

Prevention of ischemic stroke in clinical practice: a role of internists and general practitioners

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ABSTRACT

Stroke constitutes a substantial clinical and socio-economic burden. It is currently the third cause of death worldwide and results in mortality or disability in every third patient at the end of the first year following an acute cerebrovascular event. Although in-hospital mortality rates in stroke patients have decreased, prevention and cardiovascular risk control remain critical for improving the prognosis and reducing stroke burden worldwide. The definitions of stroke and transient ischemic attack (TIA) have been recently modified following the findings from neuroimaging and thrombolysis research. Both stroke and TIA are recurrent and preventable disorders. Both patients with stroke and those with TIA require prompt clinical workup, risk assessment, and appropriate management because the risk of recurrence, stroke, and coronary events is significant. The 5 most common cardiovascular risk factors (high blood pressure, smoking, abdominal obesity, diet, and lack of physical activity) are responsible for 80% of the cases. Stroke prevention involves lifestyle modification and specific treatment. Secondary prevention of ischemic stroke involves early treatment (antiplatelets and carotid interventions) and long-term management including lifestyle changes, antihypertensive therapy, antiplatelets, antithrombotic drugs in patients with atrial fibrillation, and the use of statins and other lipid-lowering drugs. Stroke patients are at risk of depression, dementia, epilepsy, and other complications that also require targeted treatment.

Stroke and transient ischemic attack as clinical challenges and preventable diseases

Almost 40 years ago, the World Health Organization (WHO) defined stroke as a “neurological deficit of cerebrovascular cause that persists beyond 24 hours or is interrupted by death within 24 hours”.¹ Accordingly, symptoms lasting less than 24 hours indicate transient ischemic attack (TIA). Until recently, TIA and mild stroke were not managed as acute states and were not considered to require quick diagnostic workup and treatment. However, according to current clinical practice, both stroke and TIA need special attention owing to a risk of fully symptomatic stroke and neurological deficit as well as effective prevention and treatment that is widely available and recommended. Recently, the classic definition of TIA based on arbitrary symptom duration of less than 24 hours has been questioned. Imaging studies have shown restricted diffusion lesions at clinically significant sites

in every third patient with traditional 24-hour TIA.² An updated definition of stroke has incorporated clinical and tissue criteria with less emphasis on time span.³ Currently, stroke, and not TIA, should be diagnosed in patients with symptoms lasting less than 24 hours and either new lesions corresponding to clinical signs or thrombolysis-induced full recovery regardless of imaging findings.

Burden of stroke Stroke constitutes a substantial clinical and socio-economic burden. Currently, it is the third cause of death worldwide (in the upper-middle-income countries, as defined by the WHO, stroke ranks even second, exceeding ischemic heart disease).⁴ Annually, about 15 million people worldwide suffer from stroke, of whom one-third die in the first year. Of 30 million stroke survivors, half becomes permanently disabled. In the United Kingdom, around 20% of all acute

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TABLE 1 Predicting the risk of stroke after a transient ischemic attack, ABCD₂ score (max. 7 points)

	0 points	1 point	2 points
age, y	<60	≥60	–
blood pressure, mmHg	normal	≥140/90	–
clinical symptoms	no speech disturbance and no unilateral (one-sided) weakness	speech disturbance present but no unilateral weakness	unilateral weakness
symptom duration, min	<10	10–59	≥60
diabetes	no	yes	–

hospital beds and 25% of long-term beds are occupied by stroke patients.⁵ As age is the major non-modifiable risk factor of stroke (the risk of stroke more than doubles for each successive decade after the age of 55 years), population aging may be associated with a growing number of individuals at risk.⁶ Although in the last decades numerous interventions for acute stroke have been successfully introduced (eg, stroke unit-based treatment, thrombolysis with alteplase, decompressive hemicraniectomy in extensive middle cerebral artery stroke, or early and intensive rehabilitation), still both primary and secondary prevention is crucial in limiting the burden of cerebrovascular diseases.

