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Contrasting features of childhood and adolescent melanomas

Diana W. Bartenstein, BA^{1,2,3}, Cassandra M. Kelleher, MD^{1,4}, Alison M. Friedmann, MD^{1,5}, Lyn M. Duncan, MD^{1,6}, Hensin Tsao, MD, PhD^{1,3}, Arthur J. Sober, MD^{1,3}, and Elena B. Hawryluk, MD, PhD^{1,3}

¹School of Medicine, Harvard University, Boston, MA, USA

²School of Medicine, Tufts University, Boston, MA, USA

³Department of Dermatology, Massachusetts General Hospital, Boston, MA, USA

⁴Department of Pediatric Surgery, Massachusetts General Hospital, Boston, MA, USA

⁵Division of Hematology/Oncology, Massachusetts General Hospital, Boston, MA, USA

⁶Pathology Service, Massachusetts General Hospital, Boston, MA, USA

Abstract

Background/Objectives: Melanoma in children and adolescents is uncommon, and there are limited data on pediatric outcomes. Several studies have shown comparable survival rates in children and adults, but other research demonstrates that pre-pubescent children have more favorable outcomes. This study aims to compare childhood and adolescent melanoma.

Methods: Retrospective cohort study of children who received a melanoma diagnosis at the Massachusetts General Hospital between January 1, 1995, and December 31, 2016. Childhood melanoma is defined as disease occurring in patients younger than 11 years old, and adolescent melanoma is defined as disease occurring in patients 11 to 19 years old. Patients diagnosed with ocular melanoma and border-line tumors of uncertain malignant potential were excluded. This analysis compares clinical, histopathologic, and outcome characteristics of childhood and adolescent melanoma.

Results: Thirty-two children with melanoma were identified (12 children, 20 adolescents). The spitzoid melanoma subtype was significantly more common in children (6/12) than adolescents (2/20) ($P = .01$). Four adolescents and no children with melanoma died from melanoma, and survival was significantly different between the age groups ($P = .04$). Median follow-up time for survivors was 3.6 years.

Conclusions: These results suggest that children and adolescents present with different melanoma subtypes and that adolescents have a more aggressive disease course than children.

Correspondence: Elena B. Hawryluk, MD, PhD, Department of Dermatology, Massachusetts General Hospital, Boston, MA, USA., ehawryluk@partners.org.

ETHICAL APPROVAL

Institutional review board approval was obtained before beginning this study.

Keywords

bumps; lumps; malignant; neoplasms

1 | INTRODUCTION

Melanoma is a rare disease in children, and it is a particularly rare diagnosis in prepubescent children. Approximately 2% of all melanomas occur in children and adolescents, and melanoma accounts for 1% to 3% of all pediatric malignancies.^{1,2} Analysis of 2000 to 2010 data from the Surveillance, Epidemiology, and End Results cancer registry estimated the incidence to be 5.93 melanomas per 1 000 000 children and adolescents.³ In this registry data, which included 1185 children with melanoma aged 0 to 19, 4% were diagnosed between 0 and 4 years old, 7% between 5 and 9 years old, and the remaining 89% between ages 10 and 19.³

Several studies have found mortality to be comparable for children and adults patients presenting at similar disease stage,^{4–6} and providers typically follow adult protocols when caring for children and adolescents diagnosed with melanoma, but very young children may have more favorable outcomes.^{7–9} Few children with melanoma younger than 11 years old die, despite findings that they tend to be diagnosed later, are more likely to present with thicker tumors, and often have sentinel lymph node metastasis.^{9,10} Given these clinical differences, researchers have proposed that pediatric melanoma in very young children may be a distinct biologic entity.^{7,10}

Adult-based protocols for treating pediatric melanoma may incur unnecessary morbidity in children if their disease follows a less aggressive disease course. Better characterization of pediatric melanoma is a crucial research priority with the potential to meaningfully affect patient management and patient counseling. In this study, more than 2 decades worth of data from children and adolescents with melanoma treated at Massachusetts General Hospital (MGH) in Boston, Massachusetts, are analyzed. MGH is a tertiary referral center with a full-service pediatric hospital, and the institution's Pigmented Lesion Clinic treated more than 3200 individuals during the last 7 years of the study period.

