After the revascularization procedure on the carotid artery all patients must follow the principles of secondary prevention of ischemic cerebrovascular events of atherotrombotic origin.

REFERENCES

- 1. Bonita R, Mendis S, Truelsen T, Bogousslavsky J, Toole J, Yatsu F. The Global Stroke Initiative. Lancet 2004; 3: 391-3.
- 2. Kings College London, European Register of Stroke (EROS). ttp://:www.ktl.fi/eros. 2005.
- 3. Khaw KT. Epidemiology of stroke. J Neurol Neurosurg Psychiatry 1996; 61: 333-8.
- 4. North American Symptomatic Carotid Endarterectomy Trial Collaborators. Beneficial effect of carotid endarterectomy in symptomatic patients with high grade carotid stenosis. N Engl J Med 1991; 325: 445-53.
- 5. Wolf-Maier K et al. Hypertension prevalence and blood pressure levels in 6 European counties, Canada and United States. JAMA 2003; 289: 2363-9.
- 6. Šelb Šemrl J. Epidemiološki podatki o možganski kapi v Sloveniji. In: Žvan B, Bobnar Najžer E eds. Spoznajmo in preprečimo možgansko kap. Društvo za zdravje srca in ožilja Slovenije: Ljubljana, 2006: 38-43.
- 7. Kearney PM et al. Global burden of hypertension: analyses of worldwide data. Lancet 2005; 365: 217-223.
- 8. Sacco RL. Extracranial carotid stenosis. N Engl J Med 2001; 345: 1113-18.
- 9. Inzitari D, Eliasziw M, Gates P, Sharpe BL, Chan RK, Meldrum HE, Barnett HJ. The causes and risk of stroke in patients with asymptomatic internal-carotid artery stenosis. North american Symptomatic Carotid Endarterectomy Trial Collaborators. N Engl J Med 2000; 342: 1693-700.
- Geroulakos G, Hobson RW, Nicolaides A. Ultrasonographic carotid plaque morphology in predicting stroke risk. Br J Surg 1996; 83: 582-7
- 11. Yuan C, Mitsumori LM, Beach KW, Maravilla KR. Carotid atherosclerotic plaque: noninvasive MR characterization and identification of vulnerable lesions. Radiology 2001; 221: 285-99.
- 12. Magyar MT, Nam EM, Csiba L, Ritter MA, Ringelstein EB, Droste DW. Carotid artery auscultation anachronism or useful screening procedure? Neurol Res 2002; 24: 705-8.
- 13. Timsit SG, Sacco RL, Mohr JP, Foulkes MA, Tatemichi TK, Wolf PA, Price TR, Hier DB. Early clinical differentiation of cerebral infarction from severe atherosclerotic stenosis and cardioembolism. Stroke 1992; 23: 486-91.
- 14. Brainin M, Olsen TS, Chmorro A, Diener HC, Ferro J, Hennerici MG, Laghorne P, Sivenius J. Organization of stoke care: education, referral, emergency management and imaging, storke units and rehabilitation. Cerebrovas Dis 2004; 17: Suppl 2: 1-14.
- 15. Filis KA, Arko FR, Johnson BL, Pipinos II, Harris EJ; Olcott C IV, Zarnis CK. Duplex ultrasound criteria for defining the secverity of carotid stenosis. Ann Vasc Surg 2002; 16: 413-21.
- 16. Demarin V. Nevrosonologija v klinični nevrologiji. In: Tetičkovič E, Žvan B eds. Sodobni pogledi na možgasnkožilne bolezni. Maribor: Obzorja, 2003: 29-40.
- 17. Žvan B. Sodobna ultrazvočna klasifikacija aterosklerotičnih leh v karotidnih arterijah. In: Blinc A, Ciglenečki I, Fras Z, Kozak M, Poredoš P, Šabovič M eds. Ateroskleroza Zbornik predavanj. Združenje za žilne bolezni SZD. Med Razgl 1998: Suppl 3: 95-102.
- European Carotid Plaque Study Group. Carotid artery plaque composition. Relationship to clinical presentation and ultrasound B-mode imaging. Eur J Vasc Endovasc Surg 1995; 10: 23-30.
- Cinat M, Lane CT, Pham H, Lee A, Wilson SE, Helical GI. CT angiography in the preoperative evaluation of carotid artery stenosis. J Vasc Surg 1998; 28: 290-300.
- Žvan B, Zaletel M, Miloševič Z, Videčnik V, Tetičkovič E, Flis V. Smernice za odkrivanje in zdravljenje karotidne bolezni. Zdrav Vestn 2004: 11: 833-8.
- 21. Žvan B. Preventiva ishemične možganske kapi. In. Švigelj V, Žvan B eds. Akutna možganska kap učbenik za zdravnike in zdravstvene delavce. Ljubljana: Aventis Pharma, 2006: 119-131.
- 22. Žvan B. Možganska kap dimenzija problemov v Sloveniji. Slov Kardiol 2007; 3: 16-20.
- 23. Aboderin I, Venables G. Stroke management in Europe. Pan European Consensus Meeting on Stroke Management. J Intern Med 1996; 240: 173–180.
- 24. Lausanne JB; European Stroke Initiative (EUSI), European Stroke Council (ESC), European Neurological Society (ENS), European Federation of Neurological Societies (EFNS). Stroke prevention by the practitioner. Cerebrovasc Dis 2003; 15 Suppl 2: 1-69.
- 25. www.eusi-stroke.com/2004.

