Sporadic pediatric meningiomas: a neuroradiological and neuropathological study of 15 cases

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OBJECTIVE Sporadic meningiomas have been classified in many different ways. Radiographically, these lesions can be described as occurring in either typical or atypical locations. The purpose of this study was to determine if there are any histopathological differences between sporadic meningiomas that arise in these varying locations in children.

METHODS The neuroimaging, histopathological findings, and clinical records in patients with sporadic pediatric meningiomas not associated with neurofibromatosis Type 2 or prior radiation therapy were retrospectively reviewed. Tumors were classified by radiological findings as either typical or atypical, and they were categorized histopathologically by using the latest WHO nomenclature and grading criteria.

RESULTS Fifteen sporadic meningiomas in pediatric patients were biopsied or resected at the authors' institution between 1989 and 2013. Five (33%) were typical in radiographic appearance and/or location and 10 (67%) were atypical. Four (80%) typical meningiomas were WHO Grade I tumors. Most (60%) of the atypical meningiomas were WHO Grade II or III.

CONCLUSIONS This study is the largest series of sporadic pediatric meningiomas in atypical locations to date. Although sporadic meningiomas are relatively infrequent in children, those with atypical imaging, specifically those with apparently intraparenchymal and intraosseous locations, may be more common than previously recognized. In this study, pediatric sporadic meningiomas arising in atypical locations, in particular intraparenchymal meningiomas, may be of higher histopathological grade. The authors' findings should alert clinicians to the potential for more aggressive clinical behavior in these tumors.

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KEY WORDS meningioma; atypical; oncology

MENINGIOMAS are the most common nonglial primary tumors of the CNS in adults and the most common extraaxial neoplasm, accounting for approximately 15% of all intracranial tumors.³² However, intracranial meningiomas in children and adolescents are rare. The incidence of meningiomas before the age of 16 years ranges from 0.4% to 4.6% of all primary brain tumors in this age group.^{3,23} Meningiomas are generally low-grade neoplasms with several distinctive pathological and imaging features. However, there are several important

histological variants of meningioma, and even a histologically classic meningioma can have unusual or misleading radiological features.

The radiologically typical meningioma is a well-demarcated, homogeneously enhancing, dural-based mass located parasagittally over the cerebral convexities or along the sphenoid wing. On the other hand, radiologically atypical meningiomas originate in unusual locations such as the orbit, paranasal sinuses, ventricles, or within the brain parenchyma or calvaria (intraosseous). In addition,

ABBREVIATIONS NF1, NF2 = neurofibromatosis Type 1, 2.

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unusual cross-sectional imaging features such as large meningeal cysts and ring enhancement can be misleading. Atypical imaging features are important to recognize, to ensure that meningiomas are included in the differential diagnosis.³⁴

Meningiomas account for fewer than 5% of pediatric brain tumors, with a reported slight male predominance.^{17,38} When encountered in children, there is an increased association with neurofibromatosis Type 2 (NF2) or prior radiation therapy for neoplasia.³² However, less commonly, sporadic meningiomas also occur in children, and they may exhibit atypical imaging features because they can arise in unusual locations, where they appear to be associated with a higher pathological grade.²⁶ Sporadic meningiomas in adults show different molecular features that correlate with location, suggesting the possibility that different mechanisms may be driving pathogenesis.⁶ Furthermore, it has been suggested that sporadic meningiomas in adults and children represent very different disease processes, as is the case with other primary brain tumors.^{2,44}

A number of studies characterizing NF2-associated and radiation-induced pediatric meningiomas have been published, but the radiological and pathological features of sporadic pediatric meningiomas are poorly characterized and limited to a few case reports.^{19,28,30} To address this, we examined all meningioma cases at our institution by performing a retrospective radiological and pathological review, and a correlation with literature review. We hypothesize that sporadic pediatric meningiomas arising in atypical locations based on imaging may predict a higher histopathological grade.

Methods

Patient Selection

A retrospective review was approved by our institutional review board. The analysis was performed in individuals who were admitted from January 1, 1989, to January 1, 2013. Hospital records were retrieved according to coding from the ICD-9. We searched records that included the ICD-9 codes 225.2 (meningioma). Patients with meningiomas associated with NF2 and those occurring after CNS radiation therapy for malignancy were excluded. Clinical records were also reviewed for demographic data.

