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Development of Aneurysm from Infundibulum of Posterior Communicating Artery with Documentation of Prior Hemorrhage

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The clinical significance of infundibular widening (junc­
tional dilatation) of the origin of an intracranial artery has
been a topic of debate; junctional dilatation is considered
preaneurysmal by some investigators and a normal variant
by others. Aneurysm formation or hemorrhage from an
infundibulum rarely has been reported. A patient is reported
in whom both aneurysm development at the infundibulum
and bleeding from this site are documented. This experience
suggests the possible value of repeat delayed angiography
when only junctional dilatation is identified in the face of
unexplained subarachnoid hemorrhage.

Case Report

A 26-year-old woman, 6 months pregnant with no signifi­
cant medical history, was discovered unconscious and brought to
the emergency room. She regained consciousness, but was confused
and lethargic, vomiting, and complaining of a severe headache.
Physical examination revealed neck stiffness without focal neuro­
logic deficits. Skull radiographs and CT scans were negative; spinal
tap was grossly bloody.

Four-vessel angiography with magnification and subtraction tech­
niques, performed later that day, revealed no evidence for aneu­
rysm, arteriovenous malformation, or vascular spasm. There was
junctional dilatation at the origin of both posterior communicating
arteries (fig. 1A). She was given aminocaproic acid (Amicar) for 1
week, treated symptomatically with analgesics, and discharged 27
days after admission.

After an uncomplicated term pregnancy and cesarian delivery,
the patient was readmitted for angiographic reevaluation. She was
asymptomatic, and neurologic examination was unremarkable.
Repeat four-vessel angiography demonstrated a widened infundibulum
on the left, with a small aneurysm arising eccentrically at the origin
of the posterior communicating artery (fig. 1B).

Elective left pterional craniotomy for clipping of the aneurysm
was performed 3 weeks after admission. The aneurysm was broad­
based and appeared not unlike a widened infundibulum, though
eccentrically located (fig. 2). Evidence of prior bleeding included a
large amount of adhesions in the area, with the left oculomotor
nerve densely adherent to the dome of the aneurysm. The aneurysm
was successfully clipped, and the postoperative course was un­
eventful.

Discussion

An infundibulum or junctional dilatation is a commonly
observed funnel-shaped outpouching at the origin of the
posterior communicating artery. According to Taveras and
Wood [1] 'the dilatation should be considered as a possible
aneurysm only when it measures more than 3 mm in diam­
er or when the posterior communicating artery does not
join the dilatation at its apex.'

Histologic reports concerning this entity are conflicting.
Hassler and Saltzman [2], finding media defects and occa­
sional defects in the internal elastic membranes of widened
infundibula, suggested that these appearances are identical
with those of a minute aneurysm. Epstein et al. [3], on the
other hand, finding normal vascular morphology in their
series of autopsy specimens, concluded that junctional
dilatation is a normal variation, and not a site having predilec­
tion to bleeding or future true aneurysmal dilatation.

Nonetheless, subsequent case reports have demonstrated
aneurysms of the posterior communicating artery
developing at the site of infundibular widening without rupture[4–6], and there is a recent documentation of a ruptured
infundibulum at autopsy [7]. In our patient, repeat panan­
giography almost 6 months after the initial bleeding dem­
onstrated the interval appearance of an aneurysm, which
was documented at surgery as a site of prior hemorrhage.
Thus, this case lends support to the view that infundibular
widening may be preaneurysmal, and illustrates the potential
value of repeat angiography under these circumstances.

The incidence of normal arteriography in patients with
subarachnoid hemorrhage has been reported fairly fre­
quently in the literature. West et al. [8] reported normal
cerebral arteriography in only 7% of 220 patients with
subarachnoid hemorrhage, comparing these results with the
significantly higher percentages in older reviews [18%–

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Fig. 1.—A. Initial angiographic evaluation, left internal carotid injection. Infundibular widening (2.4 mm), accounting for magnification. No demonstrable aneurysm. Reflux into tip of basilar artery (b). pc = posterior clinoid. B. Follow-up arteriogram 6 months later. Small aneurysm arises eccentrically at origin of left posterior communicating artery (arrow).

Fig. 2.—Representation of surgical findings: TL = temporal lobe; FL = frontal lobe; ON = optic nerve; tm = tentorial margin; om = oculomotor nerve; ICA = internal carotid artery; an = aneurysm; pca = posterior communicating artery; ac = anterior choroidal artery.

27%] and suggesting this decrease is principally due to technical improvements. Repeat angiography was performed on all normals in their study, but no mention was made of the percentage of initial false negatives. Abnormal cerebral arteriography would not be expected in instances of spinal arteriovenous malformation or tumor, rupture of a small superficial vessel, or other causes of subarachnoid hemorrhage [9]. Nonetheless, the greatest single cause of subarachnoid hemorrhage is rupture of aneurysm; the percentage of aneurysms in primary subarachnoid hemorrhage averaged 69% in one review [10].

Perret and Bull [11], correlating angiographic with autopsy findings, reported an 89% accuracy in the radiologic diagnosis of aneurysm, with a potential accuracy of 96% (assuming minimizing of observer error and attainment of adequate projections). In their series, only 2% of patients with intracranial aneurysms had normal arteriography when reviewed. Aside from observer error, inadequate technique, and arterial spasm, explanations to account for nonvisuali-

zation of aneurysms by arteriography include thrombosis [12] and self-destruction of small aneurysms at time of rupture [13].

Forster et al. [14] dealt directly with the value of repeat angiography, reporting a false-negative rate of less than 2% in a series of 56 patients, and suggested that repeat studies are seldom justified unless further bleeding episodes occur. The mean interval between the subarachnoid hemorrhage and the second angiographic study was 30 days. In their preliminary series of 529 patients with subarachnoid hemorrhage, initial panangiography was negative in 28%. Of the group selected for repeat evaluation, six of the 56 had widened infundibula, but no conclusions could be drawn about their significance.

To date, there is no accepted doctrine for further evaluation of unexplained subarachnoid hemorrhage by repeat angiography. Yet when this situation is encountered, angiography may reveal only infundibular widening. Generally in such instances, no serious consideration is given to surgical intervention, but for the neuroradiologist pressed for a definitive interpretation, the conflicting histologic reports do not ease his discomfort, and the 3 mm size criterion seems arbitrary and of little practical value. Our case illustrates that delayed repeat angiography, independent of rebleeding, may be warranted under these conditions, particularly in the young healthy patient with a long life expectancy.

The use of the term “delayed” is intentional; we wish to emphasize the interval of nearly 6 months between initial and repeat studies in this report. In previous reports of aneurysm development at the site of a widened infundibulum (without rupture), years elapsed in all instances before this development was documented angiographically [4–6].

In regard to the more general issue of repeat angiography in cases of unexplained subarachnoid hemorrhage, we await further long-range studies. Repeat angiography may prove advisable in the younger age group. For patients suffering a subarachnoid hemorrhage from aneurysm and surviving 6 months, rebleeding occurs on the average of 3.5% per year during the first decade, with a 67% mortality associated with a late rebleed [15].
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REFERENCES