

Neurosarcoid Presents Differently in Children Than in Adults

Robert J. Baumann, MD, and William C. Robertson, Jr, MD

ABSTRACT. *Background.* Neurosarcoid is seldom recognized in children. In the absence of any large pediatric series, it has been assumed that the presenting signs and symptoms are identical in adults and children.

Objective. To test the hypothesis that childhood neurosarcoid differs in presenting signs and symptoms from neurosarcoid in adults.

Methods. We tabulated the initial neurologic signs and symptoms in all reported cases of childhood sarcoid with evidence of central nervous system involvement. These data then were compared with published studies of adult neurosarcoid.

Results. Twenty-nine cases (from the English, French, and German literature) had descriptions of presenting signs and symptoms. Ages were 3 months to 18 years; 48% (14 of 29) presented before 13 years. Seizures were the most common presenting symptom (38%, 11 of 29), and 73% of these children (8 of 11) were <13 years old at presentation. Twenty-one percent (6 of 29) had cranial nerve involvement at presentation, and all were ≥ 12 years old. Twenty-one percent (6 of 29) had hypothalamic dysfunction. Five children presented with headache, 4 with motor signs, and 3 with papilledema. Twenty-four percent (7 of 29) had mass lesions on imaging.

Conclusions. Children with neurosarcoid present differently than do adults. Children are more likely to have seizures, less likely to have cranial nerve palsies, and perhaps more likely to have a space-occupying lesion. Our analysis of the cases available for review in the published literature suggests that children evolve to an adult pattern as they progress through adolescence. *Pediatrics* 2003;112:e480–e486. URL: <http://www.pediatrics.org/cgi/content/full/112/6/e480>; *adolescence, child, cranial nerve injuries, manifestations neurologic, sarcoidosis, seizures.*

ABBREVIATIONS. CNS, central nervous system; CSF, cerebrospinal fluid; MRI, magnetic resonance imaging; ACE, serum angiotensin-converting enzyme; CT, computed tomography.

Physicians seldom include sarcoid in the differential diagnosis of progressive encephalopathies of childhood.^{1,2} This apparently occurs because sarcoid is seldom diagnosed in the young and is therefore considered rare.^{3,4} The omission is surprising, because sarcoid is both treatable and known to have protean manifestations. Moreover, if

untreated, CNS sarcoid can result in death or permanent disability.

In the absence of any large pediatric series, it has been assumed that the presenting signs and symptoms of CNS sarcoid are identical in children and adults.^{3,5} We recently cared for a child who did not fit the usual adult pattern and decided to reexamine this premise. We searched for all reported cases in the world literature and analyzed them for presenting signs and symptoms. Our review suggests that prepubertal children with neurosarcoid present differently than adults, and this pediatric pattern evolves to an adult pattern during puberty.

CASE REPORT

After 3 days of headache and malaise, a previously healthy 9-year-old white girl from rural Kentucky had a generalized tonic-clonic seizure. Cerebrospinal fluid (CSF) examination revealed a white blood cell count of 350 per mL (52% lymphocytes and 48% polymorphonuclear cells), normal glucose, and protein of 68 mg per dL. Routine cultures were sterile. After intravenous phenytoin there were no additional seizures. Her hospital course was characterized by fever to 101°F, confusion, and headache. Electroencephalograms showed symmetrical slowing compatible with a nonspecific, diffuse encephalopathy. Precontrast magnetic resonance imaging (MRI) of the head was unremarkable, but gadolinium-enhanced images revealed 2 irregular areas of enhancement involving the anterior aspect of the left frontal lobe. After 10 days she was afebrile, symptom-free, and discharged on phenytoin with a presumptive diagnosis of viral encephalitis.