2 | MATERIALS AND METHODS

A retrospective cohort study design was used. After obtaining institutional review board approval (MGH IRB 2016P001413), individuals younger than 20 years old with a diagnosis of melanoma between January 1, 1995, and December 31, 2016, were identified through a query of our institution's Research Patient Data Registry for melanoma-related diagnoses. Chart review was performed to confirm diagnoses with MGH histopathology diagnosis. Patients with ocular melanoma and borderline diagnoses such as severely atypical dysplastic nevus, atypical Spitz tumor, melanocytic tumors of uncertain malignant potential, and pigmented epithelioid melanocytomas were excluded. Patient demographic information, clinical presenting features, histopathologic characteristics, management course, and outcomes were extracted from the electronic medical record. For data analysis, patients were divided into two groups: childhood melanoma (children), which includes patients diagnosed

before age 11, and adolescent melanoma (adolescents), which includes patients diagnosed at or after age 11 and before age 20; 11 was chosen in keeping with the literature as the age cut-off to separate prepubertal patients.^{9,11,12}

3 | RESULTS

Thirty-two children with melanoma were identified (Table 1). Ages ranged from 3.3 to 19.5 years at time of diagnostic biopsy. There were 12 children and 20 adolescents, and median age at biopsy was 9.3 and 15.7 years, respectively. Median follow-up time for survivors in the full cohort was 3.6 years, with 80% of patients having more than 1.6 years of follow-up.

There were no significant differences between children and adolescents in sex, race, Fitzpatrick skin phototype, or tumor site (Table 2). Many melanomas in both groups demonstrated lesional evolution before biopsy (9 children, 14 adolescents), but very few patients presented with the following risk factors: first-degree family history of melanoma (0 children, 1 adolescent), previous blistering sunburn (0 children, 4 adolescents), indoor tanning activity (0 children, 2 adolescents), significant medical comorbidity (1 child, 0 adolescents), or predisposing skin lesion (1 child, 4 adolescents).

Children and adolescents had different melanoma subtypes. Children most commonly presented with spitzoid ($n = 6$), followed by in situ ($n = 2$), nodular ($n = 2$), superficial spreading ($n = 1$), and unclassified melanoma ($n = 1$). Adolescents most commonly presented with superficial spreading ($n = 7$), followed by in situ ($n = 6$), unclassified ($n = 4$), spitzoid ($n = 2$), and nodular melanoma ($n = 1$). Spitzoid melanomas were significantly more common in children than adolescents ($P = .01$). In regards to predisposing lesions, one superficial spreading melanoma in a child developed in a medium congenital melanocytic nevus, two superficial spreading melanomas in adolescents developed in small congenital melanocytic nevi, one adolescent with a superficial spreading had an unrelated medium congenital melanocytic nevus, and one adolescent with an unclassified melanoma subtype presenting in the brain had a nevus of Ota.

Children tended to present at a more advanced histopathologic stage, although results were not significant; 58% of children versus 25% of adolescents presented with American Joint Committee on Cancer T stage 3 or 4 (Table 1), and median Breslow thickness was 3.5 mm for lesions in children versus 1.5 mm in adolescents. More children presented with Clark level IV and V tumors (41.6%) than adolescents (35%), and median mitotic index was also found to be greater in children (5 mitotic figures per mm^2) than adolescents (2 mitotic figures per mm^2). Children also were more likely to have neural invasion (Table 3).

Sentinel lymph node biopsy was performed in 8 of 11 (72.3%) children and 8 of 20 (40%) adolescents; within both groups, 50% of sentinel lymph node biopsies were positive. For additional staging, 4 of 11 (36.3%) children and 8 of 20 (40%) adolescents underwent imaging as part of their care. The percentage of adolescents receiving treatment other than excision (7/19, 36.8%) was slightly higher than of children (3/11, 27.2%), but this was not statistically significantly different. Treatments included interferon, radiation, and immune checkpoint inhibitors for children and chemotherapy, systemic chemotherapy, interferon,

interleukin-2, radiation, immune checkpoint inhibitors, and autologous granulocyte-macrophage colony-stimulating factor-secreting cell therapy for adolescents.

Four patients experienced distant metastasis and died, all of whom were adolescents (20%) (Figure 1). Survival curves were significantly different for adolescents and children based on the log-rank test ($P = .04$, Figure 1). When patients were compared according to melanoma subtype, there was no significant survival difference (log-rank test $P = .06$, Figure 2). For both of these analyses, patients with melanoma in situ were excluded, because stage 0 melanoma does not carry a risk for metastasis.

4 | DISCUSSION

This study confirms previous observations that pediatric melanoma, particularly early childhood melanoma, is uncommon. Data from this cohort are also consistent with previous findings that melanoma in early childhood is associated with more aggressive histopathologic and staging features with, paradoxically, better outcomes than those of older adolescents and adults. During more than 20 years at this tertiary referral center, no one with melanoma younger than 11 died, with a follow-up ranging from 9 to 137 months and median follow-up of 44 months.

