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Radiation dermatitis after spinal arteriovenous malformation embolization: case report

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Abstract Few cases of radiation injury related to lengthy interventional neuroradiologic procedures have been reported, although concern has been heightened, as evidenced by a 1994 FDA Public Health Advisory. We report a case of radiation-induced dermatitis in a patient undergoing multiple diagnostic and embolization procedures for treatment of a spinal arteriovenous malformation.

Key words Radiation dermatitis · Embolization · Arteriovenous malformation, spinal

Introduction

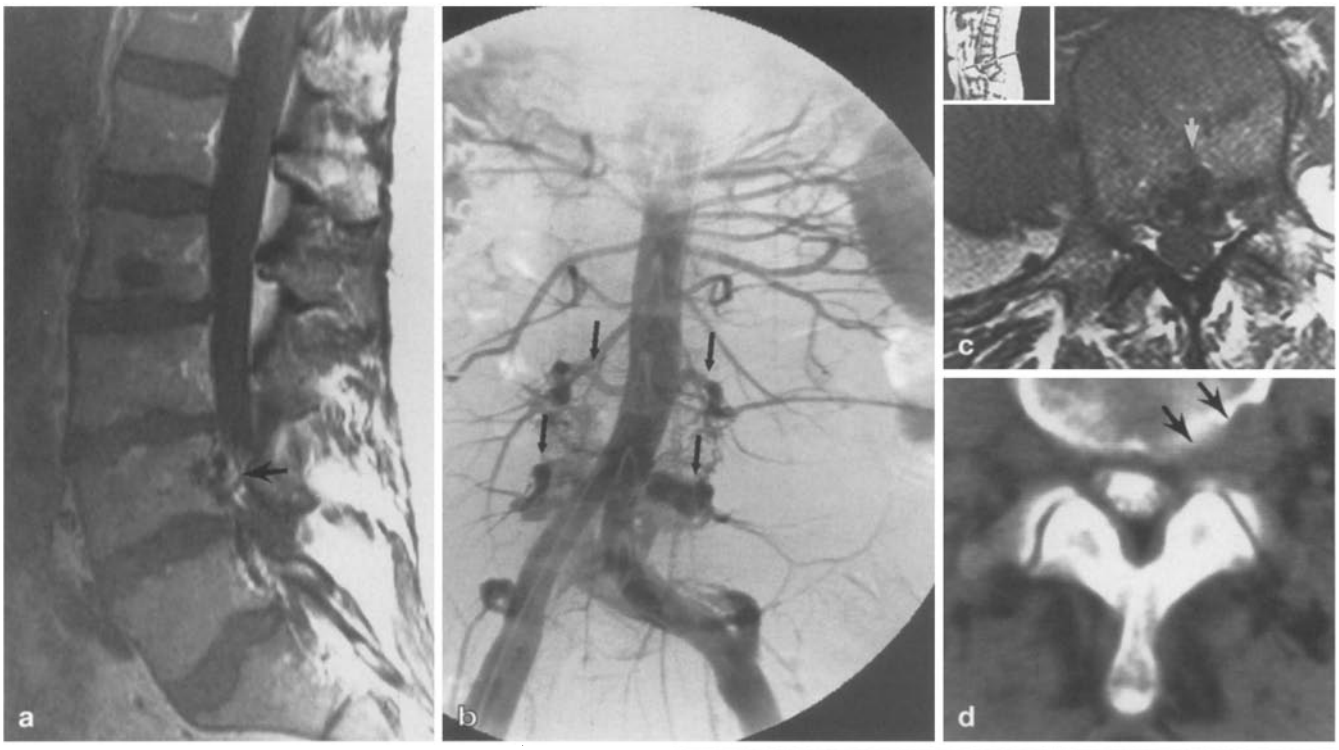
Interventional neuroradiologic procedures are being used to treat a number of neurovascular disorders, as primary therapy or a preoperative adjunct. As improvements occur in angiography systems, microcatheter technology, embolic materials and operator technique, these procedures will become more prevalent.