After 3 medically uneventful months (though marred by poor school performance), the child became acutely confused and had another generalized tonic-clonic seizure. On readmission, routine laboratory studies were unremarkable. Gadolinium-enhanced MRI showed diffuse, fine punctuate lesions with increased signal from cortical gyri on the late T2 sequence. Magnetic resonance angiogram was unremarkable. The erythrocyte sedimentation rate was 11, and the phenytoin level was therapeutic. Numerous laboratory studies including serum aldolase, antineutrophil cytoplasmic and perinuclear antibodies, antinuclear antibodies, anti-Smith antibodies, factor V Leiden, anti-smooth muscle antibodies, and anti-cardiolipin antibodies were negative. Homocysteine, protein C, and protein S were unremarkable. Spinal fluid analysis revealed 4 mononuclear cells, 1 red blood cell, glucose of 64 mg per dL, and protein of 53 mg per dL. CSF cultures including those for fungi,

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tuberculosis, and viruses were sterile. Other CSF studies including venereal disease research laboratory test, pyruvate, oligoclonal bands, and myelin basic protein were unremarkable. Valproic acid was added to the patient's medication program. She improved and was discharged after 6 days of hospitalization.

After discharge the patient had headaches daily. Over the next 3 weeks she became unsteady and had difficulty remembering numbers. After 2 generalized seizures the child was readmitted. Examination revealed confusion, a right 6th-nerve palsy, extensor plantar responses, and truncal ataxia. Multiple laboratory studies again were normal. Brain MRI now showed large areas of increased T2 signal outlining multiple, swollen cerebral cortical gyri and the caudate nuclei (Fig 1). The dot-like enhancement previously seen with gadolinium enhancement was more prominent and again corresponded to areas of increased T2 signal (Fig 2).

The child's headache was unrelenting. She developed bilateral 6th-nerve palsies, left lower facial weakness, and slow speech. Brain and meningeal biopsy revealed discrete noncaseating epithelioid granulomas consistent with sarcoid (Fig 3). The serum angiotensin-converting enzyme (ACE) level was 15 international units (normal: 41–126), and chest radiograph was normal.

The child had marked clinical improvement within 48 hours of high-dose steroid therapy. After 5 months of tapering doses of prednisone, MRI findings had completely resolved, and steroids were discontinued. There were no cranial nerve findings or focal neurologic deficits; however, the child did have frontal lobe indifference and mild cognitive difficulties. Four years later these cognitive deficits have persisted, but there has been no recurrence of sei-

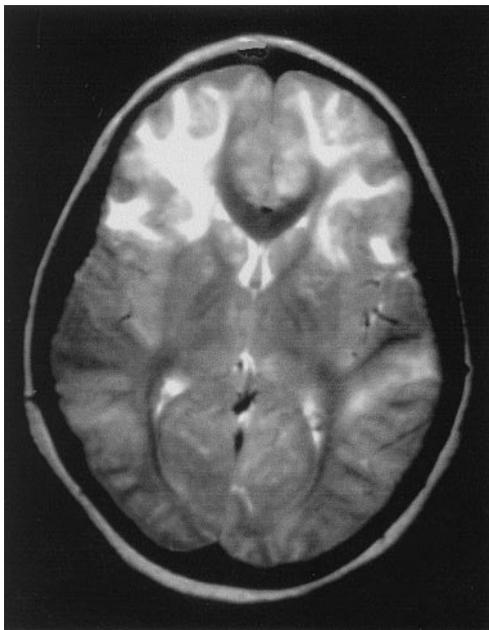


Fig 1. Axial T2-weighted image demonstrates large areas of increased signal involving primarily frontal cortex and subcortical white matter.

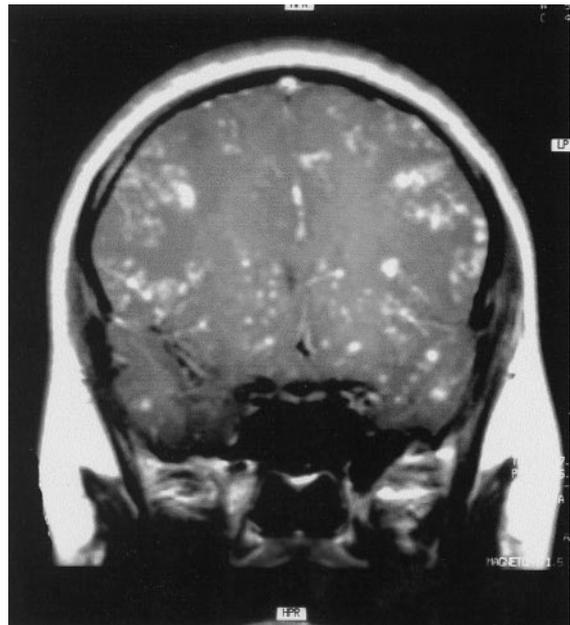


Fig 2. Coronal MRI with gadolinium shows enhancing punctate lesions within sulci and cortical gray matter.

