CASE REPORT

Simultaneous hypertensive intracerebral haemorrhages: what are the odds?

Osama S M Amin

Department of Neurology,
Sulaimaniya General Teaching
Hospital, Sulaimaniya City, Iraq

Correspondence to

Dr Osama S M Amin,
dr.osama.amin@gmail.com

SUMMARY

The simultaneous development of two (or more) spontaneous, hypertensive, non-traumatic intraparenchymal cerebral haemorrhages is rare and constitutes less than 5.6% of all hypertensive cerebral haemorrhages. In addition to having a high early mortality, these haemorrhages carry a considerable morbidity figure in patients who survive the event. We report a 68-year-old hypertensive and diabetic woman who presented with a sudden onset of headache, vomiting, and dense right-sided weakness. In less than an hour, she became obtunded. An urgent non-contrast CT brain scan revealed two left-sided supratentorial hemispheric haemorrhages; putamenal and thalamic.

BACKGROUND

These simultaneous haemorrhages are deep-seated within the brain parenchyma. Their location cancels out any benefit from surgical evacuation and leaves the role of medical treatment restricted to the control of the elevated intracranial pressure, systemic hypertension and other vascular risk factors.

CASE PRESENTATION

A 68-year-old right-handed woman was brought to our A&E by her family. The latter stated that their mother developed sudden severe headache, repeated vomiting and right-sided weakness 1 h ago. She had longstanding poorly controlled hypertension but her type II diabetes was reasonably controlled using oral metformin tablets. The family denied any head trauma, seizures and illicit drug ingestion. No history of a previous stroke of any kind was obtained and a review of her medications did not show any antiplatelets or anticoagulation administration. There was no family history of note. Her blood pressure was 248/121 mm Hg with a regular pulse rate of 113 beats/min, a respiratory rate of 21 cycles/min and a temperature of 37.1°C. She was obtunded with a Glasgow coma scale of 5/15 and she demonstrated flaccid dense right-sided weakness, areflexia and extensor planter. An urgent non-contrast CT brain scan showed two supratentorial intraparenchymal haemorrhages; putamenal and thalamic (figure 1). The patient was admitted into the intensive care unit where she stayed for 25 days. During this period, her blood pressure was controlled with enalapril, metoprolol, amlodipine and frusamide. Soluble insulin was the antidiabetic agent. The other medications were atorvastatin, ranitidine and domperidone. There was no need for assisted ventilation.

Her neurological condition gradually improved and on day 17 she was drowsy, had grade 1 right-sided pyramidal weakness, exaggerated reflexes and extensor planter. There was no speech output but testing for comprehension was intact. On day 25 she was transferred to the general neurological ward where she stayed for a further of 11 days and then discharged home. At this point, she was still somewhat drowsy but she could utter few sounds only; her limb weakness had not changed.

INVESTIGATIONS

Her blood tests at the time of admission revealed combined hyperlipidaemia (triglyceride of 268 mg/dl and total cholesterol of 254 mg/dl) and hyperglycaemia (of 237 mg/dl); the rest of her investigations (including platelet count, partial thromboplastin and activated partial thromboplastin time) were within their normal reference range and her urine testing showed a trace of albumin and a '+' of glycosuria. Because of lack of expertise

Figure 1 Urgent non-contrast axial CT brain scan of the patient at the time of A&E admission. Note the presence of two left-sided hyperdense hemispheric lesions. These are putamenal (red arrow) and thalamic (yellow arrow) haemorrhages. The lack of surrounding (cytotoxic) oedema indicates a very early presentation. Both haematomas behaved as very rapidly growing space-occupying lesions, have compressed the ipsilateral ventricular system and have resulted in a midline shift of brain structures. The patient became obtunded in less than an hour.

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in our radiology department, we did not order conventional cerebral angiography; in addition, the location of the haematomas suggests that hypertension is the culprit behind their development.

OUTCOME AND FOLLOW-UP

The patient was scheduled on a regular physiotherapy programme and follow-up visits. Two months after the development of her stroke, her neurological examination revealed right-sided spastic pyramidal weakness of grade 2 with exaggerated deep tendon reflexes and extensor planter. She was conscious and her language assessment demonstrated non-fluency, intact comprehension and impaired repetition and naming; because she was illiterate, assessment of reading and writing was not possible. Swallowing was intact. Her up-to-date non-contrast CT brain scan is shown in figure 2.

DISCUSSION

Intracerebral haemorrhage (ICH) carries a considerable death rate and comprises around 10–15% of all stroke types. The list of aetiologies behind the development of ICH is long, but systemic hypertension is responsible for at least half of the so-called non-traumatic spontaneous haemorrhages. Therefore, the majority of such devastating haemorrhages are potentially preventable.^{1–3}

In the brain, a longstanding elevation in the systemic blood pressure affects the small penetrating arteries and gradually results in a necrotising form of arteriopathy; fibrinoid degeneration and lipohyalinosis then ensue. The walls of these blood vessels weaken and with time, microaneurysms of Charcot-Bouchard develop. The sudden rupture of the latter marks the development of ICH.^{4–6}

These pathological changes are observed predominantly in the small penetrating arteries that branch off major intracerebral arteries often at 90° with the parent vessel; lenticulostriates (which supply the basal ganglia), thalamoperforators (which

nourish the thalamus), superior cerebellar and paramedian branches of the basilar artery are the usual targets.⁷ The majority (approximately 80%) of these hypertensive haemorrhages are seen in the supratentorial compartment while the rest (20%) develop in the posterior fossa (deep cerebellar nuclei and basis pontis).^{5, 8}

According to Weisberg *et al*,⁹ the most common sites of non-traumatic hypertensive haemorrhages (in order of decreasing frequency) are the putamen (which comprises 50% of all cases); the central white matter of the temporal, parietal or frontal lobes (so-called lobar haemorrhages); the thalamus; a cerebellar hemisphere (deep nuclei); and the basis pons.

