CASE REPORT

Ischemic Stroke Following Spontaneous Intracerebral Hemorrhage in a Patient with Protein S Deficiency: A Case Report

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Abstract

Intracerebral hemorrhage (ICH) accounts for 20% of all strokes and is a significant cause of mortality and disability. Intracerebral hemorrhage survivors are at high risk of recurrence of ICH with a risk of 1.3–7.4%. While we are more concerned with the recurrence of ICH, ischemic infarcts are also frequent. This raises the therapeutic dilemma about the benefit of starting antithrombotic agents without increasing the risk of ICH. Here we report a patient who presented to our hospital with hypertensive parenchymal bleed and later developed cerebellar infarct within 1 week of admission. Our patient is a 40-year-old male without any comorbidities presented with accelerated hypertension and right hemiparesis. Computed tomography (CT) was done immediately showed intracerebral bleed with intraventricular bleed. Repeat CT brain one week later showed subacute infarct in cerebellar region. On further work he was found to have protein S deficiency.

Intracerebral hemorrhage survivors have an increased risk of recurrent hemorrhage and ischemic stroke. Data on the burden of ischemic events and their predictors following an ICH is still lacking. Various studies observed that prior ischemic stroke, aggressive blood pressure lowering, and surgical evacuation of ICH were independently associated with diffusion-weighted lesions. It is also postulated that aggressive lowering of blood pressure in chronic hypertensive ICH patients can compromise cerebral perfusion pressure which contributes to ischemic injury. According to the limited studies so far, primary ICH patients are at risk of another major vascular event including ischemic stroke. This poses a therapeutic dilemma about blood pressure lowering and also the time frame to start antithrombotic. Further studies may be needed to determine and analyze the risk factors for developing ischemic injuries and the impact of these infarcts on the outcome and optimal management strategies to arrest vascular damage.

Keywords: Case report, Intracerebral hemorrhage, Ischemic stroke, Ischemic stroke following intracerebral hemorrhage, Protein S deficiency.

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Introduction

Intracerebral hemorrhage (ICH) accounts for 20% of all strokes and is a significant cause of mortality and disability. Intracerebral hemorrhage survivors are at high risk of recurrence of ICH with a risk of 1.3–7.4%. While evaluating the risk, the location of ICH is an important factor. Lobar ICH has a higher recurrence rate for ICH. While we are more concerned with the recurrence of ICH, ischemic infarcts also occur frequently. This raises the therapeutic dilemma about the benefit of starting antithrombotic agents without increasing the risk of ICH. Data on the burden of ischemic events and their predictors following an ICH is still lacking.[1] According to their study, the incidence of major ischemic events is six times higher than hemorrhage in 5-year follow-ups. In a study by Hill MD et al. the incidence of readmission with ischemic stroke is 3% per year compared with 2.4% for hemorrhagic strokes.[2]

Here we report a patient who developed a cerebellar infarct, admitted with primary ICH, and was found to have protein S deficiency.

Case Description

This 40-year-old male patient with no chronic medical problems was brought to our hospital with a history of vomiting and right-sided weakness which started 4 hours prior to admission. On arrival, patient Glasgow coma scale (GCS) was 7 and he had right-sided dense hemiplegia. His blood pressure was 225/105. Brain computed tomography (CT) showed left deep parietal white matter acute hemorrhage with a midline shift of 3.5 mm with intraventricular hemorrhage within lateral, third, and fourth ventricles (Fig. 1). Labs revealed renal impairment with normal electrolytes and other routine blood investigations. He was started on antiedema measures and antihypertensives as per protocol. The case was discussed with our neurosurgical team, who advised conservative management. On the day third patients condition deteriorated and he was intubated and ventilated. Computed tomography brain

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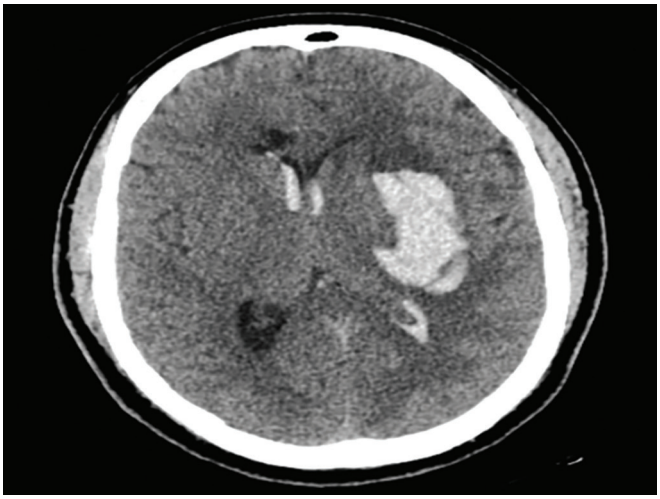

Fig. 1: Computed tomography (CT) showing left basal ganglia bleed with IVH

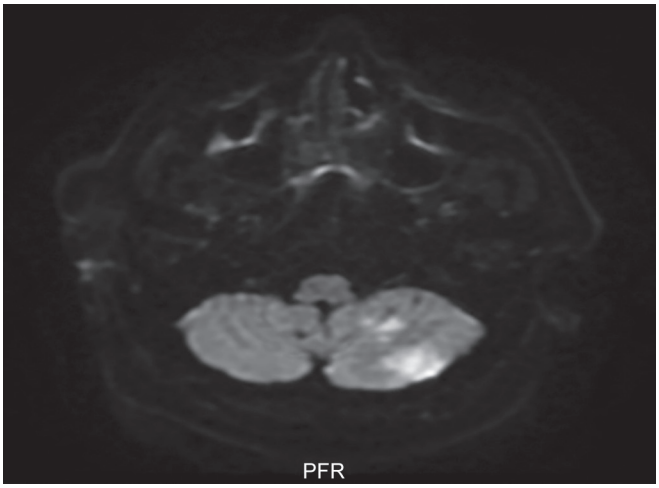

Fig. 2: Diffusion-weighted image showing left cerebellar infarct

repeated shows a mild decrease in hemorrhage density and newly developed bilateral cerebellar hypodensity suggesting subacute infarcts. Brain magnetic resonance imaging (MRI) done which revealed subacute basal ganglia hematoma with acute infarcts in the left cerebellum and left midbrain (Fig. 2). On workup, he was found to have protein S deficiency. His echocardiography (ECHO) didn't show any thrombus and the carotid artery Doppler was normal.

Discussion

Intracerebral hemorrhage survivors have an increased risk of recurrent hemorrhage and ischemic stroke. Data regarding the causes and management strategies are lacking. According to the study by Menon RS et al.[3] more than one-third of primary ICH suffer ongoing ischemia at baseline and more than one-fourth of patients develop ischemic stroke at one month. In another study by Prabhakaran S et al.[4] more than one-fifth of the patients had subclinical, subcortical acute brain infarctions as demonstrated by diffusion-weighted lesions. He also observed that prior ischemic stroke, aggressive blood pressure lowering and surgical evacuation of ICH were independently associated with diffusion-weighted lesions. He also postulated that aggressive lowering of blood pressure in chronic hypertensive ICH patients can compromise the cerebral perfusion pressure which contributes to ischemic injury. According to the limited studies so far, primary ICH patients are at risk of another major vascular event including ischemic stroke. This poses a therapeutic dilemma about blood pressure lowering and also the time frame to start antithrombotics. Further studies may be needed to determine the impact of these lesions on outcomes and optimal management strategies to arrest vascular damage.

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