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Cisternal Enhancement after Subarachnoid Hemorrhage

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Abnormal computed tomographic enhancement in the basal cisterns and cortical sulci was observed in 21 of 42 patients after subarachnoid hemorrhage. The appearance is similar to that described in granulomatous infection and metastatic disease. The enhancement was associated with an increased incidence of hydrocephalus, but it did not correlate with clinical grade, arterial spasm, location of bleed, or temporal relation to bleed. Presumably, abnormal cisternal enhancement is due initially to increased vascular permeability and later to increased vascularity associated with arachnoiditis.

Abnormal enhancement of the cisterns on computed tomography (CT) has been recognized in bacterial meningitis [1], subarachnoid seeding of tumor [2–4], and more recently after subarachnoid hemorrhage [5–7]. A retrospective review of patients with subarachnoid hemorrhage was performed to determine the incidence and significance of cisternal enhancement.

Materials and Methods

During an 18 month period, 88 patients were admitted to Kaiser Hospital, Redwood City, Cal., a neurologic referral center, for evaluation of subarachnoid hemorrhage. Of this group, 42 patients 14–74 years of age were studied with pre- and postcontrast CT and angiography 0–24 days after the subarachnoid bleed. The interval between CT scan and angiogram was 1.8 ± 1.5 days. Contrast scanning was performed with an Ohio Nuclear Delta 2005 scanner 5 min after bolus injection of 42 g I.

Radiologic and clinical findings were reviewed with special attention to: (1) presence or absence of abnormal cisternal enhancement, (2) temporal relation of cisternal enhancement to time of initial bleed, (3) clinical grade at the time of initial scan, (4) presence or absence of arterial spasm, (5) presence or absence of hydrocephalus, (6) clinical course, and (7) location of bleed. Enhancement of the basal and sylvian cisterns, as well as the interhemispheric fissure and cortical sulci, was graded on a scale of 1 to 3. The clinical grade was assigned according to the classification of Hunt and Kosnik [8]. Arterial spasm was graded on a scale of 0 to 3 with *1* representing <50% reduction in vessel diameter; *2* 50%–75% reduction in vessel caliber; and *3*, >75% reduction.

Results

The results are summarized in table 1. Abnormal cisternal enhancement was detected in 21 of the 42 patients (fig. 1). It was observed 0–16 days after subarachnoid hemorrhage, but had no relation to the number of days after bleed that CT was performed (fig. 2). There was no significant difference in the clinical grade of patients with cisternal enhancement compared with those without. Neither the location of bleeding nor arterial spasm had significant correlation with enhancement ($P = 1$, chi square test). The 27 patients who showed no spasm had angiography at a mean of 2.4 ± 3.4 days after initial bleed. The 15 patients with spasm had angiography at a mean of 6.4 ± 6.1 days.

Hydrocephalus was observed in 11 of the 21 patients with cisternal enhancement. Five required lumboperitoneal shunts. Only five of the 21 patients without cisternal enhancement

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TABLE 1: **Cisternal Enhancement after Subarachnoid Hemorrhage**

Clinical Features	No. Patients		
	Cisternal Enhancement	No Cisternal Enhancement	Total
Initial clinical grade:			
I	4	8	12
II	7	4	11
III	9	7	16
IV	0	1	1
V	1	1	2
Subtotal	21	21	42
Degree of arterial spasm:			
No spasm	14	13	27
Mild	5	4	9
Moderate	1	2	3
Severe	1	2	3
Subtotal	21	21	42
Hydrocephalus:			
None	10	16	26
Mild	7	3	10
Moderate	4	2	6
Subtotal	21	21	42
Discharge grade:			
I	10	14	24
II	4	4	8
III–V	1	1	2
Died	6	2	8
Subtotal	21	21	42

developed hydrocephalus and none required shunting. Statistical analysis of correlation between cisternal enhancement and hydrocephalus showed a p value of 0.056 (chi square test).