Agnieszka Słowik

Genetics of stroke

Department of Neurology, Jagiellonian University College of Medicine, Krakow, Poland

Stroke is a major cause of death and the major cause of neurological disability worldwide. Epidemiological data suggest that genetic factors affect significantly stroke risk.

Several genes in single gene disorders associate with stroke have been discovered, including NOTCH3 gene in patients with cerebral autosomal dominant arteriopathy with subcortical infarcts and leucoencephalopathy (CADASIL). Little is known, however, about the genes associated with complex multifactorial stroke. Genetic-association studies on candidate

genes of haemostatic and inflammatory system, homocysteine metabolism, and the renin-angiotensin aldosterone system, suggest significant effect for several SNIPs. Genome-wide linkage studies on Iceland population showed the correlation between several haplotypes in PDE4D and ALOX5AP genes and a risk of stroke. Their significance in other populations is unclear. Recently, a new technology allows looking at thousands of variants across the human genome. One small genome-wide association study in stroke was performed so far, however, SNPs affecting stroke risk were not found. Identifying genetic factors in stroke is important because it may allow identify new stroke mechanisms, which can allow formulating novel treatment strategies.

Janika Kõrv

Epidemiology of stroke

Department of Neurology and Neurosurgery, University of Tartu, Tartu, Estonia

Stroke incidence and case-fatality, their time trends and geographical variations have already been in the centre of interest for several years. Despite some progress in primary prevention the incidence of stroke remains high. Population-based stroke registries are valuable sources in providing information about stroke epidemiology in different geographical regions. However, serious restrictions for processing personal sensitive data exist at least in Estonia and limit the use of national databases for research purposes. The incidence rates of stroke vary between study centres. The variations are probably related to environmental and life-style factors, socioeconomic differences and perhaps genetics. The results from time trend studies of stroke have been conflicting. Mostly, decline in stroke mortality is reported and some centres have shown a trend of increasing stroke incidence. Increasing incidence has been linked to the implement of computerised tomography and unfavourable changes in risk factor profiles in certain communities. Stroke time trend studies have shown that improvements in primary prevention have a significantly higher impact on the incidence of stroke compared to case-fatality (CFR). A decline in CFR has been detected in several populations over time. CFR is mostly dependent on the acute care of stroke and stroke severity. The severity of stroke might be related to the extent and quality of primary prevention. It has been shown that pre-stroke use of antiplatelets agents and antihypertensive treatment for patients with hypertension results in less severe incident stroke. The goal is to prevent stroke from happening, and therefore it is necessary to intensify the primary prevention of stroke.

Danuta Ryglewicz

Post-stroke epilepsy

First Department of Neurology, Institute of Psychiatry and Neurology, Warsaw, Poland

Stroke is the most common cause of the epilepsy among those age 60 and over. The overall occurrence of post-stroke epilepsy is estimated at 3-8%. In the Oxfordshire Community Stroke Project recurrent seizures in the first year of follow-up were diagnosed in 5.7% and within 5 years in 11.5% According to others about 15% of patients experienced unprovoked seizures within 5 years following stroke. The risk of developing epilepsy following stroke remain significantly elevated for at least 20 years following stroke. In Poland on the basis of Polish National Stroke Registry that included 3238 patients, within one year follow-up period, post stroke epilepsy was diagnosed in 3.9% of patients, more frequently among those with intracerebral hemorrhage (4.1%). In the group of patients with ischemic stroke recurrent epileptic fits more frequently have been observed among patients with cardioembolic stroke -7.5% (95% CI 4.1-11.7) than among patients with lacunar stroke -3.9% (95% CI 1.9-6.8). In the Oxfordshire Community Stroke Project the difference was even higher, only 1% of patients with lacunar strokes developed poststroke epilepsy vs 11% of patients with total anterior circulation infarct.

Post-stroke epilepsy is related to different clinical factors (Copenhagen Stroke Study): 1. younger age (OR-1.7/10 years, 95% CI 1.3-2.1), 2. increasing stroke severity at the onset of stroke (OR-1.3/10 point decrease Scandinavian Stroke Scale, 95% CI 1.0-1.6), 3. lesion size $(OR\ 1.2/10 \text{ mm}, 95\% \text{ CI } 1.0-1.3)$, 4. intracerebral hemorrhage $(OR\ -3.3, 95\% \text{ CI } 1.3-8.6, 5$. early seizures $(OR\ 4.5, 95\% \text{ CI } 1.3-16.0)$.