These results support the hypothesis that melanoma in young children may be biologically distinct from melanoma in adults. Alternatively, melanoma subtype may drive survival differences between children and adolescents. Adolescents and children in this cohort had different subtype distributions, and children younger than 11 were significantly more likely to have spitzoid melanoma. A study by Carrera and colleagues corroborates the finding that, in children, spitzoid melanoma is associated with younger age than nonspitzoid melanoma.¹³ Although our results did not demonstrate a significant difference in survival according to melanoma subtype, and a recent prospective study of pediatric melanoma demonstrated that spitzoid subtype does not confer a survival benefit over nonspitzoid melanoma,¹⁴ larger studies are required to determine whether adolescents present with more aggressive melanoma subtypes than younger children.

Further research on outcomes for individuals with spitzoid melanoma is particularly important given that a subset of spitzoid tumors present histopathologic challenges.¹⁵ For these cases, even expert dermatopathologists may have difficulty achieving consensus as to whether the tumors are atypical or outright malignant.¹⁶ Histopathologic features that support a diagnosis of spitzoid melanoma include asymmetry, poor circumscription, ulceration, lack of Kamino bodies, lack of junctional clefting, dense cellularity, irregular nests with poor maturation, deep mitotic figures, heterogeneous cell types, and specific nuclear characteristics.¹⁷ At the time of this study, genomic tests had not proven helpful in distinguishing challenging spitzoid tumors in children,¹⁸ and none of these cases were evaluated using molecular techniques. All cases diagnosed as spitzoid melanoma in this cohort underwent additional histopathologic evaluation by an independent dermatopathologist consultant or consensus review before melanoma diagnosis was finalized, and we intentionally excluded borderline and atypical Spitz tumors in this study. Although previous literature is potentially limited by inclusion of atypical tumors of

uncertain malignant potential, even with the inclusion of borderline cases, spitzoid melanoma has not been shown to have a uniquely favorable clinical course.¹⁴ Larger studies are required to elucidate whether this subtype confers a survival benefit. Another explanation for better outcomes in childhood than adolescent melanoma is variation in parental surveillance. Whereas younger children may undergo frequent skin monitoring, parents may not observe suspicious lesions in adolescents, who value their privacy.

Challenges in physicians' ability to suspect and recognize melanoma in young children may contribute to the "advanced" clinical and histopathologic features of children observed in this study. Pediatric melanoma is commonly misdiagnosed as a benign mimic lesion,¹⁹ and melanoma in younger children is less likely to present with the typical ABCD features (asymmetry, border irregularity, color heterogeneity, diameter >6 mm) than in adolescents.²⁰ Pediatric melanomas may present as lesions that are amelanotic, that bleed, bumps, that are homogenous in color, and that arise de novo, and these pediatric-specific "ABCDE" detection criteria have been proposed for use in conjunction with typical detection criteria,²⁰ although a recent study showed that the majority of pediatric melanomas in an international cohort (N = 52) did not meet the modified pediatric ABCDE criteria. Characteristic dermoscopic features were helpful for diagnosis of these malignant lesions.¹³ Medical records at this institution did not consistently document ABCDE or dermoscopic features before biopsy, and we are unable to comment on the presence of these features in our cohort. Nevertheless, we recommend careful attention to pediatric and traditional melanoma features, as well as dermoscopic characteristics, when choosing whether to biopsy a suspicious lesion in a child.

Small sample size and acquisition from a single tertiary referral center limited this study. There were no cases of melanoma associated with large or giant congenital melanocytic nevi in the cohort. This subtype is known to cause disproportionate mortality in very young children, and higher mortality may have been observed if more children with giant congenital melanocytic nevi had been diagnosed at this institution and thus included in this cohort.¹² The only central nervous system melanoma identified in this study was an 18-year-old boy with unclassified melanoma of the brain who had a nevus of Ota. Nevus of Ota has been reported to undergo malignant degeneration in as many as 4.6% of cases,²¹ may present a higher risk of malignancy in white individuals,²² and has been shown to be associated with malignancy in children.²³ Longer follow-up might provide important insights. In a recent population-based review of pediatric melanoma, average time to death after diagnosis was 9.3 years.¹¹

Despite study limitations, clinical findings observed in this cohort illustrate two important differences in melanoma presenting in childhood than in adolescence. First, spitzoid melanomas appear to be more common in children. Second, melanoma may follow a more aggressive course in adolescents than children.