The often complex and labor-intensive nature of angiography required for superselective catheterization and embolization raises the issue of radiation exposure, a topic recently addressed by various groups [1–4]. Procedures are lengthy and multiple interventions are often necessary for more complex vascular malformations. Modern digital fluoroscopy, digital subtraction angiography, and high-dose fluoroscopy all contribute to increased radiation exposure for both patient and operator. Cumulative radiation dose during multiple interventional radiographic procedures is, therefore, an important issue which must be taken into consideration by the clinician. We report a case of radiation dermatitis after multiple interventional radiographic procedures.

Case report

A 38-year-old man complained of episodic bilateral pedal numbness and low back pain with radiation into the left leg. These symptoms were attributed to lumbar strain secondary to heavy lifting. He subsequently developed a crescendo pattern of bilateral gluteal and perirectal numbness. Examination demonstrated normal sphincter tone and subtle bilateral L5 weakness.

Routine MRI demonstrated mild disc bulging and multiple, variably-enhancing flow voids anterior to the duramater at lower lumbar levels, suspicious for an arteriovenous malformation (AVM). A number of these flow voids appeared to lie within and ventral to the posterior longitudinal ligament (Fig. 1 a). An aortogram followed by selective injections from T10 through L4 revealed an AVM fed bilaterally by the L3 and L4 lumbar arteries (Fig. 1 b). A large draining vein coursed inferiorly and to the left; its



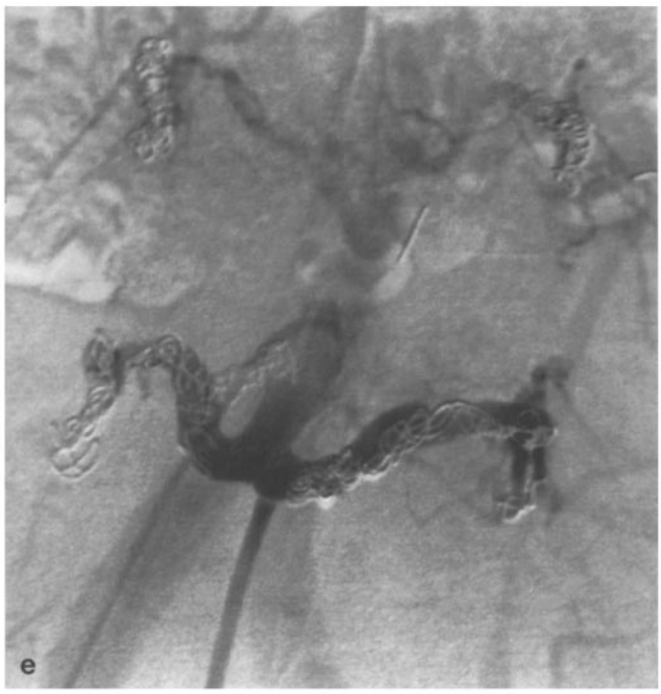


Fig. 1 a Sagittal MRI (TR/TE 600/15) demonstrating signal void at the dorsal surface of the L4 vertebral body probably representing vessels within the posterior longitudinal ligament *(arrow)*. **b** Anteroposterior aortogram demonstrating supply to the arteriovenous malformation from the L3 and L4 lumbar arteries *(arrows)*. **c** Axial MRI (TR/TE 600/15) demonstrating retroligamentous *(while arrow)* areas of signal void representing a vascular lesion. **d** Axial postmyelogram CT showing the vascular lesion's degree of mass effect and foraminal extension *(dark arrows)*. **e** Aortogram demonstrating the final appearance for the spinal arteriovenous malformation after particle and coil embolization of the L3 and L4 vascular pedicles

intraforaminal location was confirmed by both axial MRI and postmyelogram CT (Fig. 1 c, d), the latter demonstrating erosion of the vertebral body.

