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Radiologic Evaluation of Neurosarcoidosis: Role of Computed Tomography

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Radiologic evaluation of 15 patients with central nervous system sarcoidosis is reported together with a review of the literature. The hydrocephalus that is often associated is discussed in relation to known pathogenetic aspects of the disease process. Intracranial sarcoid mass lesions, formerly thought to be a very rare manifestation of this disease, have been seen more often since the advent of computed tomography. Computed tomography may contribute to a more prompt recognition of the diagnosis and help to avoid the considerable morbidity that can occur in some patients with progression of the disease within the central nervous system.

Two previous studies [1, 2] and several case reports [3–11] have described computed tomographic (CT) findings in a total of 28 cases of cranial neurosarcoidosis. Sarcoid involvement of the spinal cord is rare and has been discussed primarily in isolated case reports [12–21]. We report our experience with 15 more patients with sarcoidosis involving the central nervous system (CNS), illustrating some of the pertinent radiographic abnormalities with particular emphasis on the contribution of CT in the evaluation of this disease.

Materials and Methods

Clinical and radiologic findings are summarized in table 1. Twelve of our patients had intracranial disease, three had spinal cord or cauda equina lesions without demonstrated intracranial abnormality, and one (case 2) with intracranial disease also developed subsequent spinal involvement.

Results and Discussion

Sarcoidosis is a systemic granulomatous disease of unknown etiology with an epidemiologic predilection for the southeastern part of the United States [22]. The case material for this report was collected from five contiguous states in the southeastern region (South Carolina, Georgia, Alabama, Mississippi, and Louisiana). CNS involvement in sarcoidosis has been reported to occur in up to 16% of cases at autopsy [23–25], and neurologic symptoms are clinically present in 3%–9% of patients with known disease [25–31].

Cranial CT

The clinical manifestations of CNS sarcoid are protean, and findings on the cranial CT scan may similarly include a wide spectrum of abnormalities. Table 2 summarizes previous CT findings in cranial neurosarcoidosis and compares them with our cases.

CT findings in previous cases [1–11] indicated hydrocephalus, either focal or generalized, was the most common CT abnormality. Intracranial sarcoidosis is

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TABLE 1: **Neurosarcoidosis: Clinical Summary and Radiographic Evaluation**

Case No. (age, race, gender)	Clinical Features	Other System Involvement	Tissue Diagnosis	CT Findings	Other Radiography
1 (27, BM)	Ataxia, vomiting, change in mental status	Pulmonary	Intracranial bx of L cerebellar tonsil & meninges	Hydrocephalus	Skull: normal
2 (24, BM)	1st adm: olfactory aura, seizure, normal exam	Pulmonary, ocular	Bronchoscopic bx	Mildly dilated 4th vent; large L temporal mass low attenuation; massive dilatation L temporal horn; no abnormal enhancement	Skull: normal; angiogram: avascular L temporal lobe mass
	2d adm: severe headaches	. . .	. . .	Increased hydrocephalus	PEG: adhesions anterior to pons and at tentorium w/nonfilling of ventricular system; ventriculogram: 3d vent mass; obstruction of trigone
	3d adm: dyspnea	. . .	. . .	. . .	. . .
	4th adm: diplopia, vertigo, ataxia, limb weakness	. . .	. . .	Subependymal enhancement of frontal horns	Myelogram: multiple filling defects in cervical subarachnoid space
3 (32, WM)	1st adm: skin rash, transient visual field loss, normal exam	Pulmonary, cutaneous	Skin & Bronchoscopic bx	Multiple intraventricular enhancing lesions	Skull: normal
	2d adm, 6 mo: steroid therapy, diplopia; nystagmus, R 6th nerve paresis	. . .	Bx intraventricular mass	. . .	. . .
4 (31, BM)	6 mo progressive mental deterioration, staggering gait, generalized weakness, headaches, bilateral 6th nerve palsy, R peripheral 7th nerve palsy, bilateral hypotonic DTRs	None	Intracranial bx of mass	Mass at foramen of Monro & suprasellar; moderately dilated lateral vent	PEG: suprasellar mass indenting L lateral vent, amputation of ant 3d vent; skull: enlarged sella w/ small suprasellar calcification; angiogram: normal; CXR: normal
	. . .	. . .	. . .	F/U scan: resolution of mass w/ increase in supersellar calcification over 2 yr interval on steroid rx	
5 (17, WF)	Galactorrhea, diabetes insipidus, lethargy, sleepiness, confusion, disoriented in place & time	Pulmonary	Transbronchial & liver bx	Small, round enhancing suprasellar mass mimicking colloid cyst	Skull: normal; CXR: diffuse interstitial infiltrate
6 (29, BF)	Episodic deja vu phenomenon; olfactory aura, nausea, confusion; 2° amenorrhea; normal exam	Pulmonary normal endocrine evaluation	Scalene node & mediastinal bx	High attenuation enhancing hypothalamic mass extending into temporal lobes	Skull: normal; CXR: bilateral hilar & paratracheal lymphadenopathy
7 (58, WF)	3 wk progressive L leg weakness; L facial weakness; mild L hemiparesis; mild central facial paresis; mild decreased hearing L ear	None	Intracranial bx	High attenuation R basal ganglion mass w/ enhancement	Skull: normal; angiogram: avascular mass; CXR: normal
8 (32, BM)	Low back and L hip pain; weakness & numbness L leg; ataxia & mental confusion; L central 7th nerve palsy; L hemiparesis; increased DTRs L leg; ataxic gait	Pulmonary cutaneous	Mediastinoscopy w/ bx; skin, liver, muscle bx	Normal precontrast; enhancing lesions basal ganglia, paraventricular, corpus callosum	Skull: normal; myelogram: normal Pantopaque; CXR: mediastinal widening