Our patient developed two left-sided supratentorial hemispheric haemorrhages; putamenal and thalamic. The patient's presentation, the temporal profile of her symptomatology and the brain imaging finding indicate that both of these haemorrhages occurred simultaneously.

Several researches tried to tackle the incidence of simultaneous/multiple hypertensive ICH and they came up with diverse results. The lowest incidence was suggested by Tanno *et al*¹⁰ who found that 0.7% of their 679 patients presented with simultaneous haemorrhages. On the other hand, Shiomi *et al*¹¹ diagnosed 11 cases of simultaneous ICHs out of 1069 ones, that is, a figure of 1%. Mauriño *et al*¹² analysed 142 patients with hypertensive ICH and found that 2.4% of their patients demonstrated simultaneous haemorrhages. However, Stemer *et al*¹³ reviewed 522 consecutive patients admitted to their institution with hypertensive ICH between August 2006 and November 2009 and found that 5.6% of their patients had multiple/simultaneous hypertensive ICHs. In our previous report,⁴ the highest reported incidence was around 2%; therefore, Mauriño *et al*'s¹² work delivers a message that simultaneous ICHs might have been more common than reported.

Several researchers found that as with solitary ICH, hypertension is still the most important aetiological factor for simultaneous multiple ICHs and that there were no age or gender difference among its victims.^{4, 10–12} According to Yen *et al*,¹⁴ only hypercholesterolaemia was identified to be significantly 'associated' with this unusual brain event in their study.

The possible mechanism (or mechanisms) behind the development of simultaneous haemorrhages is still a matter of debate. However, Tanno *et al*¹⁰ and Shiomi *et al*¹¹ suggested that the bleeding might have occurred simultaneously in different regions of the brain, or that the initial bleeding was followed after a short time by a secondary one at another site due to high intracranial pressure and circulatory disturbance.

Many investigators tried to uncover the commonest targets for simultaneous haematomas. Shiomi *et al*¹¹ found that putamen–cerebellar haemorrhages were the commonest combination while Tanikake *et al*¹⁵ concluded that bilateral thalamic–thalamic location was the usual combination; the latter observation is consistent with that of Yen *et al*.¹⁴ On the other hand, Weisberg *et al*⁹ suggested that the 'lobar' location was the commonest site for homing multiple haematomas. Kohshi *et al*¹⁶ reported on two cases of simultaneous ICHs; one of their cases had two left-sided (ipsilateral) putamenal and thalamic haemorrhages, an observation that is consistent with ours. Apart from Kohshi *et al*'s paper,¹⁴ the available literature does not credit this site as one of the usual combination targets; therefore, it seems that this localisation is a rare one. Previously, we reported on the same subject but that patient had ipsilateral right-sided putamenal and cerebellar haemorrhages.⁴ The pertinent medical literature^{8–14} does not mention whether the left side of the brain is more commonly involved than the right one. In

Figure 2 Non-contrast axial CT brain scan of the patient 58 days after the first event. Note the presence of two small oval-shaped hypodensities at the left putamen and thalamus. These represent the site of the previous corresponding haemorrhages. The patient has right-sided spastic hemiparesis and is dysphasic.

addition, there were no characteristic initial symptoms or neurological signs that might suggest which haemorrhage had occurred first; the only exception was that prominent initial cerebellar symptoms and signs might indicate that the initial haemorrhage was the cerebellar one.

Surgical evacuation of simultaneous/multiple intracerebral haematomas is controversial but Shiomi *et al*¹¹ suggested that surgical treatment for multiple haematomas should be determined by the location and maximum axis of the haematoma. We consulted the neurosurgical team who declined any form of surgical intervention.

Our patient's haematomas were relatively large and created a rapid rise in the intracranial pressure with midline shift and ipsilateral ventricular compression. This observation explains the rapidly progressive cloudiness in consciousness this patient had. Both haematomas obviously compressed the intervening internal capsule and the resulting contralateral weakness was prominent, therefore. The language impairment of our patient points to a subcortical thalamic-type dysphasia. During the period of follow-up (of 2 months), the patient did not demonstrate any form of involuntary movements. She is wheel-chair bound, dysphasic and totally dependent on her caregiver.

Both, the short-term and long-term prognoses are poor. This gloomy outcome can be explained by the concomitant destruction of crossing and non-crossing fibre tracts and bilateral diaschisis phenomenon.¹⁴

Learning points

- ▶ Simultaneous intracerebral haemorrhages may be more common than reported.
- ▶ Chronic hypertension is the commonest underlying aetiology.
- ▶ The combination of ipsilateral putamenal-thalamic haemorrhages is rare.
- ▶ Both short-term and long-term prognoses are poor.

Competing interests None.

Patient consent Obtained.

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