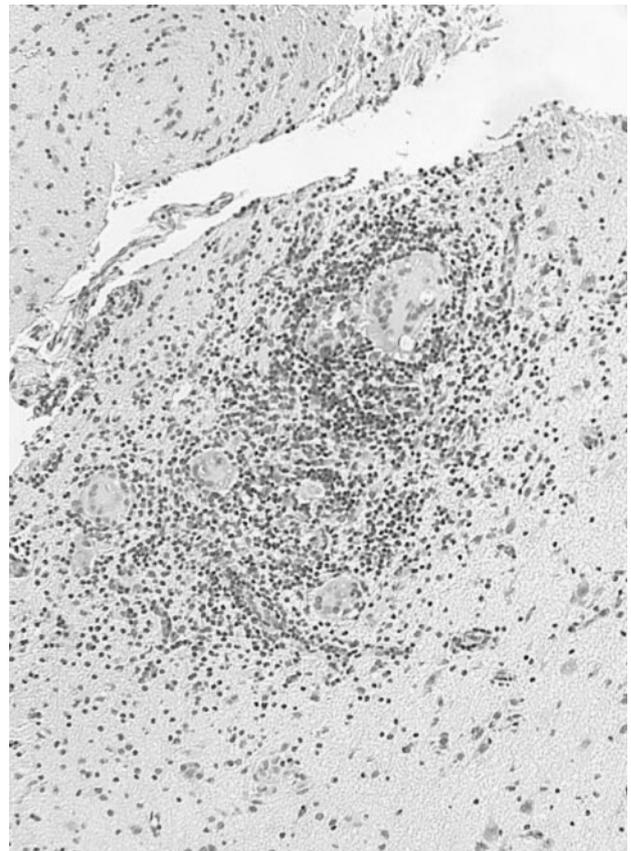


Fig 3. Biopsy specimen shows noncaseating granuloma with multinucleated giant cells in the frontal cortex (hematoxylin/eosin, original magnification $\times 100$).

zures or focal neurologic signs and repeat MRI studies have been normal.

METHODS

We attempted to identify all reported childhood cases of neurosarcoid. We searched PubMed for the common manifestations of

sarcoid in childhood (pulmonary and arthritic) as well as for neurosarcoid. Each reported case was examined for evidence of CNS manifestations, and reference lists were scanned for reports of additional cases. We also reviewed child neurology and neuroradiology textbooks for cases described in the texts but not reported otherwise.

RESULTS

Including our patient, there were 29 cases of childhood neurosarcoid with descriptions of presenting signs and symptoms (Table 1). An additional 15 cases lacked sufficient data about presenting signs and symptoms and were omitted. Because on rereview of the original articles we could not find evidence of neurologic involvement, we omitted 2 of the 23 children listed by Weinberg et al⁶ in their 1983 review of pediatric neurosarcoid. In the paper by Beier and Lahey⁷ we could not find the child with a 7th-nerve palsy referenced by Weinberg et al. In addition, in the 1956 series collected by McGovern and Merritt⁸ we were unable to identify the child tabulated by Weinberg et al as having "facial dysphagia."

Cases were found in the English, French, and German language literature. Only 13 cases were specifically published in peer-reviewed journals as neurosarcoid. Eleven other children were reported as having systemic sarcoid, but our review of the published details indicated nervous system involvement. Two other cases were reported as examples of hypothalamic complications of sarcoid. Another was found in an article on the radiologic manifestations of sarcoid, 1 was reported as an ophthalmologic complication of sarcoid, and the final case was discovered in a child neurology textbook. Ages ranged from 3 months to 18 years with 48% (14/29) presenting before 13 years. Seizures were the most common presenting symptom (38% of subjects, 11 of 29). Of the children presenting with seizures, 73% (8 of 11) were <13 years old at presentation. Twenty-one percent (6 of 29) had cranial nerve findings at presentation that appeared to be caused by direct nerve involvement and not as a secondary effect such as 6th-nerve palsies associated with increased intracranial pressure. All children with cranial nerve palsies were ≥12 years old. Only 1 child had involvement of multiple cranial nerves (age 13). Among the others, 2 had optic nerve and facial nerve palsies, and 1 had a superior oblique palsy. Twenty-one percent (6 of 29) evidenced hypothalamic dysfunction with diabetes insipidus (4 of 6), short stature (3 of 6), and sexual immaturity (2 of 6). Hypothalamic dysfunction showed no age predilection. Five children presented with headache, 4 with motor signs, and 3 with papilledema.