	...	...	...	F/U 4 yr (post-corticocosteroid rx): normal	...
9 (37, BF)	1 mo H/O memory loss, R arm & leg numbness and weakness; loss of bladder control; receptive and expressive aphasia; R hypalgesia & hemiparesis; R facial weakness	Pulmonary, cutaneous, 14 yr duration	Intracranial bx: L caudate septum pellucidum mass, L lateral vent ependymal wall & corpus callosum	Moderate hydrocephalus; more dilatation of L lateral vent; high attenuation enhancing mass of corpus callosum extending to foramen of Monro	Skull: normal; angiogram: avascular L temporoparietal mass: CXR: bilateral hilar & paratracheal lymphadenopathy
10 (15, BM)	1st adm: progressive visual acuity of 3 mo duration; visual fields grossly intact; Marcus Gunn pupil on R w/ papilledema	None	Intracranial bx of suprasellar mass w/ ant cranial fossa extension & optic chiasm compression	Initial cranial & orbital CT w contrast: neg; 1 mo later: enhancing sellar & suprasellar mass extending to anterior fossa	PEG: mass in prechiasmatic region; skull: enlarged sella w/ erosion at base of dorsum; angiogram: elevation of A-1 segment of L ant cerebral artery suggestive of hypovascular suprasellar mass; slight posterior displacement of basilar artery & stretching of thalamoperforator branches; CXR: normal
	2d adm 1 mo later: Marcus Gunn pupils bilaterally; hand motions visible R eye; blind L eye	...	...	...	...
11 (37, BF)	4 yr H/O headaches & bilateral upper eyelid swelling; 3 yr H/O anosmia; 2 yr H/O visual blurring; 1 yr H/O of amaurosis L eye; several mo H/O visual blurring R eye, worsening R sided headaches; Marcus Gunn pupil on L; decreased sensation R 5th nerve	None	Bx upper canthus R eye; Bx epidural, subdural, & R optic nerve sheath masses & dura	Sphenoid hyperostosis & dural enhancement	PEG: ventricles upper limits of normal; normal optic & infundibular recesses: skull: hyperostosis of wings of sphenoid bone, sphenoid body, & pterygoid plates; hyperostosis of orbital plates of frontal bone; angiogram: normal; CXR: normal
12 (61, WF)	Skin rash, dizziness, vertigo, nausea, vomiting, erythematous pruritic plaquelike lesions over L tibia & forehead	Osseous, cutaneous	Calvarial & skin bx	...	Skull: multiple calvarial osteolytic lesions
13 (39, WF)	Pain & weakness both legs of 1 yr duration; paresthesias R foot; 9 kg weight loss; decreased R leg strength & sensation; decreased DTRs both legs	Pulmonary	Scalene node & bronchoscopic bx	...	Myelogram: neg Pantopaque; CXR: bilateral fibronodular interstitial changes
14 (19, BM)	1st adm: 1 wk H/O tingling both feet; 4 d H/O weakness both legs until unable to walk day of admission; hypalgesia both legs w/ T4 sensory levels; marked decrease motor strength both legs; decreased DTRs both legs	None	...	...	Skull: normal; myelogram: cervical & upper thoracic cord widening
	2d adm: ascending paralysis w/ quadriplegia of 5 d duration, respiratory difficulty coughing & handling secretioins of 1 d duration; hypalgesia w/ C4	Pulmonary	Spinal cord bx	...	...

