# Successful management of an intracranial arteriovenous malformation by conventional irradiation

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 $\checkmark$  The reduction of an intracranial arteriovenous malformation (AVM) by conventional radiation therapy is described in a patient who refused surgery. The 2-year follow-up angiogram documented nearly complete obliteration of the nidus of the AVM, accompanied by progressive narrowing of the arteries supplying the lesion. The scanty literature dealing with this form of treatment is summarized.

KEY WORDS · arteriovenous malformation · radiotherapy · endarteritis

THE management of intracranial arteriovenous malformations (AVM's) includes direct surgical extirpation, embolization, and stereotaxic and other sophisticated forms of high-energy irradiation. On occasion, a combination of these various approaches may be advisable. However, some AVM's are unsuitable for any of these treatments, and some patients are unwilling to submit to aggressive management. In some of these cases, especially in situations involving medium-sized to large AVM's located in critical cortical areas, conventional radiation therapy has occasionally been reported to be of value.<sup>6,7,9,19</sup>

We describe the use of conventional radiation therapy delivered to the nidus of an AVM, and the dramatic response of the lesion over a 2-year follow-up period.

### **Case Report**

This 27-year-old man had complained of a dull headache for many years. He was first admitted in 1978 because of recent partial motor seizures involving his left extremities.

*Examination.* Physical and neurological examination were unremarkable. Computerized tomography scanning showed a right parietal lesion with significant enhancement after infusion of contrast medium. Right carotid angiography revealed a cortical-subcortical AVM of the right middle cerebral artery distribution. The malformation was  $6 \times 5 \times 5$  cm in size and was supplied mainly by two hypertrophic branches from the Sylvian group (Fig. 1). Drainage was provided by both the superficial and the deep cerebral venous system. *Clinical Course.* The patient refused any form of treatment and was simply followed as an outpatient during the ensuing 4 years. In spite of carefully monitored antiepileptic therapy, his seizures could not be controlled, and a trial course of radiotherapy of the malformation was finally accepted in early 1982. After control angiography had confirmed that the AVM was unchanged, 4500 rads was administered (150 rads for 5 days over 6 weeks) by conventional gamma irradiation. The radiation beam was centered over the nidus of the malformation. Both feeding and draining vessels were



FIG. 1. Angiogram showing the right parietal arteriovenous malformation before treatment, in 1982. Two hypertrophic branches from the middle cerebral artery supply the malformation. A severe steal phenomenon exists, resulting in lack of visualization of the anterior cerebral artery.

spared direct irradiation, although some radiation exposure could not be avoided.

Follow-up angiography revealed marked reduction of the nidus 1 year later, and the presence of only a minor residual lesion after 2 years in March, 1984 (Fig. 2). The progressive narrowing of the feeding arteries paralleled the obliteration of the AVM. During the 2 years since irradiation the headaches have progressively resolved and no further seizures have occurred. Antiepileptic drugs have therefore been discontinued.

#### Discussion

The history of conventional irradiation in the treatment of intracranial AVM's consists of only scattered reports, usually describing the ineffectiveness of this therapy. The first authoritative note was published in 1928, when Cushing and Bailey<sup>2</sup> remarked on the poor results of this procedure. Later, in 1957, Olivecrona and Ladenheim<sup>12</sup> criticized Potter's review<sup>16</sup> of the merits of radiotherapy, and confirmed that "radiation was tried on several inoperable lesions without the slightest discernible benefit" and cautioned "against the revival of this ineffectual form of therapy for arteriovenous malformations."<sup>12</sup>

Only anecdotal reports have appeared in the literature during the last four decades. Among them were four cases submitted to radiation procedures by Perret and Nishioka<sup>14</sup> in 1966, but no details about timing of irradiation or angiographic follow-up findings were given. In 1954, Krayenbühl<sup>9</sup> described the partial regression of a lesion after combined carotid artery ligation and irradiation. Svien and Peserico<sup>19</sup> in 1960 reported the complete angiographic obliteration of an AVM following repeated courses of irradiation.

Conventional irradiation as definitive treatment of cerebral AVM's is still generally criticized. The reasons

for its lack of support include the absence of evidence of any effect on the anatomic and angiographic evolution of these lesions, and previous angiographically documented reports of the spontaneous regression over time of AVM's, with partial<sup>10,13,18</sup> or even complete disappearance.<sup>1,4,5,11,13</sup> Indeed, thrombotic changes, particularly after bleeding episodes, are considered possible factors in AVM obliteration,<sup>1,5,10,13,19</sup> and also may account for the spontaneous healing of AVM's that have been submitted to irradiation.

Renewed interest in ordinary radiotherapy has recently been stimulated by the experience of Johnson.<sup>6,7</sup> He reported 100 patients with AVM's who were submitted to megavoltage irradiation, with the beam centered over the nidus of the AVM. A total dose of 4000 to 5000 rads was delivered over a 3-week period. Twenty of these patients were followed angiographically over 2 to 20 years: nine patients were "totally cured," five improved, five showed no change, and in one the lesion became larger.<sup>6</sup> These results were favorably recorded by Drake,<sup>3</sup> who wrote: ". . . it is the first time that long-term angiographic follow-up has been done to show a considerable effect from this standard therapy."

Unlike stereotaxic radiotherapy ("radiosurgery"), which uses multiple sharply collimated gamma-ray beams,<sup>17</sup> and Bragg-peak proton-beam therapy<sup>8</sup> (both techniques provide favorable results in the control of AVM's but are available in very few centers), the adoption of ordinary fractionated irradiation schedules is possible in all large hospitals. This is the reason why, during the past few years, we have recommended this treatment in selected cases, such as for inoperable AVM's in critical areas of the cortex, and, as in the case presented here, when the patient is unwilling to submit to surgery.

The mechanism of AVM obliteration seems to be



FIG. 2. Control angiograms. *Left:* One year after radiation treatment, in 1983, significant reduction of the nidus of the arteriovenous malformation is associated with narrowing of the feeding arteries. An early cortical vein draining into the transverse sinus is demonstrated. The steal effect seen in Fig. 1 has reversed, resulting in visualization of the entire cerebral circulation. *Right:* Two years after treatment, in 1984, subtotal obliteration of the malformation is seen, associated with further reduction in size of the feeding arteries. The early draining vein is still visible.

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endarteritis with progressive coarctation of vessels and late radiation necrosis.<sup>6</sup> In our case of an "intact" AVM in a young patient, we are inclined to think that irradiation played a role in the obliteration of the lesion similar to that seen as a result of bleeding<sup>1,5,10,13,19</sup> or arteriosclerosis.<sup>10,15</sup> This conclusion appears to be supported by the demonstrated stable size of the malformation during the 4 years before treatment.

The progressive decrease in size of the nidus of the AVM in our case was paralleled by significant homogeneous narrowing of the feeding arteries. Therefore, while the obliteration of the AVM is ascribable to endarteritis with occlusion and possible thrombosis of the lumina, the steady narrowing of the vessels associated with reversal of the steal effect and reappearance of the anterior cerebral artery circulation is directly related to the changing hemodynamic pattern of the AVM. Control angiograms have confirmed that hypertrophy and enlargement of vessels supplying AVM's are correlated to the blood flow; vessels increase in size to meet a higher demand and, inversely, decrease when restoration of normal flow patterns is established.

Another aspect of these cases concerns possible radiation damage with weakening of the vessel walls and increased risk of bleeding from the AVM, particularly during the short-term follow-up period. A hemorrhage would be especially distressing in patients who had not bled previously. This question will be resolved only by investigation of a large number of cases with a sufficiently long follow-up period.

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