There were six deaths in the group of 21 patients with cisternal enhancement. Three died from massive infarction presumably secondary to spasm, one from gastrointestinal hemorrhage, one secondary to a rebleed, and one at surgery. There were two deaths in the group of 21 patients with no cisternal enhancement. One died from progressively increasing intracranial pressure and the second from a postoperative hemorrhage. In those patients who survived,

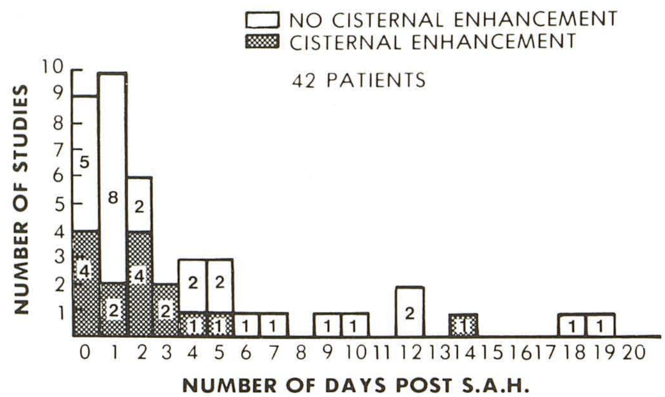

Fig. 1.—Temporal relation of cisternal enhancement and subarachnoid bleeding.

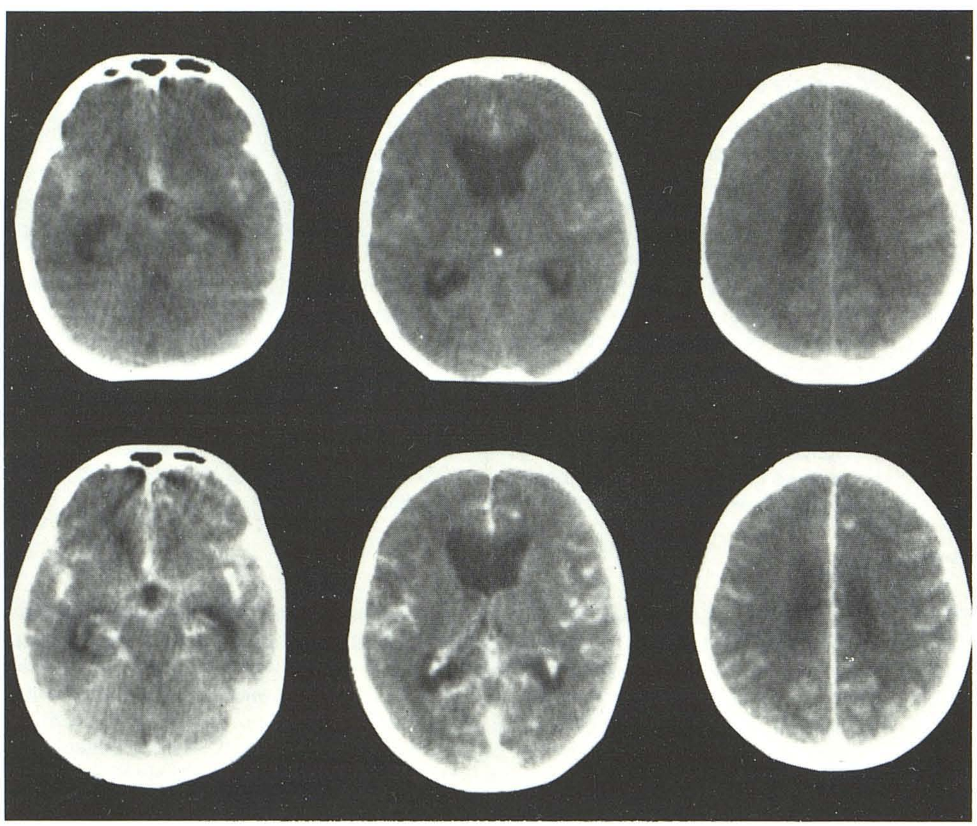

Fig. 2.—Cisternal enhancement 1 day after subarachnoid hemorrhage from anterior communicating artery aneurysm in a 55-year-old woman. Precontrast scans (*top*): blood in basal cisterns and cortical sulci with associated hydrocephalus. Postcontrast scans (*bottom*): abnormal enhancement of basal cisterns and cortical sulci.