TABLE 1: continued

Case No. (age, race, gender)	Clinical Features	Other System Involvement	Tissue Diagnosis	CT Findings	Other Radiography
	sensory level; paraplegia & minimal arm movement; S1; increased arm DTRs; decreased leg DTRs; breathing primarily with accessory muscles of respiration				
	3 yr F/U: developed c/o headaches, blurred vision R eye, feeling "sleepy" & dizziness; T9 sensory level, motor strength 2/5 biceps, 4/5 triceps, 4/5 intrinsic hand muscles, 0–1/5 leg w/ hyperactive leg DTRs	. . .	. . .	. . .	. . .
15 (22, BM)	Cramping pains in thighs for 18 mo w/ 2 wk exacerbation; weakness of both legs; absent leg DTRs; atrophy of quadriceps bilaterally & R ant tibial muscles; weakness of dorsiflexors both feet	Pulmonary	Scalene node bx	. . .	Myelogram: irregular beaded appearance of cauda equina w/ partial block at L3 (Pantopaque); normal thoracic & cervical cords; CXR: bilateral interstitial reticulonodular infiltrate

Note—bx = biopsy, L = left, adm = admission, vent = ventricle, PEG = pneumoencephalography, w/ = with; mo = month; DTR = deep tendon reflex; ant = anterior; CXR = chest film; F/U = follow-up; yr = year; rx = therapy; wk = week; H/O = history of; w/o = without; neg = negative; d = day.

TABLE 2: **CT Findings of Intracranial Sarcoidosis**

CT Finding	Reference Nos.			Our Series
	[1]	[2]	[3–11]	
Normal	3	0	0	0
Hydrocephalus (only initial finding)	7	3	1	2
Single intracerebral mass lesion with or without hydrocephalus	2	1	4	6
Multiple basal and/or intracerebral enhancing lesion	1	2	3	2
Extraaxial mass (anterior cranial fossa, orbits)	0	0	1	2
Totals	13	6	9	10

Fig. 1.—Case 1. Extraventricular obstructive hydrocephalus due to posterior fossa sarcoid granuloma. Low transverse axial CT section shows moderate dilatation of fourth ventricle.

well known to cause a granulomatous leptomeningitis with a predilection for involvement at the skull base and especially the hypothalamic region, floor of the third ventricle, pituitary infundibulum, and optic chiasm [24, 33–37]. Fibrosis with adhesions may produce intraventricular obstruction or, more often, extraventricular block, especially at the level of the foramina of the fourth ventricle [38] and basal cisterns (fig. 1).

Intraventricular obstruction was present in case 2 (fig. 2), who demonstrated the interesting finding of left temporal horn entrapment consequent to adhesions at the left trigone. Pneumoencephalography also demonstrated extraventricular obstruction due to adhesions in the pontine cistern and at the tentorial level. The patient also developed frontal horn subependymal enhancement on a CT scan 1 year after the initial CT evaluation and a shunting procedure (fig. 2D). This patient was very similar to a case of Ho et al. [8], in which a radioisotope delayed scan had also been obtained and was consistent with an ependymitis as well. Although the radiologic differential diagnosis for the CT finding of subependymal enhancement includes a wide spectrum of infectious and neoplastic etiologies in addition to sarcoidosis,