Imaging Modalities and Findings

Images of the sporadic meningiomas were reviewed by pediatric neuroradiologists (J.A.R., C.P.P.). The neuroradiologists were blinded to the original radiology report and reached independent conclusions following analyses of each tumor. Any discrepancies in the interpretation were then resolved by consensus.

Radiography, CT, and MRI studies were reviewed. Brain CT scans were performed on a GE scanner (8-slice Ultralightspeed or 16-slice VCT scanner). Optiray 240 (Mallinckrodt, Inc.) was the primary CT intravenous contrast agent used at our institution during the study period. All MRI scans were performed on 1.5- or 3-T GE magnets (Signa Genesis, Excite, or HDxt; GE Medical Systems), and gadopentetate dimeglumine (Magnevist; Bayer

HealthCare Pharmaceuticals, Inc.) was the only MRI contrast agent used. The most common MRI brain sequences performed were precontrast sagittal and axial T1-weighted; axial proton density and T2-weighted fast spin echo; axial diffusion- and susceptibility-weighted; and postcontrast axial T1-weighted and coronal T2-weighted FLAIR. The MRI cervical spine sequences performed were precontrast sagittal and axial T1- and T2-weighted, and postcontrast sagittal and axial T1-weighted. All patients underwent at least one CT or MRI study prior to resection.

Recorded imaging findings included tumor location, size (volume in milliliters), enhancement, adjacent edema, presence of a dural tail, and malignant features (restricted diffusion or rapid growth) for each tumor by consensus. Tumor volume was calculated by multiplying the anteroposterior, craniocaudal, and transverse dimensions of the lesion by 0.52, for the volume of a prolate ellipse.

Typical meningiomas were classified by the following characteristics: CT demonstrating common diagnostic features including a sharply circumscribed solitary mass with a broad-based dural attachment and occasionally thickening and sclerosis of the adjacent calvaria. On noncontrast CT, the mass appears as an area of homogeneous hyperattenuation; after the administration of intravenous contrast, the mass homogeneously enhances.³³ The MRI features include a well-circumscribed, solitary, dural-based mass with a dural tail and inward displacement of the adjacent cortex.⁴⁹ On noncontrast MRI, meningiomas are typically hypointense to isointense with T1-weighted pulse sequences, and isointense to hyperintense with T2-weighted pulse sequences. On postcontrast MRI, meningiomas enhance homogeneously.⁴⁹ Surrounding vasogenic edema, internal calcifications, and thickening or scalloping of the adjacent calvaria are variably seen.⁵

Pathological Findings

All slides of histopathological specimens for all sporadic meningiomas were retrospectively reviewed by 2 neuropathologists (C.R.P., D.R.B.). Findings, including WHO subclassification and grade, were recorded for each tumor independently, and discrepancies were resolved by consensus.²⁹ All related pathology reports were also reviewed (D.R.B.) to confirm a complete correlation with the original review's diagnosis of the pathology materials.

For each specimen successfully cultured for karyotypic analysis, 2 karyotypes and metaphase spreads from each clone and/or case were prepared with the data processing methods by the Cytogenetics Laboratory at The Ohio State University. At central karyotype review sessions, every karyotype and metaphase spread was reviewed by 2 cytogeneticists. In some cases, usually those with more complex chromosome abnormalities and/or with suboptimal banding quality, other reviewers also rendered an opinion. Once consensus was reached, each submission was judged as either acceptable with adequate banding quality, acceptable with borderline banding quality, or inadequate and consequently rejected. Reasons for rejection included poor banding quality that made unequivocal karyotype interpretation impossible and, only in cases with a normal karyotype, analysis of < 20 metaphase cells.