Stroke epidemiology and prevention Both stroke and TIA are recurrent and preventable disorders. It should be stressed that treatment of acute ischemic stroke is still a challenge. Although in-hospital mortality has improved in developed countries, an overall long-term prognosis remains poor. Thus, prevention is crucial in limiting the burden of cerebrovascular events. WHO studies revealed an up to 10-fold difference in age- and sex-adjusted mortality rates and burden among countries, with considerably higher rates in low-income countries compared with those in high-income ones. Stroke incidence has declined by over 40% in the past 4 decades in high-income countries (further improvement seems to be challenging owing to population aging) while, over the same period, it has doubled in low- and middle-income countries owing to poor risk factor control. Effective prevention and cardiovascular risk control substantially contribute to better prognosis of stroke patients in high-income countries.

The highest risk of a recurrent cerebrovascular event is observed in the first month (4%) and year (12%) after stroke. In subsequent years, the risk is still substantial (a mean of 5% per year). Recurrent strokes still account for 25% to 30% of all strokes and represent unsuccessful secondary prevention.⁷

A 90-day risk of stroke after a TIA has been reported to be 17%, with the highest risk observed in the first week (5%–10%).^{8,9} Thus, for internists and general practitioners, it is crucial to stratify patients with TIA into a low-, moderate-, or high-risk group of early recurrent stroke. In targeting high-risk patients, the ABCD₂ score was proposed in 2007 as a modified version of

the ABCD score of 2005 (TABLE 1). Recently, double TIAs (the presence of ≥2 TIA symptoms within 7 days) have been added to the ABCD₂ score to form the ABCD₃ score, which has been further developed into ABCD₃-I by adding the presence of abnormal findings on neuroimaging.¹⁰ Neuroimaging in the ABCD₃ score may facilitate prediction of long-term stroke risk after TIA.¹¹ As neuroimaging can initially be hardly available, the use of either the ABCD₂ or ABCD₃ score is the most convenient in primary care. It should be stressed that the ABCD₂ score can be used to stratify risk but not to decide on when to start the treatment, hence all patients with TIA should be immediately assessed irrespective of the ABCD₂ score because even patients with lower scores might have treatable conditions, ie, carotid stenosis or atrial fibrillation (AF). TIA clinics are recommended as acute specialty units dedicated for these patients. In everyday clinical practice, high-risk patients, patients with recurrent TIAs, or those with no access to a quick workup on an outpatient basis need immediate hospital admission.

A comparison of the Oxford Community Stroke Project (1981–1984) and Oxford Vascular Study (2002–2004) showed that the incidence of major stroke after TIA or minor stroke can be substantially reduced (almost by half) with an increased use of preventive treatments and substantial reduction in premorbid risk factors.¹² In addition, other trials on TIA (EXPRESS and SOS-TIA) have demonstrated that immediate workup and prompt implementation of preventive measures can decrease a 90-day risk of stroke by 80%.^{13–15}

Primary prevention is aimed at effective control of stroke risk factors, while secondary prevention additionally targets a number of conditions specific for stroke. As for secondary prevention, there is no need to differentiate between stroke and TIA because both require the same management.

Primary prevention: stroke risk factors Cerebrovascular diseases share common risk factors with other cardiovascular diseases. Modifiable and nonmodifiable risk factors are presented in TABLE 2.

The INTERSTROKE study revealed that 80% of strokes are associated with 5 risk factors (high blood pressure, smoking, abdominal obesity, diet, and physical activity). Those factors are additionally associated with 5 more factors (diabetes, alcohol intake, psychosocial stress and depression,

TABLE 2 The most important nonmodifiable and modifiable stroke risk factors

nonmodifiable risk factors
age
sex (male)
race (African, Hispanic, Asian-Pacific islanders)
family history of stroke
low birth weight
modifiable risk factors
hypertension
smoking
obesity and diet
low physical activity
diabetes
alcohol abuse
psychosocial stress and depression
atrial fibrillation
high cholesterol
history of stroke or transient ischemic attack
peripheral artery disease
carotid artery disease
heart failure

cardiac causes, and lipid abnormalities), thus together accounting for 90% of strokes. Those 10 modifiable risk factors contribute the most to stroke risk and should be targeted in clinical practice. This is particularly true for hypertension, which unlike for ischemic heart disease by far more than lipids determines stroke risk, particularly hemorrhagic stroke.¹⁶ The above risk factors are used in several prognostic scoring systems, including CHADS₂ and CHA₂DS₂-VAsC scores, which are recommended in patients with AF.