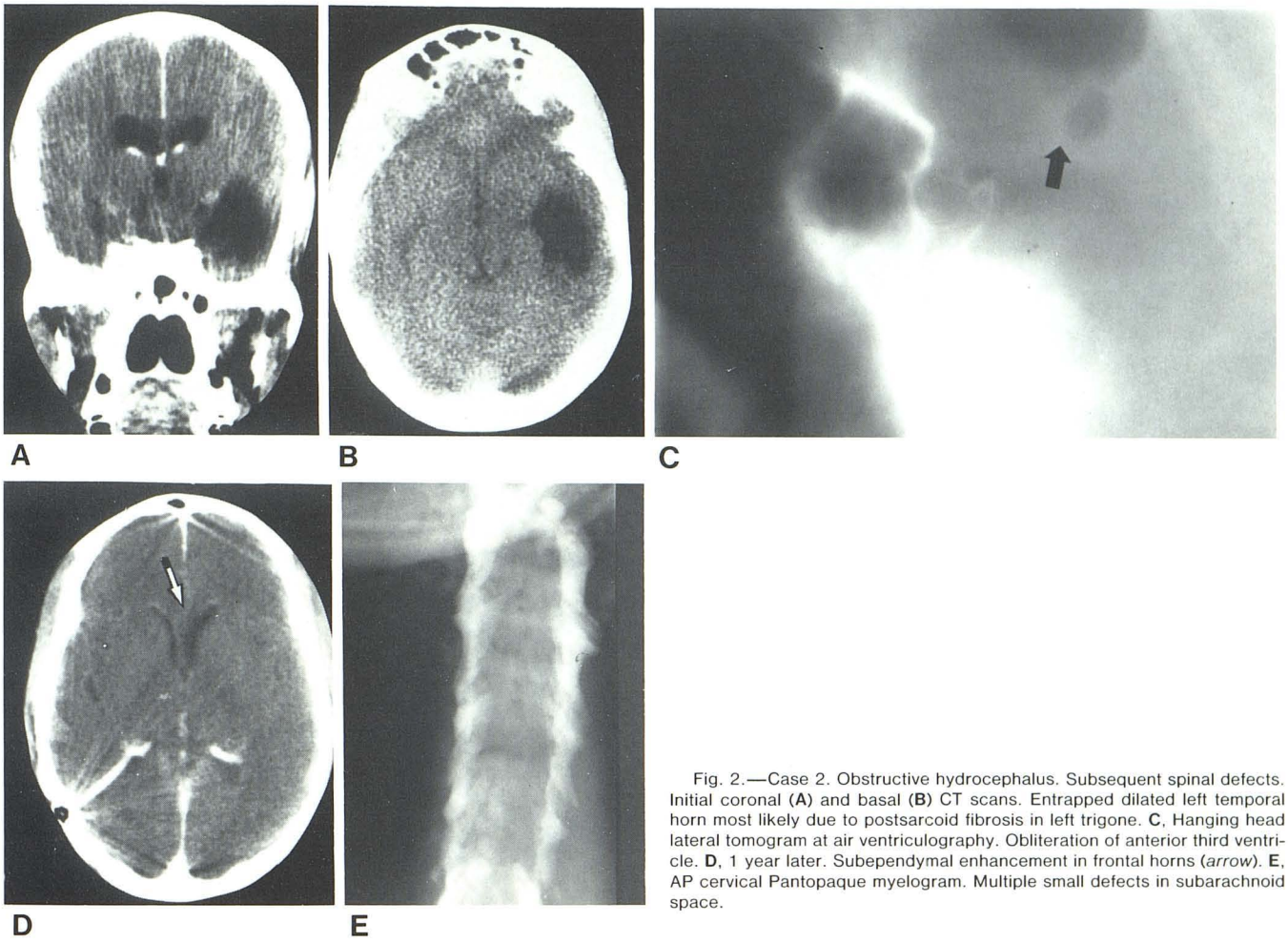


Fig. 2.—Case 2. Obstructive hydrocephalus. Subsequent spinal defects. Initial coronal (**A**) and basal (**B**) CT scans. Entrapped dilated left temporal horn most likely due to postsarcoid fibrosis in left trigone. **C**, Hanging head lateral tomogram at air ventriculography. Obliteration of anterior third ventricle. **D**, 1 year later. Subependymal enhancement in frontal horns (*arrow*). **E**, AP cervical Pantopaque myelogram. Multiple small defects in subarachnoid space.

this finding may still be helpful in the appropriate clinical setting. The same CT appearance may be seen in bacterial, fungal, and tuberculous meningitis as well as with subependymal seeding of primary intracerebral tumors [40–43].

Case 3 (fig. 3) is an example of hydrocephalus consequent to both intra- and extraventricular obstruction in a patient with multiple enhancing intraventricular masses. The literature has included pathologic descriptions of intraventricular sarcoid masses, but no CT findings. In 1958, Saltzman [39] described five patients with cerebral sarcoidosis, one of whom had a large intraventricular sarcoid granuloma that filled the right temporal horn similar to case 3. Our patient had enhancing lesions near the foramen of Monro, within the right frontal horn and in the left trigone, causing dilatation of the lateral ventricles, and extraventricular obstruction with dilatation of the fourth ventricle as well. The lateral ventricle dilatation was more pronounced on the left side with location of the left trigone mass probably within the choroid plexus. Postcicatrization of this lesion could result in focal hydrocephalus with entrapment of the left temporal horn similar to case 2.

