Management of Large Hemispheric Infarction

K.E. Wartenberg and S.A. Mayer

Introduction: Natural History of Large Hemispheric Infarction

Large hemispheric infarctions due to middle cerebral artery (MCA) or internal carotid artery (ICA) occlusion are an important cause of morbidity and mortality in the neurological intensive care unit (ICU). Neurological deterioration occurs as a consequence of malignant cerebral edema in approximately 5–10% of hemispheric ischemic strokes [1–3], but in over two-thirds of patients when the complete MCA territory is infarcted [1, 3]. The reported mortality of these ‘malignant’ hemispheric infarctions varies between 42 and 80% [1–5].

Patients with complete MCA infarction are generally 10 years younger (mean age 56 years) than the average stroke patient [1]. The initial presentation usually includes contralateral conjugate gaze paresis, hemineglect, and reduced level of consciousness in addition to the expected sensorimotor and language deficits [1, 6, 7].

Most patients experience neurological decline within 48 hours [1, 3]. Of those who deteriorate, worsening occurs within 24 hours in 36% and within 48 hours in 68% [3]. The first sign of transtentorial herniation is usually drowsiness, followed by pupillary asymmetry, hyperventilation, and contralateral motor posturing [8, 9]. Autonomic abnormalities may include hyper- or hypoventilation, bradycardia, and sustained hypertension or blood pressure lability [1, 3, 5, 7]. Bilateral motor posturing and lower extremity rigidity then follows as the midbrain and diencephalon are subjected to physical distortion and compression [8]. Without life support, death typically occurs within five days [1, 3, 5] as a result of brain death, respiratory failure, cardiac arrhythmia, or pneumonia [1–3].

Infarction of the brain parenchyma and the vasculature results in a delayed break down of the blood brain barrier with extravasation of serum proteases and worsening of brain edema 24 to 72 hours after the initial infarct signs [9]. Hemispheric brain swelling leads to brain tissue shifting with subsequent brain stem distortion, bihemispheric dysfunction through mechanical displacement, vascular compression, uncal and transtentorial herniation (Fig. 1). Intracranial pressure (ICP) is usually not elevated early in the process of transtentorial herniation from large hemispheric infarction, but increases later as severe cytotoxic edema ensues. Ongoing ischemia is usually not the cause of neurological deterioration beyond 24 hours of onset, but this can result from vascular compression of the anterior and posterior cerebral arteries against the falx or tentorium, and is a universal finding in patients who become brain dead [4].
**Fig. 1.** Schematic diagram of the importance of tissue shifts and hypothetical significance of pressure differentials in clinical worsening from large hemispheric infarction with edema. $P_1$ represents the pressure in the injured hemisphere and $P_2$ the pressure in the uninjured hemisphere. As edema ensues, pressure differentials occur and accentuate, leading to tissue shifts and clinical worsening. From [4] with permission.

## Etiology of Large Hemispheric Infarctions

Large hemispheric infarctions occur as the consequence of an occlusion of the distal ICA or proximal MCA trunk without sufficient collateral flow (Figs. 2 and 3). Total ICA occlusions lead to infarction of the anterior cerebral artery (ACA) and MCA territories [7].

Most patients have risk factors for vascular disease such as hypertension, diabetes, hypercholesteremia, tobacco abuse, history of transient ischemic attacks or ischemic strokes, congestive heart failure (CHF), and coronary artery disease. Atrial fibrillation is more frequent in patients with MCA and ICA territory strokes compared to the remaining stroke population [1–3, 6]. ICA dissection is a significant cause of large territory infarctions in younger patients (12%) [6]. In one series of 610 patients with large hemispheric strokes 42% were attributed to focal or general atherosclerosis and 33% to a cardioembolic source [6].

## Diagnosis of Early MCA Infarction

Computed tomography (CT) of the brain obtained within 6 hours of symptom onset has a sensitivity of 82% for ischemic hemispheric infarctions [10]. Early infarct signs on CT include:

- Hyperdense MCA sign (high contrast in the MCA that is brighter than the adjacent brain tissue and other intracranial arteries in the absence of calcification) (Fig. 4)