Subsequent ischaemic event after intracerebral haemorrhage - case report

Djambazovska Zikova S[1], Vojtikiv Samoilovska D[2], Jovevska S[3], Adili Gongo F[4]

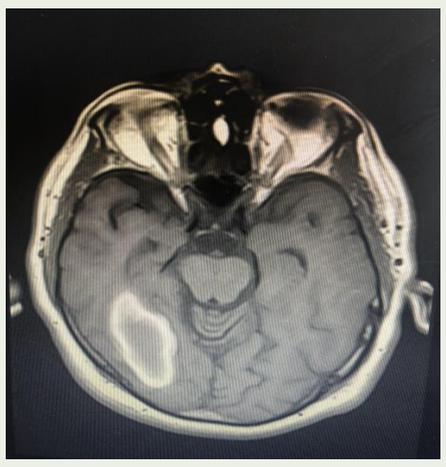
[1]Department of Neurology, CGH “8 th September Skopje, N.Macedonia, [2]Private Neurology Practice, Tetovo, N.Macedonia, [3]Faculty of Medical Science, Goce Delčev University Štip,N. Macedonia, [4]Neurology, Healthcare Center Skopje, N.Macedonia;

Introdaction

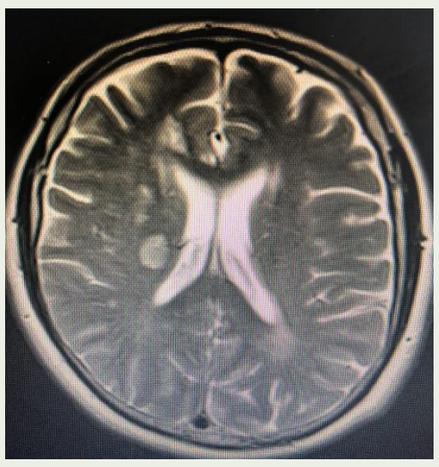
Mechanisms of ischemic stroke after intracerebral haemorrhage (ICH) include blood pressure lowering, intracranial pressure elevation and microvasculopathy. A combination of local compression, oedema, inflammation, increased viscosity at varying stages of haemorrhage may lead to thrombotic events soon after ICH. Patients with cerebral microangiopathy may have higher risk for brain ischemia. We present a case of a 55 years old man with subsequent ischemic stroke 2 weeks after ICH.

Case description

A 55 yeart old man was admitted to our hospital presented with headache and left homonymous hemianopsia. On examination his blood pressure was 200/90 mmHg, regular heart rate 95/min and ECG was unremarkable. Initial CT scan showed right occipital hematoma (56X25 mm). Laboratory values including coagulation tests were unremarkable. CT angiography of cerebral and cervical vessels was unremarkable. The patient had a recent past medical history of arterial hypertension and myocardial infarction with percutaneous coronary intervention 10 years ago. His regular medical therapy was single antiplatelet therapy, statin and antihypertensive. Antiplatelet therapy was discontinued and the patient was treated by antiedemic therapy, antihipertensive medication and diuretic. During the hospital stay he was in stable condition with blood pressure values from 150/90 mm/Hg to 170/90 mm/hg , but after two weeks the clinical picture worsened with left hemiparesis, left facioparesis and mild dysarthria without any changes in blood pressure of cardiac rhythm disturbances. He underwent MR brain that demonstrated hematoma resorption and infarction in right basal ganglia.



T1-weight MRI, axial image, right occipital intracerebral hematoma resorption



T2-weighted MRI, axial image, increased signal abnormality involving right basal ganglia

Discussion

In a recent prospective observational study, about 20% of ICH patients had incident ischemic events, with a cumulative incidence of 5.9% at the end of 1 year [1]. There appears to be a heightened short-term risk of ischemic stroke after ICH. Although this risk is the highest in the first month, it remains significantly elevated for approximately 6 months [2]. The elevated risk for ischemic sequelae is unclear but potential mechanisms may be attributable to antiplatelet drug cessation, aggressive blood pressure (BP) lowering, small vessel disease and leukoaraiosis, other factors related to hematoma volume or poor risk factor control after ICH. Elevations in intracranial pressure in the setting of medium to large hematomas or those associated with ventricular obstruction may further compromise cerebral perfusion pressures (CPP). Thus, aggressive BP lowering beyond the lower limits of cerebral autoregulation might induce cerebral ischemia in chronic hypertensive ICH patients. Alternatively, perforator disease attributable to lipohyalinosis likely coexists in chronic hypertensive patients presenting with spontaneous ICH, where relative hypoperfusion could result in ischemia in a single or multiple perforators [3]. The elevated risk of ischemic stroke may be attributable to antithrombotic drug cessation after the diagnosis of ICH and the unclear optimal time frame for resumption of these medications [2]. The link between ICH and subsequent ischemic stroke remains unclear and new researches are needed for better understanding of the ischemic risk after ICH.

Conclusion

Further study and new strategies are needed for early use of antiplatelet therapy in patients with cardiovascular and cerebrovascular risk to prevent secondary ischemic stroke after ICH. This case presents a challenge for deciding what are the appropriate prevention strategies: whether the antiplatelet therapy should be stopped, restarted or continued?

• References

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