Basal cistern enhancement due to granulomatous basal arachnoiditis was described by Enzmann et al. [44] in two cases of fungal and one tuberculous meningitis. As might

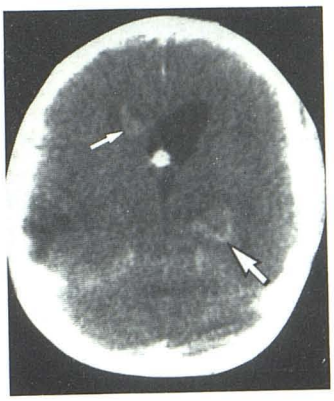
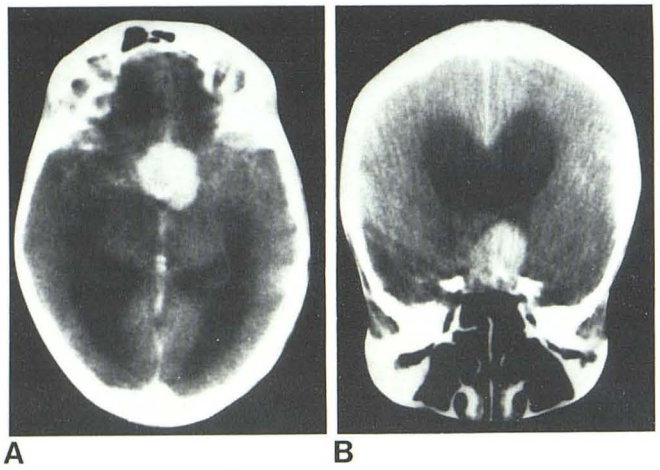


Fig. 3.—Case 3. Intraventricular sarcoid granulomas with hydrocephalus. Enhanced scan. Ventricular shunt and multiple intraventricular enhancing lesions (*arrows*). Foramen of Monro lesion (not shown) probably caused hydrocephalus.

be expected, basal arachnoiditis due to sarcoid may produce similar findings. In some patients who are scanned before the development of arachnoidal adhesive changes, CT may demonstrate only basal enhancing lesions due to the granulomatous leptomeningitis. This was noted in one of the 10 patients reported by Kendall and Tatler [1] and also in one of the six patients in the series reported by Bahr et al. [2]. Similarly, multiple areas of cortical enhancement on CT have been reported [1, 2, 10] due to coalescence of



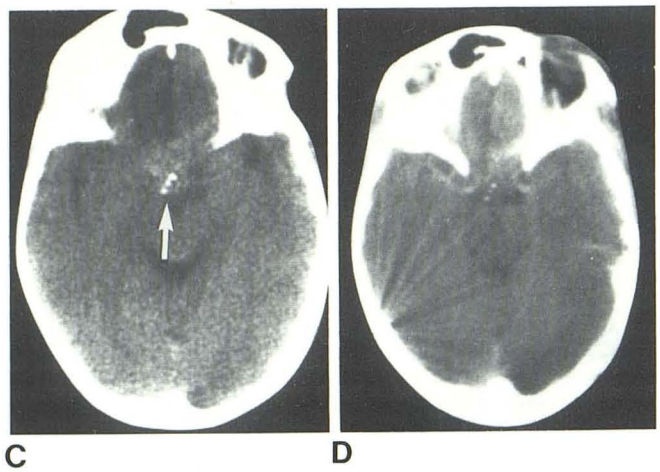


Fig. 4.—Case 4. Suprasellar mass obstructing foramen of Monro regressing with steroids and calcifying. **A**, Basal scan. Large, well defined suprasellar mass. **B**, Coronal section. Mass produces intraventricular obstructive hydrocephalus. **C**, Follow-up unenhanced scan. Suprasellar calcifications (*arrow*). **D**, Enhanced scan no longer shows abnormal mass.

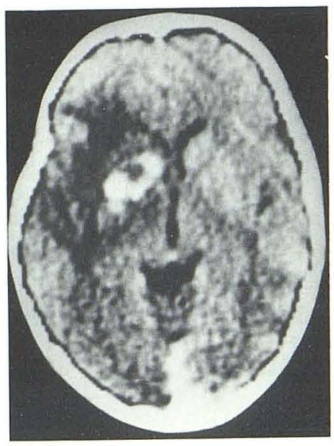


Fig. 5.—Case 7. Enhancing mass in right basal ganglia.