Twenty-four percent (7 of 29) had mass lesions or focal edema on imaging: One 8-year-old child with seizures had a "ring cortical lesion" on computed tomography (CT)⁹; our case (age 9) had papilledema and swollen frontal gyri on MRI; one 12-year-old child had nodular lesions in the optic chiasm and right sylvian fissure on MRI¹⁰; a 13-year-old child had a pontine mass on MRI¹¹; a 16-year-old child had hemiparesis and a left frontal mass¹²; a 17-year-old child with seizures had a temporal mass on MRI¹³;

and an 18-year-old child had hemiparesis and papilledema with a mass on angiogram.¹⁴

DISCUSSION

Our patient presented with an encephalopathy that lacked unique or distinguishing features, similar to many of the cases of childhood neurosarcoid reported in the literature. She lacked evidence of pulmonary sarcoid, had no adenopathy, and had a normal ACE level. Neurosarcoid was not considered in the differential diagnosis, although there were hints as to the correct diagnosis on MRI (punctate, enhancing lesions with gadolinium). She developed signs of increased intracranial pressure (papilledema and bilateral 6th-nerve palsies), leading to a biopsy of the mass-like, intracranial lesion and the correct diagnosis. Her response to corticosteroid therapy was prompt and probably life-saving. She has been off medication and in remission for >4 years.

Sarcoid is a systemic disorder of unknown cause characterized by noncaseating granulomas.¹⁵ It is well known in adults for its protean features including abnormal chest radiographs and progressive multiorgan failure.¹⁵ In adults, "the lungs and thoracic lymph nodes are almost always involved; most patients report acute or insidious respiratory problems."¹⁵ Neuropathy, especially facial nerve palsy, is the most common neurologic complication among adults.¹⁶⁻¹⁹ Among older children sarcoid is primarily viewed as a pulmonary or ocular disorder,¹⁰ with the triad of skin, joint, and eye disease occurring in younger children.²⁰⁻²² In younger children, systemic sarcoid differs clinically from the adult form of the disease and can mimic various disorders unique to childhood.²³ Adult and childhood sarcoid also differ epidemiologically. Children do not appear to show the female predominance noted in adults²³; additionally, in the United States, they do not seem to demonstrate the higher prevalence in African Americans seen in adult populations.^{15,22} Although our patient lives in a rural area, it is unknown if American children as a group show the rural predominance noted in American adults with this disorder.^{15,24}

In adults, neurosarcoid is estimated to occur in 5% to 10% of patients with systemic sarcoid, although this may be an underestimate.^{25,26} Adult neurosarcoid characteristically presents with cranial nerve palsies, most commonly involving the 7th nerve.^{19,27} A recent review reported that 50% of adults presented with cranial nerve palsies, 30% with headaches, and 10% each with seizures, pituitary dysfunction, sensory deficits, neuropsychological deficits, and cerebellar signs (patients could have >1 sign on presentation).^{19,27} In adults, both seizures^{28,29} and CNS involvement²⁶ have been considered markers of poor outcomes.

Our review of the literature finds a very different pattern for childhood neurosarcoid. As with other encephalopathies, prepubertal children are likely to present with seizures. The adult pattern of cranial nerve involvement appears only after puberty. Evidence of hypothalamic dysfunction is also common in children, with patients showing growth failure, diabetes insipidus, and lack of sexual maturation.

