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Simultaneous bleeding from multiple lenticulostriate arteries in hypertensive intracerebral haemorrhage

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Abstract Angiography within 1 h of the onset of an intracerebral haematoma in a hypertension man showed active bleeding from at least two lenticulostriate arteries. We discuss the pathophysiological significance of this finding.

Key words Haemorrhage, intracerebral, spontaneous · Hypertension · Lenticulostriate arteries

Introduction

As angiography in acute intracerebral haemorrhage declines in frequency, extravasation of contrast medium is rarely seen. We describe a case in which a study performed in the hyperacute stage revealed multiple bleeding points.

Case report

A 62 year-old man suddenly developed altered consciousness and a severe right hemiparesis. His past medical history was of uncontrolled hypertension for 2 years. On admission, he was somnolent with a right hemiparesis. Laboratory data, including haematological investigations, were within normal limits. CT showed a 5-cm-diameter left putaminal haemorrhage. A ruptured aneurysm could not be excluded since the base of the haematoma was near the left sylvian fissure. Cerebral angiography was therefore performed an hour after the ictus. The first injection into the left common carotid artery (frontal view) showed extravasation of contrast medium from the medial and lateral lenticulostriate arteries (LSAs) (Fig. 1). The appearance and pooling of the contrast medium leaking from the two branches were exactly synchronous. No further angiography was attempted. Subsequent CT showed a larger haematoma, with a diameter of 8 cm extending into both the lateral ventricles and marked midline shift (Fig. 2). The CT numbers in haematoma were about 65 HU, while extravasated contrast media with CT numbers of 150–230 HU was seen at its anterior and posterior poles. These two higher density areas were distinctly separate. The patient remained unresponsive after the angiogram and died 7 days later.

Discussion

With the advent of CT, cerebral angiography is seldom performed in hypertensive intracerebral haemorrhage, except to exclude an underlying aneurysm or arter-

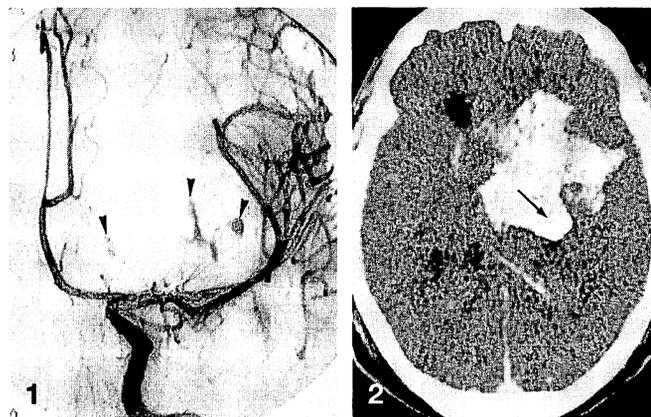


Fig. 1 Left common carotid injection demonstrates extravasation of the contrast medium from both the medial and lateral lenticulostriate arteries (*arrowheads*)

Fig. 2 CT following the angiogram shows the enlarged haematoma, which has broken through into the ventricles. The markedly high-density areas of the extravasated contrast medium were located at the anterior and posterior poles (*arrow*) of the haematoma and were distinct from one another

iovenous malformation. There are a few reports, from before the CT era, of extravasation of contrast medium in the very early stage in the hypertensive intracerebral haemorrhage [1-5].

It is uncertain whether hypertensive intracerebral haemorrhage occurs from single or multiple branches of the LSAs. It is said that rupture of a single perforating artery cannot produce a haematoma more than 1 cm in diameter [6]; if this were true, a larger haematoma would imply simultaneous bleeding from several vessels or the primary haemorrhage inducing a secondary haemorrhage.

Our case differs from previous reports in that extravasation occurred from multiple perforating vessels si-

multaneously. The angiogram clearly demonstrated simultaneous bleeding from multiple LSAs. This would count against the belief that a single bleed, without subsequent ruptures, is responsible for all cases of hypertensive intracerebral haemorrhage. Although we cannot differentiate a single primary, haemorrhage with subsequent bleeding from adjacent arteries and/or veins and multiple primary simultaneous haemorrhages from the LSAs, we favour the former, since primary rupture should occur at a single site.

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References

1. Westberg G (1966) Arteries of the basal ganglia. *Acta Radiol Diagn* 5: 281-296
2. Huckman MS, Weinberg PE, Kim KS, Davis DO (1970) Angiographic and clinico-pathologic correlates in basal ganglionic hemorrhage. *Radiology* 95: 79-92
3. Leeds NE, Goldberg HI (1970) Lenticulostriate artery abnormalities. Value of direct serial magnification. *Radiology* 97: 377-383
4. Kowada M, Yamaguchi K, Matsuoka S, Ito Z (1972) Extravasation of angiographic contrast material in hypertensive intracerebral hemorrhage. *J Neurosurg* 36: 471-473
5. Wolpert SM, Schatzki SC (1972) Extravasation of contrast material in the intracerebral basal ganglia. *Radiology* 102: 83-85
6. Ooneda G (ed) (1974) Pathology of cerebral haemorrhage (in Japanese). *Bunkodo, Tokyo*, pp 96-114