TABLE 1. Demographic data and associated meningioma findings in patients with sporadic meningiomas

Imaging Finding	Case No.	Age (yrs)	Sex	Lesion Location	Histological Type	WHO Grade	No. Mitoses per 10 hpf	MIB-1 %	Necrosis	Brain Invasion	Karotype	Management	Outcome
Typical													
	1	1	M	Lt fronto-temporal convexity	Fibrous	I	0	0	Neg	Neg		Resection ×2	Living
	2	13	F	Rt parietal convexity	Transitional	I	5	0	Neg	Neg		No additional therapy	Living
	3	15	F	Lt falx parasagittal	Transitional	I	1	2	Focal	Neg		No additional therapy	Living
	4	16	F	Rt temporal convexity	Meningothelial	I	0	0	Neg	Neg		No additional therapy	Living
	5	6	F	Cervical extraaxial	Clear cell	II	0	5	Neg	Neg		No additional therapy	Living
Atypical													
	6	21	F	Intraparenchymal	Papillary	III	8	10	Neg	Pos	–X, –20, –21, –22 (monosomy 22)	Radiation	Living
	7	11	M	Intraparenchymal	Chordoid	II	2	5	Pos	Neg	t (12; 22)	No additional therapy	Living
	8	14	M	Intraventricular	Transitional	I	0	2	Neg	Neg		No additional therapy	Living
	9	12	M	Intraparenchymal	Transitional	II	0	10	Neg	Pos		No additional therapy	Living
	10	18	F	Intraosseous	Transitional	II	10	10	Neg	Neg		No additional therapy	Living
	11	17	F	Intraosseous	Transitional	I	1	2	Neg	Neg		No additional therapy	Living
	12	18	M	Intraparenchymal	Chordoid	II	0	1	Neg	Neg	t (14; 22)	No additional therapy	Living
	13	16	F	Intraparenchymal	Rhabdoid/anaplastic	III	9	50	Pos	Pos	Trisomy for chromosomes 3, 7, 9, 15, 21	Chemo + GK	Deceased
	14	7	F	Intraosseous	Metaplastic xanthomatous	I	0		Neg	Neg		No additional therapy	Living
	15	7	M	Intrafissural	Meningothelial	I	1	1	Neg	Neg		No additional therapy	Living

chemotherapy = chemo; GK = Gamma Knife; neg = negative; pos = positive.

Results

Forty-six meningiomas underwent either biopsy or resection at our institution between 1989 and 2013. Meningiomas associated with NF2 (6 children) and radiation therapy (16 adults, 9 children) were excluded, resulting in the remaining 15 pediatric sporadic meningiomas, accounting for 1.7% of pediatric brain tumors at our institution during the study period (Table 1).

Imaging Groups

The 15 meningiomas were divided into 2 imaging groups based on tumor location, and were designated as typical and atypical. Typical meningiomas were dural based, and atypical tumors were intraparenchymal, in-

traventricular, or intraosseous based on imaging findings alone. Five (33%) tumors were radiographically typical and 10 (67%) were radiographically atypical. Typical meningiomas occurred in 4 females and 1 male, ages 1–16 years (mean 11 years). Four (80%) of these lesions were intracranial; 1 (20%) was in the cervical spine. The tumors ranged in volume from 4 to 116 ml (mean 29 ml). All typical tumors showed CT and/or MR contrast enhancement (5/5, 100%) and most had adjacent edema (4/5, 80%) and/or a dural tail (4/5, 80%). No restricted diffusion or rapid growth was seen in the 3 typical tumors that had diffusion-weighted imaging (Table 2, Fig. 1A–D).

The 10 atypical meningiomas were seen in 5 female and 5 male patients with a similar age range of 7–21 years

TABLE 2. Demographic data and characteristics of lesions included in the study of sporadic atypical and typical meningiomas

Characteristic	Typical, n = 5	Atypical, n = 10
Mean age in yrs (range)	11 (1–16)	10 (7–21)
Females	4 (80%)	5 (50%)
Tumor location		
Intraparenchymal	4 (80%)	5 (50%)
Spinal	1 (20%)	
Intraosseous		3 (30%)
Intraventricular		1 (10%)
Fissural		1 (10%)
Mean vol in ml (range)	29 (4–116)	16.2 (1–35)
Enhancement	5 (100%)	8 (80%)
Adjacent edema	4 (80%)	3 (30%)
Dural tail*	4 (80%)	1 (10%)
WHO Grade		
I	4 (80%)	4 (40%)
II	1 (20%)	4 (40%)
III		2 (20%)

* Dural tail in this table is based on imaging findings only.