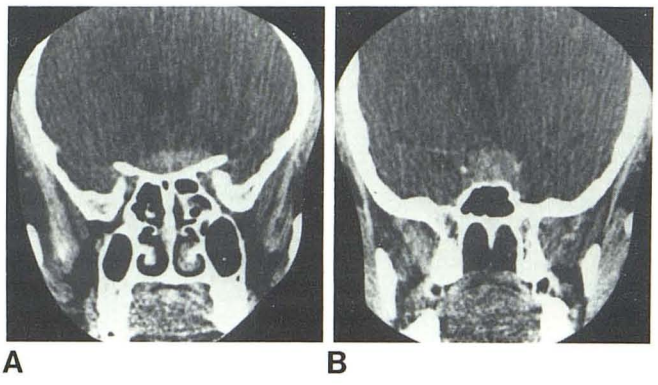


Fig. 6.—Case 10. Extraaxial, anterior fossa, and suprasellar sarcoid. **A**, Anterior fossa plaque enhancement. **B**, Enhancing lesion extends to suprasellar region.

sarcoid granulomas in the cortex and/or adjacent meninges. Other patients may demonstrate both meningeal enhancement and hydrocephalus, which, as Kendall and Tatler [1] have postulated, may be indicative of ongoing active inflammatory meningitis concurrent with end-stage fibrotic adhesive changes in other sites producing hydrocephalus.

Before the advent of CT, sarcoid mass lesions were generally regarded as the least common presentation of intracranial involvement [25]. However, as is apparent from the tabulation of CT findings (table 2), the incidence of mass lesions is much higher. These mass lesions have all shown higher attenuation than normal brain parenchyma on nonenhanced scans and homogeneous enhancement after administration of contrast material. In our series, mass lesions were present in 10 of the 12 patients with intracranial disease and were the most common CT abnormality (figs. 3–7). Suprasellar location was most common while the two intracerebral masses reported by Kendall and Tatler [1] were both frontal lobe lesions.

Two extraaxial masses were present in our series (figs. 6 and 7). One of these (case 11) had sarcoid masses involving the optic nerve sheath with sub- and epidural extension and evidence of bony involvement of the skull base. In the other patient (case 10), a prechiasmal mass extended into the anterior cranial fossa. A subfrontal extraparenchymal sarcoid mass was reported recently that mimicked a cribiform plate meningioma on CT and angiography [9]. A pre–CT era case reported by Goodman and Margulies [45] had a similar subfrontal sarcoid mass that appeared like a meningioma at the time of surgery.

CT has also proven to be a very useful noninvasive means of following response to treatment with corticosteroids. Kendall and Tatler [1] illustrated regression of a frontal lobe mass on follow-up CT over a 9 month interval, and a case report by Brooks et al. [6] also documented similar evolution on follow-up CT scans, as did two of six cases reported by Bahr et al. [2]. In our case 4, progressive decrease in size of a foramen of Monro mass was followed by CT over a 2 year period (fig. 4C) while the patient was receiving steroids. Case 8 had complete resolution of the CT abnormalities on