Lifestyle modification Smoking cessation can be supported by behavioral therapy and pharmacological agents (nicotine replacement therapy, bupropion, cytosine, or varenicline).^{17,18} Alcohol intake should not exceed 2 drinks per day (14 drinks for males and 9 drinks for females weekly). Moderate physical activity such as walking, jogging, cycling, and swimming for 30 minutes to 1 hour 4 to 7 days a week can improve prognosis. Patients with cardiac diseases are advised to participate in medically supervised exercise programs. Body weight reduction is also beneficial; a target body mass index is below 25 kg/m², while a target waist circumference is less than 80 cm for women and less than 94 cm for men. In terms of healthy and balanced diet, fresh fruit, vegetables, dietary and soluble fiber, whole grain, protein from plant sources, dairy products low in saturated fat and cholesterol, as well as daily sodium intake below 2300 mg (the older the patients, the lower the consumption) are recommended.^{19,20}

Potentially harmful drugs or drugs of unknown efficacy in stroke Hormone replacement therapy and oral contraceptives increase the risk of stroke. Thus, such treatments should be avoided in patients

with cardiovascular diseases, and additionally, oral contraceptives are contraindicated in women aged over 35 years, those who smoke, are obese, or have a history of thromboembolism. Nonsteroidal anti-inflammatory drugs (NSAIDs), apart from acetaminophen (paracetamol), can reduce the antiplatelet effect of aspirin. Therefore, NSAIDs should be administered 8 hours before or 30 minutes after aspirin intake. NSAIDs can also increase blood pressure (decrease the efficacy of antihypertensive agents), contribute to endothelial dysfunction, or impair renal function. The net effect can translate into increased cardiovascular risk, although an increase is rather slight for an individual. The European Medicines Agency has listed coxibs, and recently also diclofenac, as the least safe NSAIDs. A large meta-analysis of individual patient data revealed that of 1000 patients randomized to a coxib or diclofenac arm for a year, 3 more had major vascular events, one of which was fatal, compared with placebo.²¹ The study also confirmed previous findings showing that high-dose naproxen was associated with lower cardiovascular risk than other NSAIDs.

Overused vasoactive and neuroprotective drugs (nicergoline, piracetam, vinpocetin, etc.), although extensively studied and shown to be effective in the animal model, have not been proved beneficial in large translational clinical trials either in acute management of stroke or prevention and treatment of its sequelae.

Secondary prevention Treatment and early prevention of acute ischemic stroke It is important to start prevention early, particularly in acute stroke. Therefore, we discuss a number of treatment options for acute ischemic stroke in the context of internal and general medicine practice. So far, a couple of interventions have been identified to improve early prognosis in patients with ischemic stroke. The first one is stroke unit-based treatment. A Cochrane review of 28 trials confirmed that organized stroke unit care (ie, hospital care provided by nurses, doctors, and therapists who specialize in caring for stroke patients and work as a coordinated multidisciplinary team) is associated with a reduced risk of death, institutionalized care, or dependency. Of note, the patient should be admitted to a stroke unit as quickly as possible²² to make sure that thrombolysis with alteplase—the only specific treatment—is introduced without delay (a number needed to treat increases by 1 every 20 minutes of delay in thrombolysis initiation). Alteplase is currently recommended for use within 4.5 hours from symptom onset. The third International Stroke Trial (IST-3) showed some benefit in selected patients within 6 hours from symptom onset and demonstrated benefit in elderly patients over 80 years of age (previously excluded from treatment) and those with severe stroke.²³

Owing to a significant risk of early recurrence of stroke (>3%),²⁴ all patients should start aspirin of 100 to 300 mg immediately after the diagnosis

