rating scale for abnormalities seen in scleroderma and related disorders. *Arthritis Rheum.* 1981;24:1159-1165.
2. Andrade ECA, Gabriel A, Assad RL, Ferrari AJL, Atra E. Panoramic nailfold capillaroscopy: a new reading method and normal range. *Semin Arthritis Rheum.* 1990;20:21-31.
3. Ferri C, Bernini L, Cecchetti R, Neri R, Latorraca A, Marotta G, Pasero G, Bombardieri S. Cutaneous and serological subsets of systemic sclerosis. *J Rheumatol.* 1991;18:1826-1832.
4. Fisher CM. Lacunar strokes and infarcts: a review. *Neurology.* 1982; 32:871-876.
5. Horowitz DR, Tuhrim S, Weinberger JM, Rudolph SH. Mechanisms in lacunar infarction. *Stroke.* 1992;23:325-327.

Cervicocephalic Arterial Dissections Related to Skiing

Cervicocephalic arterial dissections are responsible for 1% to 2.5% of the strokes in patients admitted to a hospital and up to 20% of cerebral infarction in the young.[1] Traumatic carotid artery dissections have often been reported in relation to motor vehicle accidents, fist fights, strangulation, violent coughing, head banging, heavy load carrying, and sports activities (eg, trampolining, basketball, polo, football, and water skiing).[1,2] Vertebral artery dissections have been related to cervical manipulations, yoga, tennis, neck turning, ceiling painting, archery practice, and swimming. However, traumatic dissections due to snow skiing have rarely been reported.[3-5]

From 1986 to 1993, 12 patients with cervical artery dissections related to skiing were admitted to the stroke unit (see the Table). They accounted for about 15% of the cervical artery dissections and 60% of the traumatic cervical artery dissections admitted to the stroke unit. Ten patients had an internal carotid and 2 a vertebral artery dissection. The diagnosis was established in all patients with four-vessel cervicocerebral arteriography. Magnetic resonance imaging was obtained in 7 patients with carotid artery dissection and showed (with T_1-weighted images in the axial plane) in 6 of them the typical crescent hyperintense signal of the subacute hematoma expanding the arterial wall and the narrowed residual lumen. All patients had been downhill skiing but one, who had been cross-country skiing. The clinical data are reported in the Table.

The high rate of men (11 of 12) differs from the sex ratio close to 1 usually reported in spontaneous cervical artery dissections. It may be related in part to the difference in the sex ratio of skiers, and more probably to the usual sex-related difference in risks taken when skiing. Our patients had a mean age of 47.1 years, close to the mean ages reported in two series of spontaneous cervical artery dissections.[6,7] However, despite the risks taken by young people when skiing, we did not observe patients under the age of 25. This may suggest that traumatic cervical artery dissections when skiing may occur in patients in the third and fourth decades of life with a predisposing age-related weakness of the arterial wall.

In the debate of the role of trauma in cervical artery dissections due to skiing, the relation was evident in 6 patients who had severe trauma. In 5 patients the trauma was moderate to minor, and the role of a preexisting weakness of the arterial wall may be suggested. In one patient, no trauma was reported, and the spontaneous occurrence of carotid artery dissection may be considered.[2] Cervical artery dissections have been related to the following predisposing conditions: fibromuscular dysplasia, elastic tissue disease, and sometimes arterial risk factors and migraine. We did not find support of the hypothesis of an underlying fibromuscular

Clinical Data and Severity of Trauma in 12 Patients With Cervical Artery Dissection Related to Skiing

Patient	Sex	Age, y	Severity of Fall	Initial Loss of Consciousness	Delay of Stroke Onset	Clinical State at Admission	Risk Factors
1	F	26	Severe	Yes	1 hour	R hemiplegia, aphasia	None
2	M	40	Severe	Yes	2 hours	L hemiparesis	Smoking
3	M	41	Moderate	No	3 days	L sensorimotor deficit	None
4	M	49	Moderate	No	3 days	L hemiplegia, Horner's syndrome	None
5	M	65	Minor	No	1 day	R hemiplegia, aphasia	None
6	M	56	Severe	No	5 days	Transient L hemianopia	Hypercholesterolemia
7	M	45	No fall	No	. . .	R hemiparesis, aphasia	Smoking
8	M	58	Severe	Yes	3 days	L sensorimotor deficit	None
9	M	55	Neck hyperextension	No	15 minutes	R hemiplegia, aphasia	None
10	M	47	Severe	No	15 minutes	R transient monocular blindness	Smoking
11	M	43	Severe	No	12 days	L sensorimotor deficit, L hemianopia	None
12	M	49	Minor	No	1 to 2 hours	Vertigo, neck pain, vomiting R-hand paresthesia	Hypertension