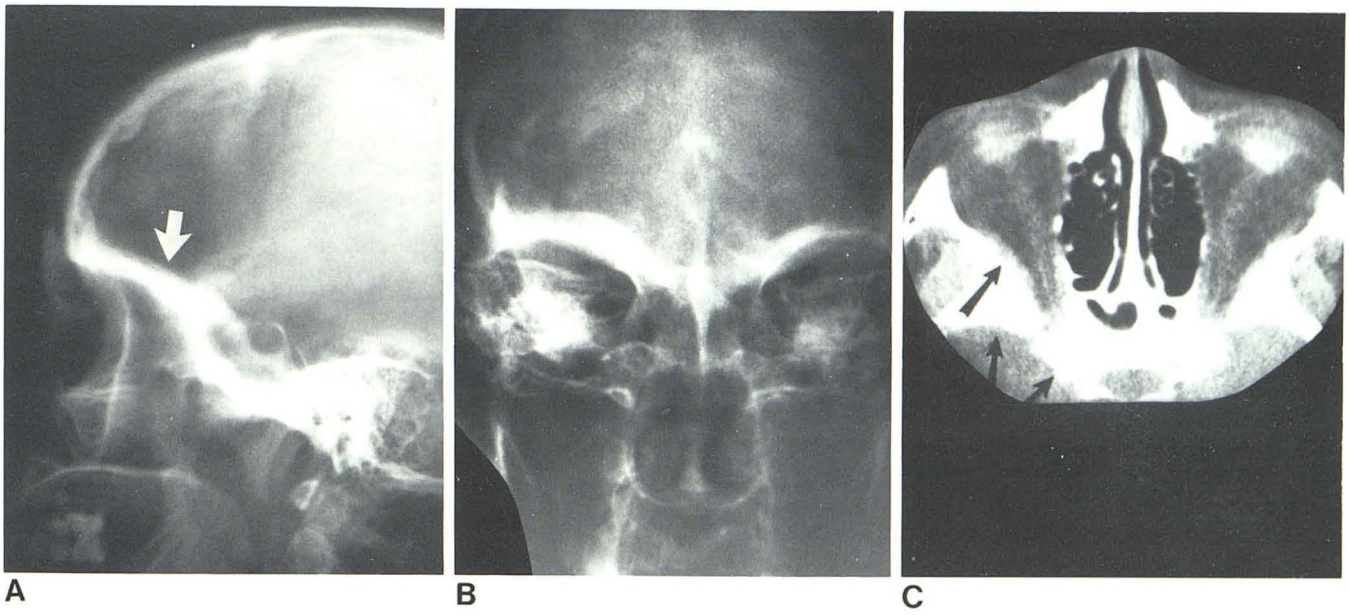


Fig. 7.—Case 11. Sarcoid in skull. **A** and **B**, Sclerosis of right orbital plate of frontal and sphenoid bones. **C**, CT scan after contrast enhancement. Bone thickening and paraosseous plaque enhancement in right middle cranial fossa and right orbit (*arrows*), probably due to involvement of dura and periosteum.

a 4 year follow-up scan after the patient had received corticosteroid therapy.

Follow-up CT may be useful when there is a strong clinical suspicion of intracranial sarcoidosis after a negative initial CT study. Our case 10 had initially normal noncontrast and contrast CT examinations and developed rapid progression of visual loss over a 1 month period. Repeat CT with the addition of coronal sections then disclosed an enhancing sellar and suprasellar mass. Sarcoid granulomatous tissue compressing the optic chiasm and extending into the anterior cranial fossa was verified at surgery.

Skull Radiography

Osseous lesions in sarcoidosis occur in about 2%–4% of patients [45]. Most of these are in the short tubular bones of the hands and feet, with the classical finding being that of a lacy reticulated trabecular pattern with small punched out lytic lesions [46].

In the skull and facial bones, destructive lesions of the nasal bones and maxillary antra have been reported, as has unilateral and bilateral optic foramen enlargement due to sarcoid masses involving the optic nerve sheaths [47–49]. A destructive lesion of the occipital bone was reported by Kendall and Tatler [1]. About 20 skull lesions have been described as well defined lytic defects, usually with no marginal sclerosis, due to sarcoid granulomatous involvement of the diploic space [50]. Radiologic differential diagnosis includes consideration of eosinophilic granuloma, multiple myeloma, and lytic lesions due to metastatic disease.

Four of our 15 patients had positive skull findings. Two had sellar enlargement, one with associated suprasellar calcification. Case 12 (fig. 8) had multiple lytic lesions associated with skin lesions and was similar to other reported cases. Reactive bone formation due to sarcoidosis is even more unusual [45] and case 11 (fig. 7) demonstrated this interesting finding in the orbital plates and sphenoid body, wings, and pterygoid plates. This occurred in association with encasement of the optic nerves and sarcoid infiltration of the epidural and subdural spaces which was verified at surgery. Overall, in the radiologic evaluation of patients with suspected neurosarcoidosis, skull radiography appears warranted primarily in patients with cutaneous lesions of the face or scalp or with intraorbital or nasopharyngeal involvement.

Angiography

Although CNS sarcoid is known to cause a granulomatous angiitis and periangiitis [34, 51] pathologically, only one case of angiographically illustrated sarcoid angiitis has been reported previously [52]. In our series, angiographic evaluation was noncontributory except to exclude vascular lesions.

Myelography

There have been a number of reports of sarcoid involvement within the spinal canal demonstrated by myelography [12–15, 18–20]. The findings have been nonspecific, including either a partial or complete obstruction, an intramedullary, intradural-extramedullary, or extradural mass, or evidence of an arachnoiditis. Our case 13 had strong clinical and laboratory evidence of sarcoid involvement of the cauda equina but a normal Pantopaque myelogram. Similarly, Kendall and Tatler [1] reproted normal myelograms in four of six