(delayed until after 24 hours for those treated with alteplase and following control neuroimaging with no signs of intracranial hemorrhage). Aspirin has a minor but significant effect on post-stroke mortality and disability (about 10 deaths or recurrent strokes avoided during the first few weeks per 1000 treated).²⁵ Although the effect of clopidogrel on reducing the risk of early recurrence has not been confirmed, it was considered as an alternative in Chinese patients taking aspirin before TIA or ischemic stroke (a loading dose of clopidogrel, 300 or 600 mg followed by 75 mg daily).²⁶ A recently published Clopidogrel in High-risk patients with Acute Nondisabling Cerebrovascular Events (CHANCE) study showed that intensification of antiplatelet therapy based on aspirin and clopidogrel is beneficial in Chinese patients.²⁷ However, owing to genetic discrepancies between Caucasians and Asians, these findings should be treated as preliminary and should be confirmed in future studies (eg, the ongoing Platelet-oriented inhibition in new TIA and minor ischemic stroke [POINT] study).²⁸

Another treatment option for acute ischemic stroke is surgical decompression for cerebral edema induced by extensive middle cerebral artery stroke. Surgical decompression performed within the first 48 hours in patients younger than 60 years lowers the risk of death or severe disability, which also emphasizes the need for a prompt referral of these patients to a specialized stroke unit.²⁹

All patients with ischemic stroke should undergo noninvasive imaging of the carotid arteries, and revascularization should be performed without delay. Ideally, carotid endarterectomy should be considered within the first week after carotid territory TIA or nondisabling ischemic stroke and ipsilateral internal carotid artery stenosis of 70% to 99%.³⁰ However, a total rate of perioperative complications (neurological and nonneurological) should be less than 5%. A secondary analysis suggested that revascularization can be beneficial in internal carotid artery stenosis of 50% to 69%, but this was reported only for males, patients aged over 75 years, and patients with carotid territory hemispheric stroke.³¹

Stenting is associated with a higher short-term risk; therefore, it can be considered as an alternative to endarterectomy in patients younger than 70 years old and high risk surgery because of technical, anatomical, or medical reasons.³²⁻³⁴ Stenting is not recommended for intracranial arterial stenosis, for which the available evidence supports aggressive medical management.³⁵

Long-term prevention of ischemic stroke and transient ischemic attack Long-term prevention should be established on discharge from a stroke unit. Lifestyle modification after stroke is as necessary as before the event, although physical activity should not be too strenuous and adjusted to patient's abilities.

Antihypertensive therapy Hypertension is the most important modifiable risk factor. Effective reduction of blood pressure is crucial in stroke prevention. In primary prevention, all standard antihypertensive agents have been reported to be effective. However, recent studies have shown that a visit-to-visit variability in blood pressure is a significant risk factor for stroke and that it can be increased by some antihypertensive drugs, for example, β -blockers, especially non-selective agents.^{36,37} De Lima et al.³⁸ reported that β -blockers were ineffective in secondary prevention after stroke or TIA, although they claimed that additional studies were needed to confirm their findings. The randomized data for secondary prevention in stroke patients are limited. Based on the PROGRESS trial, all patients after stroke, both ischemic and hemorrhagic, should be offered dual therapy including an angiotensin-converting enzyme inhibitor (or angiotensin II receptor antagonist) and diuretics. Antihypertensive drugs should be selected based on comorbidities. Diuretics and calcium antagonists reduce variability in blood pressure, which was shown to increase cerebrovascular risk.³⁹ Fluctuating blood pressure increases stroke risk independently of the mean blood pressure values, thus blood pressure reduction and avoiding fluctuations represent a treatment target. The optimal time to start hypotensive therapy after stroke has not been determined, but it should generally be started after the first week and before discharge.

In patients with acute ischemic stroke, blood pressure should definitely not be lowered aggressively, and, usually, there is no need for such treatment unless blood pressure exceeds 220/120 mmHg or 185/110 mmHg in patients eligible for thrombolysis therapy. Antihypertensive therapy prior to a cerebrovascular event is usually suspended in acute stroke (it may be continued in patients with mild stroke or TIA with stable blood pressure, but careful monitoring is needed). In general, the evidence on the safety and benefits of antihypertensive drugs in acute stroke is limited.