(mean 10 years). Half of these tumors appeared to be intraaxial rather than dural based, and were classified as intraparenchymal (5/10, 50%). Three tumors (30%) were intraosseous, 1 (10%) was intraventricular, and 1 was fissural (10%). Atypical tumor volume ranged from 1 to 35 ml (mean 16 ml). Most of these tumors enhanced after contrast administration (8/10, 80%), a small percentage showed adjacent edema (3/10, 30%), and rarely was there a dural tail (1/10, 10%). One of the intraparenchymal tumors showed restricted diffusion and rapid growth. All sporadic meningiomas, regardless of location, showed relatively well-circumscribed borders without radiologically infiltrative margins (Table 2, Fig. 1E–H).

Pathological Findings

Four (80%) of the typical tumors were classified as WHO Grade I, with the other one classified as WHO Grade II (clear cell histopathology). None of the radiographically typical tumors had malignant features, meaning that no anaplastic cytological features or abnormally increased mitoses were observed. Brain invasion and necrosis are more common in malignant meningiomas, but these features alone are insufficient to define a meningioma as high grade (Tables 1 and 2, Fig. 1A–D).

Most of the radiographically atypical tumors (6/10, 60%) were classified as WHO Grade II or III. Intraparenchymal tumors were classified as WHO Grade II (transitional [1], chordoid [2]) or WHO Grade III (papillary [1], rhabdoid/anaplastic [1]) pathology (Table 1). One intraparenchymal atypical meningioma with Grade II chordoid histopathological findings demonstrated rapid growth over a 3-month period (Fig. 1G and H). At gross and histopathological evaluation, 3 of the 5 intraparenchymal meningiomas, which appeared to have no dural tail at imag-

ing, were ultimately found to have dural attachments on pathological evaluation. Tumor cells grew for karyotype analysis from 3 intraparenchymal meningiomas. These tumors had abnormal karyotypes involving chromosome 22, and included 2 WHO Grade II (both chordoid) atypically located meningiomas and one WHO Grade III (papillary) atypically located meningioma.

There were 3 intraosseous tumors (1 WHO Grade II and 2 WHO Grade I), of which 2 showed transitional histopathological features; all 3 of these meningiomas were seen in female patients (Fig. 1I and J). Both the intraventricular and fissural radiographically atypical meningiomas showed WHO Grade I pathology.

Discussion

There is significant literature on the unique pathological features of meningiomas associated with NF2 and prior radiation, which often have a higher pathological grade.^{10,20,28} There are fewer publications regarding pediatric sporadic meningiomas. Most articles are in the form of case reports or small series in which sporadic tumors and their pathology were not specifically evaluated.^{2,7,31,44}

The purpose of our retrospective study of 15 pediatric sporadic meningiomas collected over 24 years was to determine if sporadic pediatric meningiomas with atypical radiological features would be of a higher pathological grade. Tumors were considered typical if they appeared to be dural based and atypical if they were intraparenchymal, intraventricular, or intraosseous. Even though this is the largest series of its kind, the number of cases is still relatively limited; however, in our experience most of the atypically located tumors were WHO Grade II or III, whereas tumors arising in more typical locations were primarily WHO Grade I or II.

In particular, our study had a fairly large number of intraparenchymal meningiomas, the largest number in a single study. In previous meta-analyses, 80%–90% of meningiomas are Grade I, whereas 5%–15% are Grade II, and 1%–3% are Grade III.^{27,39} Histopathologically, the meningothelial subtype is the most common (54.3%), whereas the fibrous subtype accounts for 17.2%.²⁷ Interestingly, in a recent review of intraparenchymal meningiomas, 28% of the pediatric cases were Grade II–III²⁶ (Table 3).^{8,9,11–16,18,21–25,35,37,40–43,45–48} WHO Grade II–III meningiomas seem to be more common in the pediatric population.³¹ In one review of meningiomas in children and adolescents, 78.9% were WHO Grade I, 9.9% were Grade II, and 8.9% were Grade III.¹⁷

Recent large genetic analyses of meningiomas have demonstrated mutations in a number of genes other than the prototypical Neurofibromin 2 mutation on chromosome 22, which is found in most WHO Grade I tumors.^{4,6} Interestingly, in our small series, the 3 tumors with successfully obtained karyotypes showed involvement of chromosome 22, these tumors were primarily WHO Grade II–III, were all atypical in location, and were all intraparenchymal lesions (Table 1). These recently characterized mutations are believed to alter specific signaling pathways and suggest that meningiomas can arise via different molecular mechanisms and that they may play a role in WHO