TABLE 1. Childhood Sarcoid With Well-Documented Nervous System Involvement

No.	Age at CNS Onset (y)	Sex	CNS Presentation	Other Clinical Findings	CNS Imaging	Diagnostic Tests	Pathology	Reference
1	3/12	F	Convulsions	Hepatosplenomegaly and skin rash	N/A	CSF: pleocytosis	Autopsy: sarcoid lesions in falx cerebri and meninges	35
2	2	F	Short stature	Lymphadenopathy	N/A	Abnormal chest x-ray?	Autopsy: granulomas in multiple tissues including the pituitary	36
3	6	F	Intermittent confusion, focal seizures, and papilledema	Fever, rash, lymphadenitis, and uveitis	N/A	CSF: pleocytosis, increased pressure, and increased protein	Lymph node biopsy positive	30
4	7	M	Seizures	Arthritis, rash, and uveitis	MRI: disseminated hyperdense areas	N/A	Skin biopsy positive	20
5	8	F	Seizure	Wheezing, macular skin lesions, and adenopathy	Ring lesion in cortex on CT	Chest x-ray: disseminated lesions in lungs	Lymph node biopsy positive	9
6	9	F	Polydipsia and polyuria	Shortness of breath and rash	N/A	Chest x-ray: disseminated lesions throughout the lungs	Skin biopsy positive	8
7	9	F	Headache and seizures	Isolated CNS sarcoid	MRI: increased T2 of caudate and multiple cortical gyri		Brain biopsy positive	This study
8	9	F	Seizures and somnolence	Arthritis, rash, and uveitis	N/A	CSF: elevated protein	Skin biopsy positive	20
9	9	F	Seizures and motor signs	Arthritis, rash, and uveitis	N/A	N/A	Autopsy: granulomas in brain and multiple other organs	20
10	11	F	Seizure with slow intellectual and motor deterioration	Isolated CNS sarcoid	N/A	N/A	Autopsy: granulomas in brain and lungs	37
11	11	M	Stiff neck and headache	Fever, chills and weight loss	N/A	Kveim test positive	Lymph node and liver biopsies positive	38
12	12	M	Papilledema and headache	Parotitis	N/A	Chest x-ray: hilar lymphadenopathy	Lymph node biopsies positive	31
13	12	F	Asymptomatic	Lymphadenopathy	MRI: nodular lesions in optic chiasma and right sylvian fissure, below right lenticular nucleus	Chest x-ray: hilar adenopathy	Cervical mass biopsy	10
14	12	F	Decreased vision (unilateral)	Enlarged lymph nodes, liver, and spleen and conjunctivitis	N/A	Chest x-ray: wide upper mediastinum	Inguinal node positive	39
15	13	F	Diabetes insipidus and sexual immaturity	Adenopathy	N/A	Chest x-ray: hilar adenopathy	Supraclavicular node positive	39
16	13	M	Superior oblique ocular palsy	Panuveitis (bilateral)	MRI: multiple CNS masses	EEG showed left midtemporal seizure focus; CSF: 1 white blood cell, and protein: 49	Pontine mass biopsy: necrotizing granuloma	11
17	13	F	Focal seizures and headache	Fever, malaise, vomiting, and weight loss	CT normal		Positive skin biopsy	7
18	13	F	Multiple cranial nerve signs	Parotiditis	N/A	Chest x-ray: hilar adenopathy	Parotid biopsy positive	25 and 40