Gradual and persistent blood pressure lowering in all stroke patients is recommended but special attention should be paid to carotid or vertebrobasilar occlusive disease owing to a possibility of hemodynamic hypoperfusion.^{40,41} Target systolic blood pressure levels have not been unequivocally established and should be individualized, but it is generally recommended to achieve blood pressure levels lower than 140/90 mmHg and, eventually, 130 mmHg in patients with lacunar stroke.

Antiplatelet drugs All patients, apart from those eligible for antithrombotic therapy, should be offered antiplatelet drugs following ischemic stroke or TIA, and, most often, they continue aspirin started during hospital stay. Dual antiplatelet therapy with aspirin and clopidogrel over 3 months is a standard treatment in patients after acute coronary syndrome; however, in stroke

patients, it was associated with a higher risk of major or life-threatening bleedings including intracranial ones.⁴² Current guidelines, including those developed by Polish stroke experts, recommend the following evidence-based antiplatelet drugs: aspirin at a daily dose of 50 to 325 mg; clopidogrel at a daily dose of 75 mg; and a combination of aspirin (25 mg) and extended-release dipyridamole (200 mg) twice daily.⁴³ Triflusal or cilostazol and less safe ticlopidine, which are hardly available in Poland, might be considered as a second-line alternative.

Antithrombotic drugs Because AF is the strongest individual risk factor associated with a mean 5-fold increase in stroke risk and because strokes associated with AF are more disabling, a more aggressive anticoagulation therapy is necessary in this patient group. Currently, all stroke or TIA patients with nonvalvular AF should be offered antithrombotic therapy rather than antiplatelet drugs. Antithrombotic drugs reduced stroke risk roughly by two thirds, while antiplatelets only by about 20%. Dual therapy with aspirin and clopidogrel is twice as effective as antiplatelet monotherapy but is associated with increased bleeding risk. Antithrombotic therapy with vitamin K antagonists (VKAs) or novel oral anticoagulants (NOACs; direct thrombin inhibitor, dabigatran; factor Xa inhibitors, rivaroxaban and apixaban) are effective in either primary or secondary stroke prevention. Based on recent studies, NOACs compared with VKAs consistently reduce the risk of intracranial bleeding and are generally preferred to VKAs, although they are still not reimbursed in Poland.⁴⁴ Patients with creatinine clearance exceeding 25 ml/min, considered unsuitable for VKAs, should be treated with apixaban rather than with aspirin.⁴⁵ Apixaban is also preferred to dabigatran and rivaroxaban in patients at risk of gastrointestinal bleeding.⁴⁶ Combined use of anticoagulants and antiplatelets should be limited to specific conditions (mechanical heart valve, recent acute coronary syndrome, or coronary stenting).⁴⁷

The overlap of ischemic stroke predictors in CHADS₂ and CHA₂DS₂-VASc scores and major bleeding in HAS-BLED score make the antithrombotic therapy challenging. The common predictors that are associated more with ischemic events are older age and previous stroke or TIA, while others (hypertension, diabetes, alcohol intake, or renal impairment) do not predict ischemic or hemorrhagic stroke.⁴⁸

The selection of an anticoagulant drug should be individualized based on renal and hepatic function, potential drug interaction, patient preference, tolerability, previous anticoagulation effectiveness and safety, as well as cost. Risk of falls, advanced age, cognitive impairment and dementia, cerebral small vessel disease (manifesting with microbleeds on neuroimaging) do not exclude anticoagulant therapy.

All AF patients with TIA can be offered oral anticoagulation immediately because the bleeding

risk, including intracranial hemorrhage, is low. Such treatment should be delayed in stroke patients with the delay depending on the extent of neurological deficit (in patients with severe stroke, it should be started even 2 weeks after stroke). Moreover, it should be stressed that antithrombotic therapy affects thrombolysis in acute ischemic stroke. VKAs are easily monitored with international normalized ratio (INR); thus, a possible bleeding risk and benefit of recombinant plasminogen activator (rtPA) can be assessed. Thrombolysis is not contraindicated when INR is lower than 1.7. The lack of a widely available and consistent anticoagulation-specific monitoring test for NOACs makes rtPA treatment challenging, and, in many cases, treatment must be discontinued. A standard coagulation test does not allow to test the patient for a recent use of NOACs. In patients who are at high risk of stroke and are ineligible for long-term oral anticoagulation, left percutaneous atrial appendage closure (or excision during an open heart surgery) may be considered.⁴⁹ The currently available data are insufficient to establish whether anticoagulation is equivalent or superior to aspirin for secondary stroke prevention in patients with patent foramen ovale (PFO), unless the venous source of embolism is confirmed, which is an indication for a more aggressive therapy (anticoagulation or, if contraindicated, an inferior vena cava filter). For PFO and coexisting deep vein thrombosis, PFO closure by a transcatheter device might be considered.⁴⁶ In patients with cryptogenic stroke, transcatheter closure may be beneficial in reducing the risk of recurrent vascular events when compared with medical treatment, especially in patients with a substantial shunt.⁵⁰