The patient underwent the first of three embolizations; at least three of the four feeding arteries were occluded. Two days after this procedure, the patient's symptoms resolved completely, but 2 weeks later the symptoms recurred and repeat arteriography demonstrated residual or recurrent arterial supply through each of the arteries described above. The L3 vascular pedicles were once again occluded by means of particulate and platinum microcoil embolization. Two weeks later again the patient returned for a third procedure, during which the L4 pedicles were embolized. No residual nidus or AV shunting was seen (Fig. 1 e). The patient tolerated these procedures well and, except for a 3×3 cm area of numbness on the sole of the left foot, made a complete neurologic recovery.

Four weeks later the patient noted a "rash over his low back" and was referred for dermatologic examination, which revealed a well-circumscribed erythematous, edematous rectangular plaque with several overlying necrotic papules (Fig. 2 a). This area corresponded to the collimated ports used for his radiologic studies. It was mildly tender, not pruritic and nondermatomal. A viral culture of a necrotic papule was negative. A 4-mm punch biopsy of the skin revealed necrosis of the epidermis with a few areas of regenerating epithelium beneath, associated with proliferation of small blood vessels (Fig. 2 b). Scattered lymphocytes and histiocytes were present in the dermis. A diagnosis of acute radiation dermatitis was made.

Over the next 4 weeks, the necrotic epidermis sloughed (Fig. 2 c), after which the epithelium regrew. Topical mupirocin and