disease in our patients. Renal angiography was normal in the four patients in whom it was obtained. In 11 patients, only one cervical artery was affected at arteriography. Because the majority of the patients were living far from our center, arteriographic control could be obtained 6 months later in only 4 patients. Three had normal arteriography, and 1 had a pseudoaneurysm. None of our patients had clinical signs of elastic tissue disease. Seven patients had no arterial risk factor. For all these reasons (absence of evidence of fibromuscular dysplasia, elastic tissue disease, and arterial risk factor), the causal relationship between skiing and arterial dissection is suggested in our patients. To explain the mechanism of traumatic carotid dissection, it has been suggested that stretch, traction, and rotation forces are applied to the internal carotid artery as it crosses the transverse processes of the second and third cervical vertebrae. The forces can produce intimal fractures and induce eruption of blood into the arterial wall, leading to dissection. Another presumed mechanism may be the direct compression of the artery between the angle of the mandible and upper cervical vertebrae.[8,9] In vertebral artery dissection, rotation of the head can stretch and compress the vessel at its most mobile cervical part, leading to an injury in the V3 segment, where the wall is less rich in elastic fibers.[10] Stroke is bound to two mechanisms: it may be related to thrombus development at the dissection site that can embolize in the distal arterial branches or to a hemodynamic mechanism related to the occlusion of the arterial lumen.

The delay between the initial trauma and stroke was similar to the delays reported in the traumatic dissections related to other causes.

Two patients (6 and 12) recovered totally. The outcome was good, with recovery of activities of daily life, for seven patients (1, 2, 3, 7, 8, 10, and 11). Patients 4, 5, and 9 had a loss of autonomy in activities of daily living. In our population, the prognosis was not related to the severity of the fall. However, our population may be biased. Very severely comatose patients may die before admission or may be admitted to intensive care units and not to the stroke unit. On the other hand, patients presenting only with local signs or symptoms (headache, Horner's syndrome, or lower cranial nerve palsies) may not be admitted to the stroke unit or may remain undiagnosed.

Minor-to-severe trauma has been reported in internal carotid and vertebral artery dissections. Our patients had minor-to-severe falls followed by a stroke due to cervicocephalic artery dissection related to skiing. Moreover, the diagnosis of traumatic dissection related to skiing can open up a legal problem because of the imputability of the stroke to the skiing. This question is of legal importance because of the economic consequences of an established relationship between the trauma and the stroke.

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References

1. Hart RD, Easton JD. Dissections and trauma of cerebral arteries. *Neurol Clin.* 1983;1:155-182.
2. D'Anglejan Chatillon J, Ribeiro V, Mas JL, Bousser MG, Laplane D. Dissections de l'artère carotide interne: 62 observations. *Presse Med.* 1990;19:661-667.
3. Mokri B, Piepgras DG, Houser OW. Traumatic dissections of the extracranial internal carotid artery. *J Neurosurg.* 1988;68:189-197.
4. Bonneton G, Guidicelli H, Benabid AL, Banon F, Crouzet G, Larribau E, Riondel JP, Gautier R. Les traumatismes de l'artère carotide interne au cours des accidents de ski. *Chirurgie.* 1974;100:255-260.
5. De Rougemont J, Crouzet G, Barge M, Benabid AL, Perret J, Chatelain R. Une nouvelle observation de lésion traumatique de la carotide interne. *J Med Lyon.* 1969;50:1303-1309.
6. Fisher MC, Ojemann RG, Roberson GH. Spontaneous dissection of cervico-cerebral arteries. *Can J Neurol Sci.* 1978;5:9-19.
7. Sellier N, Chiras J, Benhamou M, Bories J. Dissections spontanées de la carotide interne. *J Neuroradiol.* 1983;10:243-259.
8. Zelenok GB, Kazmers A, Whitehouse WM, Graham LM, Erlandson EE, Cronenwett JL, Lindenauer SM, Stanley JC. Extracranial internal carotid artery dissections. *Arch Surg.* 1982;117:425-432.
9. Stringer WL, Kelly DL. Traumatic dissection of the extracranial internal carotid artery. *Neurosurgery.* 1980;6:123-130.
10. Wilkinson IMS. The vertebral artery extracranial and intracranial structure. *Arch Neurol.* 1972;27:392-396.