FIG. 1. Representative MR images of both typical and atypical meningiomas with corresponding pathological findings. **A and B:** Case 4. A 16-year-old girl with extraaxial typical meningioma. **A:** Axial T1-weighted postcontrast MR image shows an extraaxial mass in the anterior right middle cranial fossa with homogeneous enhancement. There was adjacent vasogenic edema (not shown). **B:** Photomicrograph of a WHO Grade I, meningothelial meningioma shows a meningothelial pattern with mostly uniform tumor cells containing oval nuclei. Lobules of cells are demarcated by thin collagenous septae. **C and D:** Case 2. A 13-year-old girl with extraaxial typical meningioma. **C:** Axial T1-weighted postcontrast MR image shows an extraaxial mass adjacent to the right posterior frontal lobe with homogeneous enhancement. There was adjacent vasogenic edema (not shown). **D:** Photomicrograph of a WHO Grade I, transitional meningioma shows meningothelial and fibrous patterns and subtle whorling appearance. **E and F:** Case 12. An 18-year-old man with intraparenchymal atypical meningioma. **E:** Axial T2-weighted MR image shows a heterogeneous right frontoparietal mass along the falx, with extensive surrounding edema. **F:** Photomicrograph of a WHO Grade II, chordoid meningioma (cords of meningioma cells in a mucinous background), which is attached to and invades the dura mater (lower half of panel). **G and H:** Case 9. A 12-year-old boy with intraparenchymal atypical meningioma. **G:** Coronal postcontrast T1-weighted image shows left temporal lobe mass with enhancement. **H:** Photomicrograph of a WHO Grade II, transitional meningioma (lower half of panel) with marked intermixed chronic inflammation, infiltrating the brain (upper half of panel). Tumor surrounds the brain and infiltrates along Virchow-Robin spaces of vessels (arrow). **I and J:** Case 14. A 7-year-old girl with intraosseous atypical meningioma. Lytic lesion in the left parietal bone. **I:** Coronal T2-weighted FLAIR postcontrast image shows no involvement of underlying brain parenchyma. **J:** Photomicrograph of a WHO Grade I transitional meningioma shows metaplastic (xanthomatous) change and a fibrous subtype component (spindle cells with collagen deposition). H & E, original magnification $\times 400$. Figure is available in color online only.

Grade II and III lesions. Studies show that skull base and ventral meningiomas manifest distinct genetic alterations, indicating that meningiomas differ biologically based on location. However, none of these studies included pediatric patients, so these data may only reflect the genetics of adult meningiomas.

It is very likely that meningiomas in adult and pediatric patients arise by different mechanisms, because the clinical setting is markedly different.² Therefore, apply-

ing molecular analyses in pediatric meningiomas may help elucidate potential biological differences between sporadic pediatric and adult meningiomas to account for the higher rates of invasion and recurrence in the former. Cellular proliferation is not substantially different between pediatric and adult meningiomas, so this alone cannot account for differences in tumor behavior.^{1,36} This further indicates that meningiomas arising in adults and children develop according to different molecular mecha-