Statins and lipid-lowering drugs In patients with ischemic stroke or TIA, low-density lipoprotein cholesterol levels can be lowered with diet, physical activity, and statins to reach the target level of less than 2 mmol/l (70 mg/dl).^{51,52} Among statins, only atorvastatin was studied specifically in patients after stroke (a subgroup analysis from the Heart Protection Study [HPS] is also available for simvastatin).^{53,54} Atorvastatin at a high daily dose of 80 mg improved outcomes regardless of the baseline lipid levels; thus, statins should be considered in all poststroke patients. However, the benefit started to be evident after 2 years of treatment, and atorvastatin slightly increased the risk of nonfatal hemorrhagic stroke. New lipid-lowering compounds are being introduced into the clinical practice, but currently only a novel drug, ezetimibe, is reimbursed in Poland for poststroke patients who are not reaching the target level of low-density lipoprotein cholesterol.

Stroke sequelae Generally, stroke results in mortality and disability in every third patient at the end of the first year following an acute cerebrovascular event. Stroke patients are at risk of depression, dementia, epilepsy, and other complications

TABLE 3 Major short-term and long-term complications and sequelae of stroke

Type of complication	Epidemiology	Treatment	Comment
dysphagia	almost 50% of patients with acute stroke; swallowing disorders resolve within a few days and become long-term complications in 5% of stroke survivors	swallowing screening test; gastric tube in some patients may even reduce the risk of death	increases the risk of aspiration pneumonia
seizures	5% of patients during a few initial weeks after stroke	diazepam (10–20 mg), lorazepam (4–8 mg), or phenytoin to treat acute seizures	no need for long-term use of antiepileptic drugs, unless there have been ≥ 2 seizure episodes that required neurological consultation and possibly chronic antiepileptic treatment
spasticity and painful muscle spasms	risk depends on the quality of care	physical therapy, muscle relaxant agents, botulinum toxin	spasticity can lead to disability, pain, pressure ulcer
urinary tract infections	common; on average in every fourth patient during the initial 2 months after stroke	antibiotics; proper hydration of patients and avoiding a prolonged use of an intravesical catheter	
respiratory tract infections	common; on average in every fifth patient during the first month after stroke	early movement, physical therapy, prevention of aspiration, pneumonia antibiotics, oxygen therapy	needs to be differentiated from pulmonary embolism common cause of stroke-associated fatalities
malnutrition	common; on average in every fifth patient	proper nutrition, use of a gastric tube in patients with dysphagia	no need for routine oral use of nutritional supplements
electrolyte disturbances	common; associated with dehydration, inadequate fluid supply, and diuretic misuse	monitoring of water–electrolyte balance, adequate oral or parenteral fluid supply	may worsen neurological deficit; consciousness deficit and lower seizure threshold
thromboembolic complications (deep vein thrombosis and pulmonary embolism)	deep vein thrombosis manifests clinically in 5% of stroke patients; pulmonary embolism is a common cause of death	early movement, low-molecular-weight heparins, antiplatelet drugs, elastic compression stockings	in advanced deep vein thrombosis in patients with hemorrhagic stroke, the use of heparins should be judged by possible risks and benefits
urinary and fecal incontinence	common; almost half of hospitalized stroke patients	worsening factors must be considered (eg, diuretic misuse); intravesical catheter in some patients for a possibly short period of time	
bedsores – pressure sores	3% of the patients	appropriate care essential: change of position, mattresses for pressure ulcer prevention, adequate nutrition and fluid supply (use of dietary supplement, vitamins), wound dressing, antibiotics, physical therapy, surgical removal of the necrotic tissue	
falls and bone fractures	falls occur in every third patient; 5% of the falls lead to fractures	prevention of falls, use of hip pads	
hemiplegic shoulder pain and shoulder subluxation after stroke	common	physical therapy, analgesics on demand, appropriate placing of the patient and shoulder protection	
emotional disturbances (pathological crying, pseudobulbar affect)	10%–20% of the patients; spontaneous improvement over time	antidepressants, behavioral therapy	can significantly reduce the effectiveness of rehabilitation of cognitive and motor functions
depression	common; difficult to evaluate in patients with speech disorders	antidepressants, behavioral therapy	