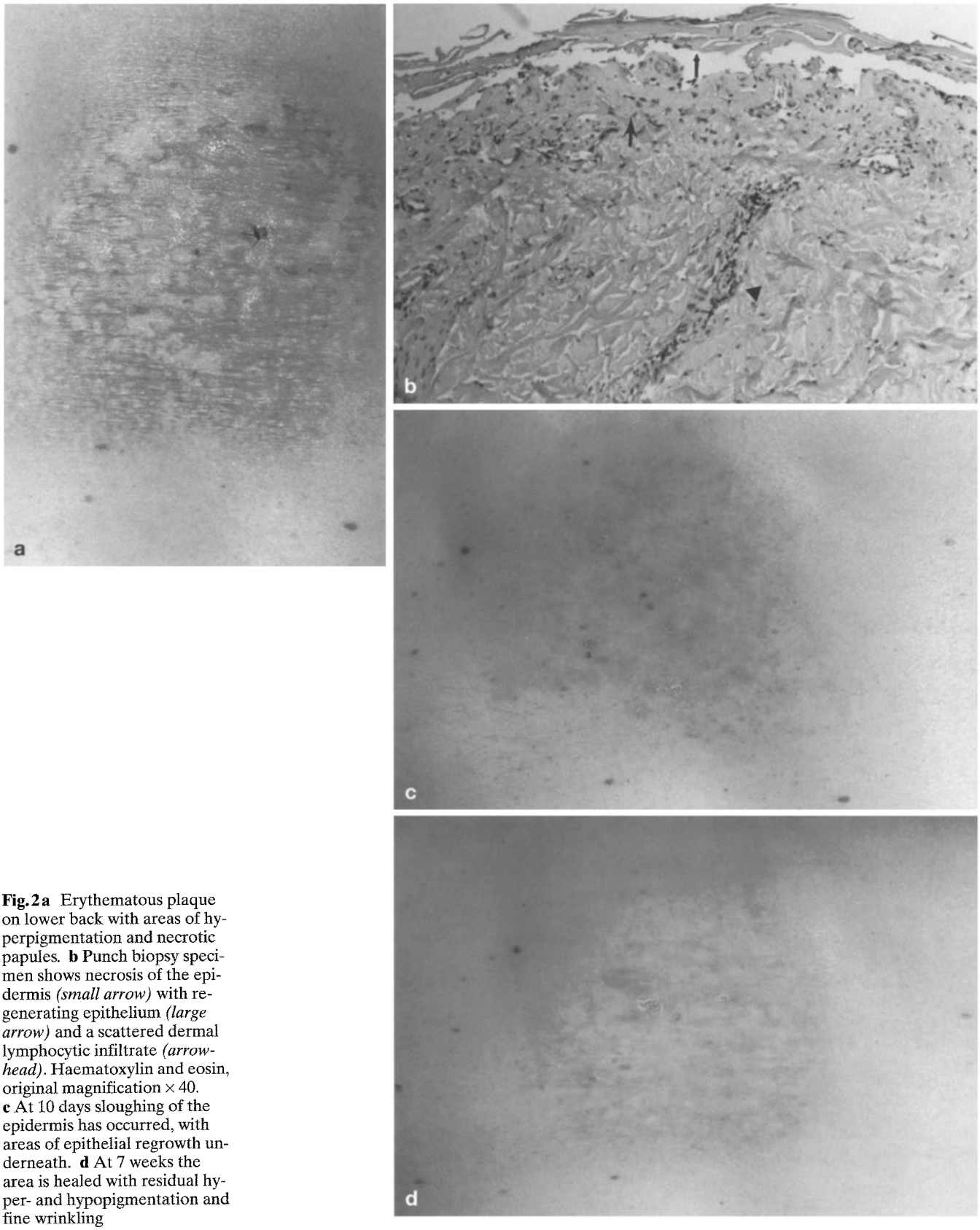


Fig. 2a Erythematous plaque on lower back with areas of hyperpigmentation and necrotic papules. **b** Punch biopsy specimen shows necrosis of the epidermis *(small arrow)* with regenerating epithelium *(large arrow)* and a scattered dermal lymphocytic infiltrate *(arrowhead)*. Haematoxylin and eosin, original magnification × 40. **c** At 10 days sloughing of the epidermis has occurred, with areas of epithelial regrowth underneath. **d** At 7 weeks the area is healed with residual hyper- and hypopigmentation and fine wrinkling

a hydrocolloid dressing were used to prevent infection. At a follow-up visit 7 weeks after presentation with the skin lesion, the skin was well healed with areas of hyper- and hypopigmentation, and fine superficial wrinkling (Fig. 2d). The pigmentary and textural changes indicated that the patient had a superficial second-degree burn.

Discussion

Radiation dose

Over a 4-month period, our patient underwent multiple procedures including a dedicated spinal arteriogram, a myelogram and postmyelogram CT of the lumbar spine and three embolizations before occlusion of his AVM was achieved. We thought it important to determine his total skin dose. Rather than using dosimeters, we employed technique data including estimates of fluoroscopy time, the number of angiographic runs per procedure and the number of exposures per run at a given frame rate. Further, the kVp, mA and mAs for each run were recorded for all angiography, plain film myelography and CT examinations. Our calculations suggested the maximum entrance skin dose to be 2470 cGy. The skin dose delivered for each procedure was a follows: first arteriogram plus myelogram/CT 190 cGy; second arteriogram/embolization 980 cGy; third arteriogram/embolization 560 cGy; fourth arteriogram/embolization 740 cGy. The accumulated dose of 2470 cGy was delivered over a 4-month period. Our patient's skin changes occurred after his last study, which delivered 740 cGy. His last three studies were separated by 12 days each and over this period he received 2280 cGy. This was preceded by 190 cGy given in two stages separated by 12 days. Every attempt was made to minimize patient exposure. The field of view was minimized by using optimal collimation. In fact, the rectangular configuration of the erythema corresponded to the configuration of the collimators. Filtration was maximized to increase beam hardening, thus reducing skin exposure, and fluoroscopy and run times were kept as brief as possible.

Radiation injury may be stochastic or deterministic. In the former, which is dose and not dose-threshold related, radiation-induced malignancies and genetic mutations may be seen; in the latter, the severity of the effect is directly related to the radiation dose with a threshold value present. While dermatitis alone is deterministic, later related malignancy would be considered stochastic.

Absorbed radiation dose may be measured in grays (Gy) or centigrays (cGy); 1 Gy = 1 joule/kg. This is a measure of the energy absorbed per unit mass of radiated tissue and depends upon the energy source and the distance from the source to the tissue [5]. When multiple doses or treatments are administered, predicting the biologic effect of the absorbed energy becomes difficult because of a dose-time relationship [6].

