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Abstract

Bilateral thalamic ischemia and hemorrhages are unusual [1-5]. They almost exclusively affect the posterior paramedian thalamic region supplied by the artery of Percheron and rarely the anterior polar artery territory causing a variety of cognitive, arousal and personality deficits [6].

To our knowledge, we report the first patient who had the unusual presentation of a left paramedian hypertensive thalamic hemorrhage followed within three days by a right paramedian thalamic infarction.

Keywords
Hypertension, Hemorrhagic

Case Report

An 84-year-old woman was found with an altered mental status and right hemiparesis. She had CHF, hypertension and hyperlipidemia under adequate pharmacologic treatment. An initial CT (Figure 1A) showed a left paramedian thalamic hemorrhage with mass effect over the third ventricle (arrow). Her blood pressure on admission was 190/100 mmHg requiring IV labetalol and sodium nitroprusside for control. Her EKG revealed AF and the general laboratory results were normal including sed rate. Transcranial doppler results did not reveal abnormal velocities suggestive of stenosis in the posterior circulation. At 72 hrs from admission her mental status deteriorated and a new CT (Figure 1B) did not show hydrocephalus or significant changes in the hemorrhage although a new hypodensity was observed in the right paramedian thalamic territory. On

Figure 1: CT evolution over a week showing the left thalamic hemorrhage (green arrow in figure A) and a right thalamic infarction (red arrows in figures B and C).
day 5, she did not follow commands, her speech was severely
dysarthric and weakness on the right became more severe.
A CT (Figure 1C) confirmed the infarction on the right
paramedian thalamic territory. On day 7 the patient was
stuporous, was intubated and died 24 hrs later.

Hemorrhagic vascular thalamic lesions occur most
commonly in the lateral thalamo-geniculate artery territory
(75.9%) followed by the paramedian (15.5%), posterior
ciliary (5.3%) and the anterior polar artery (3.3%)
territories [7]. Thalamic hemorrhages are more common in
elderly individuals with hypertension as was the case of our
patient [7, 8]. Bilateral thalamic hemorrhages have rarely
been reported [4]. Bilateral thalamic ischemic vascular
lesions have been reported as 0.6% of all infarctions [5].
Bilateral ischemia may occur because the artery of Percheron
supplies the paramedian nuclei bilaterally in up to 12% of
individuals [1, 2, 9]. At times, the anterior thalamic nuclei,
usually supplied by the polar artery arising from the posterior
communicating artery on each side, may receive their vascular
supply from the Percheron artery or from a single polar artery
explaining bilateral anterior paramedian infarctions [3]. A
venous infarction would be unlikely in an 84-year-old without
history of a hypercoagulable state and imaging is supportive
of arterial territory rather than venous (more diffuse and with
associated edema) compromise. Atrial fibrillation could cause
small vessel disease; however, the CT was obtained within a
few hours from stroke onset without allowing time for such a
significant hemorrhagic transformation. Our patient had a left
thalamic paramedian hemorrhage secondary to hypertension
followed by an ischaemic stroke in the same vascular territory
of the contralateral side. The left sided arteriolar branch
of Percheron’s artery likely developed a saccular Charcot-
Bouchard aneurysm that ruptured causing a hemorrhage. We
postulate as probable explanations of the exceptional scenario
of hemorrhage and contralateral thalamic infarction that
mass effect from the hemorrhage strangled the right sided
Percheron’s arteriole branches leading to infarction in that
territory or alternatively that vasospasm occurred secondary
to the presence of blood resulting in ischemia and infarction
on the right.

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