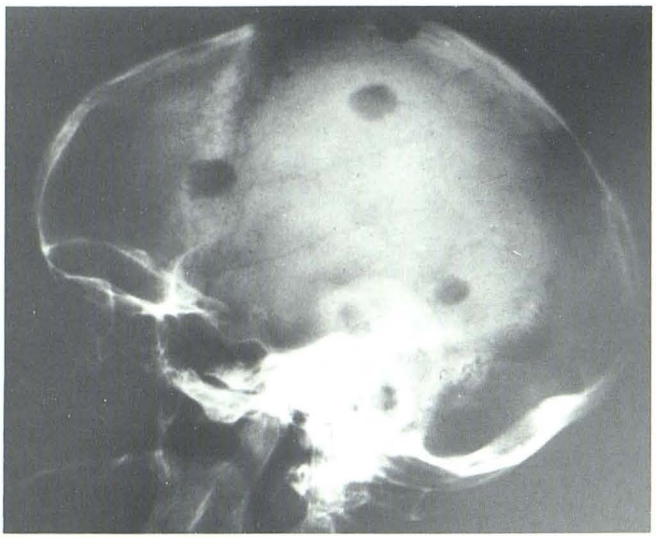
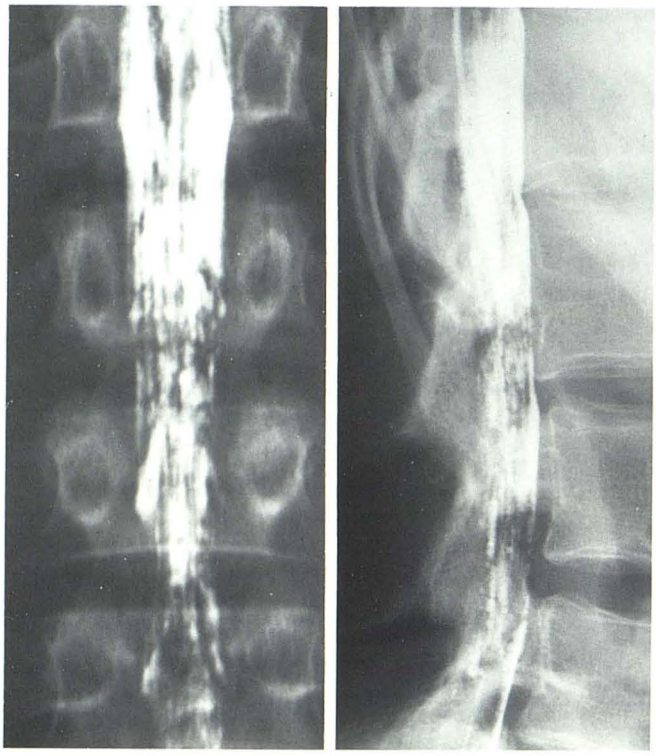


Fig. 8.—Case 12. Multiple large lytic lesions due to sarcoid.



A B

Fig. 9.—Case 15. Sarcoidosis of cauda equina. Pantopaque myelogram. Irregular defects and obliteration of subarachnoid space with partial block. Pantopaque ran freely showing normal dorsal and cervical regions.

patients; one patient had sacral nerve root involvement and five had cord lesions. However, Pantopaque myelography in our case 15 demonstrated the interesting finding of multiple beaded lesions involving the roots of the cauda equina (fig. 9) due to sarcoid granulomatous involvement.

Myelography was performed in case 2 (fig. 2E) 1 year after diagnosis of intra- and extraventricular obstructive hydrocephalus because the patient developed new complaints of generalized motor weakness and ataxia. Multiple filling defects in the cervical subarachnoid space consistent with sarcoid granulomas were demonstrated. The cerebrospinal fluid protein at the time was 1,753 mg/ml and concurrent CT demonstrated the new finding of frontal horn subependymal enhancement.

Postmyelography CT is useful in the evaluation of lesions that cause cord widening, such as syringomyelia and arteriovenous malformation. In patients with suspected spinal canal sarcoidosis, metrizamide may be the preferable contrast medium, both because of better demonstration of possible granulomatous involvement within the subarachnoid space and the added value of a CT scan, which can be obtained subsequently.

Summary

Cranial CT in patients with suspected neurosarcoidosis may reveal only nonspecific findings of hydrocephalus, basal cistern enhancement or multiple small cortical or basal enhancing lesions, or it may demonstrate intracranial masses in the region of the optic chiasm, pituitary infundibulum, hypothalamus, and floor of the third ventricle. Such mass lesions due to sarcoidosis have been diagnosed with greater frequency since the advent of CT. CT has also been shown to be very useful in the follow-up of patients with CNS sarcoidosis with steroid treatment both for intracranial and intraspinal disease. In the evaluation of patients with suspected intraspinal involvement, myelography with a water-soluble contrast material is preferred, both for the better visualization of sarcoid granulomatous involvement of nerve roots and subarachnoid space and for the added value of a postmyelography CT scan.

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