The capacity of irradiated tissue to repair itself depends upon the ability of remaining cells to divide and repopulate an area. As cell destruction exceedes cell production, tissue necrosis or ulceration occurs. With minor degrees of radiation (1–600 cGy) no skin changes are seen except for temporary epilation. At doses of 600 cGy the skin may appear red and dry and may be tender to touch and firm due to underlying edema [6]. With higher doses (1500–2000 cGy) moist desquamation, dermal necrosis, and secondary ulceration may become apparent, yet complete or incomplete healing generally occurs within days to weeks [6, 7].

The time course for skin changes is usually as follows. Following skin exposure in excess of 600 cGy the epidermis may become reddened, simulating a sunburn. Erythema usually increases for 24 h and subsides by day 4. A second more pronounced episode of erythema may develop 10 days following therapy and last 2–4 days. Occasionally, a third phase is seen 6–7 weeks after therapy which lasts 2–3 weeks. Radiation-induced erythema may be prolonged and delayed when fractionated doses are used. Blistering and ulceration occur following exposure to several thousand centigrays. Transient epilation occurs after several hundred centigrays and permanent epilation occurs with more than 700 cGy. Twelve days following this skin dose, dark hyperpigmentation may develop at the periphery and hypopigmentation near the center. Pigmentary changes may be present indefinitely. Chronic radiation dermatitis is characterized by dyspigmentation, atrophy, telangiectasies, and induration. This is a precancerous condition which may develop malignancies at a later date, most commonly squamous, basal cell and spindle cell carcinomas [8].

The fact that skin biopsy revealed only epidermal and superficial dermal injury is a good prognostic sign for our patient. As in all cases of chronic radiation dermatitis, this area will need to be monitored in the future for the development of cutaneous malignancies. Scar formation at the site cannot be fully assessed due to the short follow-up period. He will probably have mild atrophy and dyspigmentation as a long-term result of the radiation injury.

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BOOK REVIEW

Advances and Technical Standards in Neurosurgery. Wien: Springer 1995. XV, 381 pp, 149 Figs., (ISBN 3-211-82634-3), DM 298,–.

This book, sponsored by the European Association of Neurosurgical Societies, is one of a series published since 1974. In the first section of the text the aim is to present fields of neurosurgery and related areas in which important recent advances have been made. The topics covered include the classification and molecular biology of pituitary adenomas, the biomechanics and classification of traumatic lesions of the spine, and space-occupying lesions of the sensory-motor region. In the second section a novel feature is the description of standard operative procedures covering the surgery of cavernomas (cavernous angiomas), both supra- and infratentorial, gliomas and other mass lesions of the brain stem, and hearing preservation in acoustic tumour surgery.

The chapter on traumatic lesions of the spine provides a detailed description of biomechanics and classification of traumatic lesions and with its extensive bibliography provides a valuable reference chapter for the neuroradiologist.

In the chapter on space-occupying lesions of the sensory-motor cortex, features of most interest to the neuroradiologist will be the description of the surgical anatomy of this region. Successful surgery of the sensory-motor region requires precise pre- and intraoperative localisation of the sensory-motor region and pyramidal tract. MRI provides superb details of the sulcal anatomy of the sensory-motor cortex.

There is a detailed section on neuroimaging of cavernomas (cavernous angiomas). Once thought to be rare, these are being more frequently demonstrated by CT and to a greater extent MRI. Their natural history is not always benign due, to haemorrhage, the anatomical position of the lesion and epileptogenic potential. The vast majority of cavernomas are accessible surgically.

In the section on surgery for gliomas and other mass lesions of the brain stem, the authors stress the value of MRI in differential diagnosis and, in the case of tumours, distinguishing a focal from a diffuse, infiltrating lesion. They advocate ablative surgery when neuroimaging suggests a focal noninvasive tumour and report that such tumours can be removed completely or almost so with acceptably low mortality and morbidity.

Neuroradiologists are unlikely to buy this book, but it should be available in most neuroscience libraries and they will benefit from browsing through it to see the value of neuroimaging from a neurosurgical prospective.

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