(TABLE 3). Apart from specific preventive methods, a comprehensive rehabilitation program is crucial for patient recovery and limitation of disability (TABLE 3).

In conclusion, preventive measures—antiplatelet or antithrombotic therapy for patients with AF, carotid interventions, antihypertensive drugs, statins, and lifestyle modifications (including physical activity, diet, and smoking cessation)—can reduce the risk of recurrent stroke by 80%.⁵⁵ However, it is still a challenge to translate

the efficacy of the interventions reported in clinical trials into everyday clinical practice. So far, studies have failed to translate evidence-based guidelines into clinical practice and maintain them on daily basis.^{56–58} Therefore, stroke specialists as well as internists and general practitioners will need to cooperate to achieve those targets. This is especially true for Poland where a global study has revealed everyday guideline-oriented practice to deteriorate along with a decrease in income and general economic decline.⁵⁹

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Profilaktyka udaru niedokrwiennoego mózgu w praktyce klinicznej – rola lekarza internisty i lekarza rodzinnego

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SŁOWA KLUCZOWE

czynniki ryzyka,
następstwa udaru,
profilaktyka,
przemijający atak
niedokrwiennoy, udar
niedokrwiennoy

STRESZCZENIE

Udar mózgu stanowi ważny problem kliniczny i socjoekonomiczny. Obecnie jest trzecią najczęstszą przyczyną zgonów na świecie. W ciągu roku od zachorowania umiera lub pozostaje niepełnosprawny co trzeci chory. Mimo że śmiertelność wewnątrzszpitalna w przebiegu udaru mózgu się zmniejszyła, to jednak ciągle skuteczna prewencja i kontrola czynników ryzyka ma podstawowe znaczenie w poprawie rokowania i ograniczaniu następstw chorób naczyniowych mózgu. Klasyczne definicje udaru mózgu i przemijającego niedokrwienia mózgu (*transient ischemic attack* – TIA) uległy ostatnio modyfikacjom pod wpływem wyników badań neuroobrazujących oraz efektów zastosowania leczenia trombolitycznego. Udar mózgu i TIA nawracają, ale można im skutecznie zapobiegać. Zarówno pacjenci z udarem mózgu, jak i z TIA wymagają szybkiej diagnostyki, oceny ryzyka i podjęcia właściwego leczenia profilaktycznego ze względu na wysokie ryzyko nawrotu, udaru mózgu lub zgonu wieńcowego we wczesnym okresie po wystąpieniu TIA. Pięć najczęstszych czynników ryzyka (nadciśnienie tętnicze, palenie papierosów, otyłość, niewłaściwa dieta i brak aktywności fizycznej) odpowiada za 80% przypadków udaru. Profilaktyka udaru mózgu obejmuje zmianę stylu życia oraz specyficzne postępowanie terapeutyczne. W udarze niedokrwiennoym mózgu prewencja obejmuje wczesne leczenie (kwas acetylosalicylowy oraz endarterektomię tętnic szyjnych) oraz długotrwałą profilaktykę, polegającą na leczeniu hipotensyjnym, przeciwplatekowym, przeciwkrzepliwym w przypadku chorych z migotaniem przedsionków oraz stosowaniu statyn i innych leków hipolipemizujących. U chorych po udarze mózgu zwiększone jest ryzyko depresji, otępienia, padaczki i innych powikłań, które także wymagają ukierunkowanego leczenia.

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