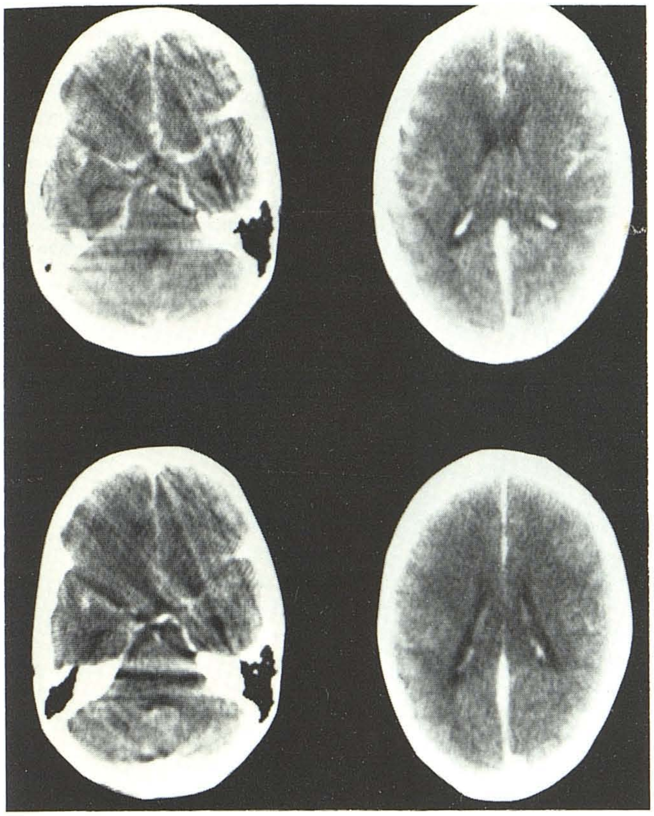

Fig. 3.—Cisternal enhancement after subarachnoid hemorrhage from an anterior communicating artery aneurysm in a 58-year-old woman. Postcontrast scans 1 day after bleed (*top*): abnormal enhancement of basal cisterns and cortical sulci. (Precontrast scan was normal.) Postinfusion scan at 7 days (*bottom*): resolution of cisternal and sulcal enhancement. Hydrocephalus is absent.

there was no significant difference in clinical grade between the two groups at the time of discharge.

A source of bleeding was identified in 36 of the 42 patients. Thirty-five patients had aneurysms and one had an arteriovenous malformation of the corpus callosum. In the other six patients, the cause of hemorrhage was unknown despite repeat angiography in three of the six.

Discussion

The pathogenesis of cisternal enhancement secondary to subarachnoid hemorrhage can probably be explained by two related mechanisms. An initial inflammatory response secondary to subarachnoid blood results in increased vascular permeability. Subsequently, an arachnoiditis similar to that seen in granulomatous arachnoiditis develops. The increased vascular permeability or the increased vascularity associated with arachnoiditis or both can account for increased levels of contrast material in the cisterns or adjacent arachnoid.

Edema of the arterial adventitia and media is seen within 24 hr after subarachnoid hemorrhage [9]. Necropsy series in patients with subarachnoid hemorrhage reveal inflammation, degeneration, and necrosis of the walls of both arteries and veins [10, 11]. These changes may be seen with [11] or without [10] associated spasm. Electron microscopic observations in experimentally induced subarachnoid hemorrhage in rhesus monkeys show abnormal corrugations of the internal elastic membrane as early as 8 hr and loss of endothelial tight junctions within 2–7 days [12]. These features are restricted to vessels exhibiting spasm. Early contrast enhancement of the basal cisterns in the first week after subarachnoid hemorrhage may reflect leakage due to increased vascular permeability secondary to such changes. However, our clinical data suggest that spasm is not a requisite for this increased permeability.

Leptomeningeal fibrosis is a well recognized sequela of subarachnoid hemorrhage. Hammes [13] observed this in over one-half of 53 patients surviving 10 days or longer. Others have described similar findings in conjunction with the development of hydrocephalus [14]. In experimental subjects, leptomeningeal fibrosis developed 10 days to several weeks after subarachnoid hemorrhage [15, 16]. Presumably, enhancement of cisterns developing more than 10 days after subarachnoid hemorrhage could be explained at least in part by the presence of leptomeningeal fibrosis.

We observed hydrocephalus in eight of the 25 patients scanned within 48 hr after subarachnoid hemorrhage. Six of these eight showed enhancement of the basal cisterns. Another four of the 25 patients scanned within 48 hr after hemorrhage showed cisternal enhancement without hydrocephalus. Davis et al. [17] found hydrocephalus in seven of 12 patients scanned within 48 hr after subarachnoid hemorrhage. The early development of hydrocephalus is attributed to clogging of the arachnoid villae by erythrocytes [18, 19]. Thus, initially, subarachnoid blood may cause increased vascular permeability resulting in cisternal enhancement, and it also may cause hydrocephalus secondary to clogging of the arachnoid villae. However, the two phenomena may occur independently (fig. 3). It might be expected that in severe bleeds, both would occur.

