Non-Hypertensive Intracerebral Haemorrhage: Some Interesting Observations

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Abstract

Hypertension is the commonest cause of intracerebral hemorrhage (ICH) but non-hypertensive intracerebral hemorrhages (NHICH) are not rare. We report three interesting cases of NHICH subsequent to amyloid angiopathy, alcoholic hepatitis and amphetamine abuse. They suggest the importance of recognizing these conditions and need for urgent specific therapy which may play a vital role in therapeutic planning and prevention of ICH.

INTRODUCTION

Though hypertension is the commonest cause of intracerebral hemorrhages (ICH), non-hypertensive intracerebral hemorrhages (NHICH) are not rare. There are many non-hypertensive causes of ICH including cerebral amyloid angiopathy (CAA), vasculitis, vascular malformations, and the use of anticoagulants, fibrinolytic or sympathomimetic agents, etc.1, 2 We report three interesting and important cases of NHICH illustrating its pleomorphism. They suggest the importance of recognizing these conditions and need for urgent specific therapy which may play a vital role in therapeutic planning and prevention of ICH.

CASE REPORTS

Case 1

A 64-year male had ICH about 6 years back (1999) from which he recovered completely, without neurological deficit. Cranial MRI had revealed ICH in right temporoparietal region with perifocal oedema (Fig. 1a). He then presented 2 years later with acute headache, vomiting and delirium. On examination, he was confused and talking irrelevantly (later this was observed to be due to Wernicke’s aphasia). Cranial CT revealed left parietal hematoma (Fig. 1b). Previous DSA was normal. Patient was managed conservatively. There was significant recovery with mild nominal aphasia as residual deficit.

This patient continues to report and in a span of 5 years had four recurrent cerebral hemorrhages at different locations in brain. There was a remarkable recovery each time in sensorium, mental functions, speech, but progressive decline in motor functions due to increasing spasticity resulting in significant invalidity. Since he was never detected to be hypertensive on successive hospitalization and had multiple lobar bleeds, a provisional diagnosis of CAA was kept. The diagnosis of CAA is established by cerebral biopsy, which was not considered by the relatives due to his significant recovery.

Fig. 1(a) : Cranial MRI revealing lobar hemorrhage in right temporoparietal region.
Case 2

A 50-year, non-hypertensive male presented 2 years back with acute occipital headache, vomiting, right hemiparesis and delirium, evolving over 1-2 hours. He was an established case of alcoholic liver disease (edema, ascitis and icterus were present). Right hemiparesis (power grade 3) and left homonymous hemianopia were evident. Platelet counts were reduced (53,000/mm³). Liver functions were deranged (serum bilirubin- 3.2 mg, SGOT- 95, SGPT- 105, total serum proteins- 5.6gm%, serum albumin- 2.5 gm%, and prothrombin time (PT)= 21 sec against control of 14 sec and activated partial thromboplastin time (APTT) = 60 sec with control of 30 sec). Cranial CT revealed a large right parieto-occipital hematoma (Fig. 2). Patient recovered significantly with injection of vitamin K, fresh frozen plasma and platelet transfusion.

His consumption of alcohol increased and he reported 2 years later with fresh ICH and deranged hepatorenal functions. He developed bleeding at various sites including upper and lower gastrointestinal tract and expired.

Case 3

A 40 year, non-hypertensive, young male businessman, used to take amphetamines during his exams as a student. Within a period of 3 years he started requiring it in his daily studies also. Consumption increased in frequency and quantity when he took to business and became significantly habitual to it. He presented with severe headache and altered sensorium. This was followed by weakness of right side. Examination revealed Glasgow scale of 7, hemiparesis of right side with power grade 2. All investigations including cardiac, vasculitis, hepatorenal and metabolic parameters were normal. Cranial CT was normal but MRI revealed a linear hemorrhage. (Fig. 3) He improved with conservative management. Psychiatric assistance helped him to recover from amphetamine habituation. He continues to report without any residual deficit.

DISCUSSION

The first case was provisionally diagnosed as CAA since confirmation is possible only by postmortem demonstration of amyloid in cerebral vessels by Congo red staining. It is probably the commonest cause of lobar hemorrhage in elderly, in which arteriolar degeneration occurs and amyloid is deposited only in walls of cerebral arteries and nowhere else. It can cause both single and multiple episodes of recurrent lobar ICH over months or years. Amyloid angiopathy is probably underrepresented due to lack of autopsy correlation. Our patient of chronic alcoholic hepatitis developed ICH subsequent to thrombocytopenia, which is a major risk factor for spontaneous ICH, especially in non-hypertensive patients. This has been attributed to increased PT and APTT values and lower platelet
counts, compared to the hypertensive.\textsuperscript{3,4}

Patients with spontaneous non-hypertensive basal ganglia and cerebellar ICH have no evidence of prior protracted hypertension, especially in geriatric age group. Analysis of circumstances associated with non-hypertensive, basal ganglia hemorrhages should help in understanding the mechanism of the hemorrhages. Other circumstances in which non hypertensives basal ganglia bleeds have been reported are drug abuse (cocaine, amphetamine, and phenylpropanolamine), embolic infarcts, correction of congenital heart defects and after carotid endarterectomy.\textsuperscript{5,6} Cocaine abuse has been independently associated with significantly increased risk for ICH.\textsuperscript{5,7} The important concept is the relatively acute augmentation of cerebral blood flow, most often by an abrupt increase in blood pressure in the region of either normal or injured blood vessels. This situation is most common at onset of arterial hypertension.\textsuperscript{1,3,4} This mechanism along with the damage to the penetrating blood vessels caused by chronic arterial hypertension probably explains most of spontaneous intracerebral hemorrhages (excluding AVM bleed, bleeding disorders and CAA). The two mechanism may often co-exist.\textsuperscript{1,2}

We conclude that though hypertension remains the commonest cause of ICH, other causes should be contemplated especially if patient is young, non-hypertensive or bleeding occurs at sites not typical for hypertension.\textsuperscript{5,6} In such cases all efforts should be directed to establish the etiology of ICH so that optimal therapy can be provided.

\textbf{REFERENCES}