TABLE 1. Continued

No.	Age at CNS Onset (y)	Sex	CNS Presentation	Other Clinical Findings	CNS Imaging	Diagnostic Tests	Pathology	Reference
19	13	F	Diabetes insipidus	Vitreous hemorrhage	Skull x-ray normal	Chest x-ray: hilar adenopathy; CSF: 8 lymphocytes, protein = 76 mg%	Lymph node biopsy positive	41
20	13	F	Right-sided facial weakness and right visual loss	Bilateral uveitis, rash, and lymphadenopathy	N/A	Chest x-ray: hilar adenopathy	Lymph node positive	42
21	15	F	Bilateral facial palsies without parotid swelling	Uveitis	N/A	N/A	Iris and lymph node biopsies positive	43
22	15	M	Polyuria, polydipsia, and short stature	Cervical and axillary adenopathy	N/A	Water-deprivation specific gravity of 1.006	Cervical lymph node positive	44
23	<16*	M	Headache, vomiting, and somnolence	Arthritis, rash, and uveitis	N/A	N/A	Skin biopsy positive	20
24	16	M	Monocular visual loss		Radionuclide brain scan negative	Chest x-ray: hilar adenopathy	Optic nerve granuloma	45
25	16	M	Hemiparesis and papilledema	Isolated CNS sarcoid	Radionuclide scan & EEG: left frontal abnormality		Brain biopsy positive	12
26	16	M	Failure of growth and of sexual maturation	Joint mass	N/A	Chest x-ray: hilar adenopathy	Joint mass: noncaseating granuloma	41
27	17	M	Seizures	N/A	MRI: enhancing temporal mass	Chest x-ray: hilar adenopathy	"Histologic confirmation"	13
28	17	M	Seizures	Cough and adenopathy	Normal pneumoencephalogram	CSF pleocytosis	Autopsy: leptomeningeal and grey matter granulomata	46
29	18	M	Hemiparesis and papilledema	Isolated CNS sarcoid	Mass on Technetium 99m brain scan and angiogram	Kveim test negative	Brain mass: noncaseating granuloma	14

EEG indicates electroencephalography.

*Child had onset of sarcoid at 2 months of age and was reported at 16 years of age. Age at onset of CNS involvement was not given.

Most surprising of all is the large number of children with mass-like lesions on imaging. We found 7 cases not including 2 children with papilledema who were reported in 1937³⁰ and in 1969³¹ before the advent of modern imaging. It is also striking that 1 of the 7 children had no neurologic symptoms, and the mass-like lesion was discovered by MRI done "for completeness."¹⁰ Although adults with seizures and other intracranial lesions respond poorly to therapy¹⁵ and have a higher rate of chronic handicap,³² this does not seem to be true of our 9-year-old patient or of other children. Previous authors have regarded mass-like lesions as rare.¹⁰ It is unclear if they are wrong and this is an important and previously unrecognized feature of childhood neurosarcoid or if this is an artifact of a survey of reported cases, with unusual findings being more likely to be published.

Sarcoid is characterized by noncaseating granulomas, and all cases that we accepted were biopsy- or autopsy-proven, although biopsy material was often not from the CNS. Other tests can be useful in establishing the diagnosis, but biopsy still provides the strongest evidence for this disease.³³ Elevated ACE levels are not specific for sarcoid but are elevated in the serum of 70% to 80% of patients with systemic disease and in the CSF of ~50% of patients with neurosarcoid.¹⁹ Analysis of CSF may show a mild lymphocytosis (10–100 cells per μ L), mildly elevated protein (to 70 mg%), mildly decreased glucose, and increased immunoglobulins with oligoclonal banding.¹⁹ When available, the Kveim test can be positive in 85% of patients.³³ MRI has become increasingly useful. Findings in neurosarcoid include periventricular high-signal lesions on T2-weighted images,^{13,33,34} leptomeningeal enhancement,^{33,34} and solitary masses.³⁴ Many of the reported pediatric cases antedate the availability of MRI, and whether there are different patterns in children than in adults is unclear.

The most appropriate treatment for pediatric neurosarcoid is uncertain. Corticosteroids are widely used and are often effective.¹⁰ For adult patients for whom corticosteroid therapy fails, azathioprine or cyclosporin A have been prescribed.¹⁹

An important caveat to our analysis is that published case reports may not be representative of the population of patients with a disorder. Furthermore, many of these cases were published by nonneurologists whose interests were in pulmonary, rheumatic, or other manifestations of the disease. The neurologic findings were presented for completeness. Although we omitted those cases where we could not confidently extract a detailed picture of the patients' findings on presentation, it is always possible that if experienced neurologists rather than nonneurologists had reported these patients, they would have conveyed different findings.

CONCLUSIONS

An analysis of 29 pediatric neurosarcoid cases indicates that compared with adults, children are more likely to have seizures, less likely to have cranial nerve palsies, and perhaps more likely to have a space-occupying lesion. Analyzing these children by

age group, preadolescents and adolescents, suggests that pediatric cases evolve to adult patterns of disease, with more frequent cranial neuropathies and less frequent seizures as the patients progress to adulthood.

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