Chronic hydrocephalus cannot be explained on the basis of clogging of the arachnoid villae [19] but rather on the development of dense pial arachnoid adhesions in the basal cisterns with resultant impaired cerebrospinal fluid flow over the convexities [14] (fig. 4). In those patients with less dense or patchy pial arachnoid adhesions, hydrocephalus may not ensue. These pathologic findings may explain why hydrocephalus is more common in the presence of cisternal enhancement but is not always present.

To conclude, abnormal cisternal enhancement after subarachnoid hemorrhage was observed in one-half of 42 cases. Enhancement is associated with an increased occurrence of hydrocephalus, but does not appear to correlate with clinical grade, arterial spasm, temporal relation to bleed, or location of bleed. Although there was an increased number of deaths in the groups showing cisternal enhancement, the sampling group is too small to draw any valid conclusions.

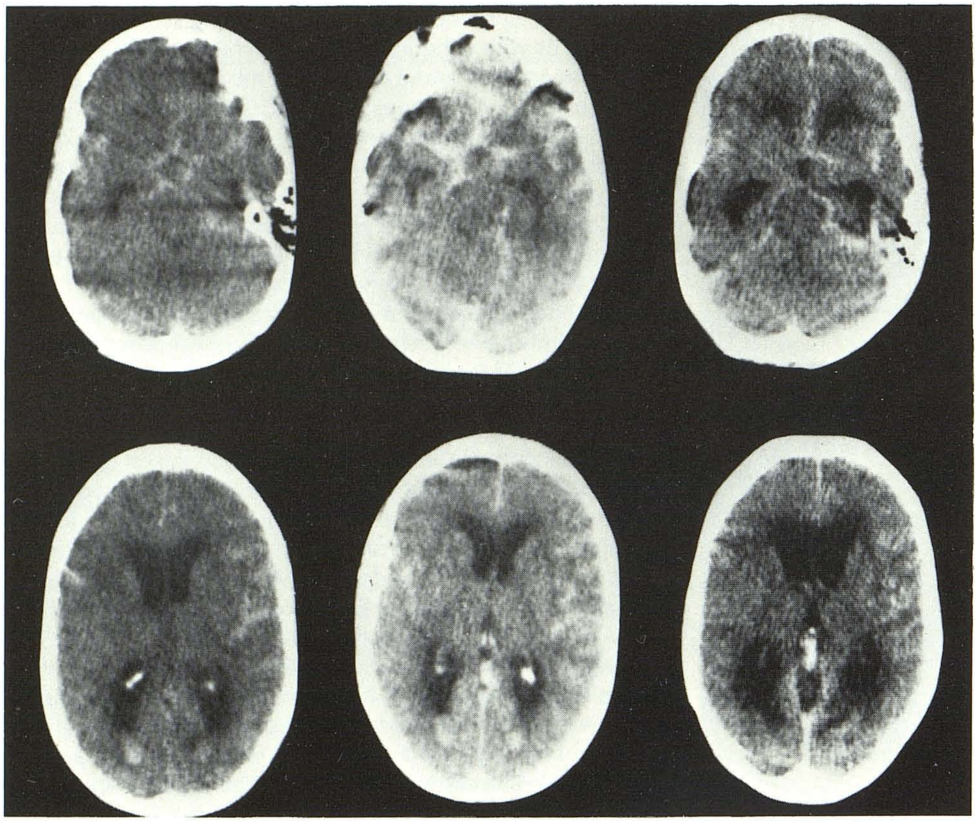

Fig. 4.—Cisternal enhancement after subarachnoid hemorrhage in a 71-year-old woman. No bleeding source was identified at angiography. Precontrast scans 5 days after subarachnoid hemorrhage (*left*): blood in basal cisterns and cortical sulci with associated hydrocephalus. Postcontrast scans (*center*): marked enhancement of basal cisterns and cortical sulci. Repeat postcontrast scans at 14 days (*right*): diminished cisternal enhancement, but increased hydrocephalus.

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