TABLE 3. Literature review of reported cases of intraparenchymal meningiomas

Authors & Year	Age (yrs)	Sex	Symptom	Location	Pathology	WHO Grade
Taptas, 1961	0.3	F	Enlargement of head	Temporoparietal	Fibroblastic	I
Mendiratta et al., 1967	0.5	M	Seizure	Temporal	Meningothelial & fibroblastic	I
Koos & Miller, 1971	0.9	M	Seizure	Frontal	Fibroblastic	I
Suematsu et al., 1974	0.4	M	Seizure	Parietal	Fibroblastic	I
Satyanarayana et al., 1975	0.5	M	Enlargement of head	Frontoparietal	Fibroblastic	I
Morimoto et al., 1976	17	F	Seizure	Parietal	Malignant	III
Dong et al., 1980	0.3	M	Enlargement of head	Temporal	Fibroblastic	I
Schut et al., 1994	5	M	Facial twitching	Temporal	Mix (fibroblastic, transitional, psammomatous)	I
Legius et al., 1985	1.2	M	Seizure	Parietal	Fibroblastic	I
Drake et al., 1985–1986	12	M	Seizure	Temporal	Transitional	I
Sakaki et al., 1987	0.9	M	Seizure	Frontal	Fibroblastic	I
Schroeder et al., 1987	7	M	Seizure	Frontal	Fibroblastic	I
Mamourian et al., 1991	2	F	Enlargement of head	Frontal	Meningothelial	I
Matsumoto et al., 1992	6	M	Seizure	Frontal	Transitional	I
Kaneko et al., 1993	12	F	Seizure	Temporal	Meningothelial	I
Kohama et al., 1996	1.8	F	Seizure	Frontal	Fibroblastic	I
Sanli et al., 1996	23	F	Seizure	Frontal	Psammomatous	III
Teo et al., 1998	2	F	Bulbar dysfunction & rt hemiparesis	Brainstem	Clear cell	II
Karadereler et al., 2004	14	M	Seizure	Temporal	Fibroblastic	I
Zhang et al., 2007	16	M	Seizure	Parietooccipital	Atypical	II
Shimbo et al. 2011	10	M	Seizure	Frontal	Meningothelial w/ chordoid	II
Jiang et al., 2012	23	M	Lt upper limb & facial nerve palsy	Brainstem	Papillary	III
Jung & Song, 2012	1.7	M	Seizure & lt hemiparesis	Frontoparietal	Transitional	I
Pinto et al., 2012	17	F	Seizure	Temporal	Not specified	I
Nayil et al., 2015	3	M	Headache, vomiting	Frontal	Anaplastic	III

nisms, are different biologically, and may need to be treated differently. The application of unbiased genetic profiling techniques has demonstrated that other adult and pediatric brain tumors that share similar histopathological features are distinct biological entities at the molecular level, which could very well be the case for meningiomas also.^{1,2,6,36}

We were interested in testing whether atypical histopathological findings could be predicted based on atypical imaging findings and, in this series, radiologically atypical sporadic meningiomas were more likely to be high grade (60% were WHO Grade II or III). We found radiographically typical meningiomas mostly in girls (4 females and 1 male), but radiographically atypical meningiomas arose in both sexes equally (5 female and 5 male patients). In adults, meningiomas tend to arise in females, in contrast to children, where males reportedly predominate in some studies.^{17,21,32} More cases will need to be studied to fully elucidate the effect of sex in pediatric patients.

Limitations of our study include the relatively small size of this single-institution cohort, and because our institution is a pediatric brain tumor referral center this may affect the ability to generalize our data. The retrospective design may also be limiting—cases were collected over a period of many years, during which technological advances occurred. For this reason diffusion- and susceptibility-

weighted sequences were only performed in a small number of the more recent cases, disallowing comprehensive imaging analysis of these parameters across the entire cohort. Even though this is the largest series of sporadic pediatric meningiomas in atypical locations, caution is warranted due to the limited total number of cases. It is still worth noting, however, that the incidence of meningioma histopathological subtypes seen in this series was higher than would be expected in an adult series.³²

Conclusions

This is the largest series of its kind to analyze both intraosseous and intraparenchymal meningiomas among sporadic and familial meningiomas. In a previous meta-analysis of more than 677 meningiomas (including NF1- and NF2-affected patients), there were only 6 intraparenchymal and 1 intraosseous lesion.¹⁷ Therefore, our study represents the most complete investigation to date examining the imaging appearance of typical and atypical meningiomas and their associated pathological features. Sporadic meningiomas are uncommon in children, and most show atypical histopathological features that appear to correlate with a higher WHO pathological grade. Awareness of these distinctions is important for proper patient management. In the future, molecular profiling hopefully

will improve our understanding of the apparent biological differences between pediatric and adult meningiomas.

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Disclosures

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Conception and design: Huntoon, Pierson. Acquisition of data: Huntoon, Pluto, Pierson. Analysis and interpretation of data: Huntoon, Boué, Pierson, Rusin. Drafting the article: Huntoon, Pluto, Boué, Pierson, Rusin. Critically revising the article: Huntoon, Ruess, Boué, Pierson, Rusin. Reviewed submitted version of manuscript: Huntoon, Pierson, Leonard. Approved the final version of the manuscript on behalf of all authors: Huntoon.

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