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FIGURE 1.

Kaplan-Meier survival curve according to age group (Censored observations indicate maximum follow-up time for living patients)

FIGURE 2.

Kaplan-Meier survival curve according to melanoma subtype (Censored observations indicate maximum follow-up time for living patients)

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TABLE 1

Cohort characteristics

Cohort characteristics										Time from diagnosis to distant metastasis				AJCC T stage ²⁴		Stage	
Age, years	Sex	Race	Time to diagnosis, months	Primary site	Melanoma subtype	Breslow thickness, mm	Ulceration	Mitotic index (per mm ²)	Lymph node status	Treatment other than excision	Outcome	Time from diagnosis to distant metastasis & death, months	Survival time, months	AJCC T stage ²⁴		Stage	
3.3	Female	NR	6	Leg	Spitzoid	3.5	No	14	SLNB positive after lymphadenectomy (4 nodes positive)	Interferon	Alive	-	19	T3a		IIIC	
3.6	Female	White	10	Back	Spitzoid	2.6	No	7	Negative SLNB	None	Alive	-	101	T3a		IIA	
5.4	Male	White	Unknown	Arm	Spitzoid	1.75	No	5	Negative SLNB	None	Alive	-	137	T2a		IB	
6.7	Female	White	NR	Scalp	Nodular	10	No	15	SLNB positive after lymphadenectomy (1 node positive)	Interferon, radiation, immune checkpoint inhibitor	Alive	-	108	T4a		IIIA	
6.8	Female	Hispanic	12	Arm	Spitzoid	4.3	No	1	Negative SLNB	None	Alive	-	10	T4a		IIB	
9.2	Female	White	17	Buttocks	Spitzoid	5.5	Yes	7	SLNB positive after lymphadenectomy (1 node positive)	Interferon	Alive	-	24	T4b		IIIB	
9.3	Male	White	19	Sole of foot	In situ	NR	NR	NR	SLNB not performed	None	Alive	-	20	-		0	
9.3	Male	White	24	Dorsum of foot	In situ	NR	NR	NR	SLNB not performed	None	Alive	-	87	-		0	
9.6	Male	Other	36	Leg	Spitzoid	85	No	4	SLNB positive after lymphadenectomy (1 node positive)	None	Alive	-	20	T4a		IIIA	
10.1	Female	Unknown	Unknown	Face	Unclassified	0.9	Yes	2	Unknown	Unknown	Alive	-	61	T1b		IB	
10.5	Female	White	Unknown	Scalp	Nodular	3.4	Yes	2	Negative SLNB	None	Alive	-	121	T3b		IIB	
10.8	Male	White	8	Leg	Superficial spreading	0.9	NR	Few	SLNB not performed	None	Alive	-	94	T1b		IB	
11.5	Female	White	12	Arm	Unclassified	36	No	10	SLNB positive after lymphadenectomy (1 node positive)	Interferon, topical chemotherapy, interleukin n-2, vaccine therapy, immune checkpoint inhibitor, autologous granulocyte-macrophage colony-stimulating factor–secreting cell therapy	Dead	30 & 55	-	T4a		IIIA	

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Age, years	Sex	Race	Time to diagnosis, months	Primary site	Melanoma subtype	Breslow thickness, mm	Ulceration	Mitotic index (per mm ²)	Lymph node status	Treatment other than excision	Outcome	Time from diagnosis to distant metastasis & death, months	Survival time, months	AJCC T stage ²⁴	Stage
12.4	Male	White	0.25	Leg	Superficial spreading	1.02	No	2	SLNB positive after lymphadenectomy (1 node positive)	Interferon	Alive	-	80	T2a	III A
12.5	Female	White	Unknown	Arm	Superficial spreading	0.6	No	0	SLNB not performed	None	Alive	-	111	T1a	IA
13.0	Female	White	Unknown	Back	In situ	NR	NR	NR	SLNB not performed	None	Alive	-	14	-	0
14.1	Female	White	Unknown	Dorsum of foot	In situ	NR	NR	1	SLNB not performed	None	Alive	-	95	-	0
14.1	Female	White	Unknown	Arm	In situ	NR	NR	NR	SLNB not performed	None	Alive	-	86	-	0
14.2	Male	Other	36	Dorsum of hand	In situ	NR	NR	NR	SLNB not performed	None	Alive	-	31	-	0
14.6	Female	White	12	Leg	Superficial spreading	1.87	Yes	6	Negative SLNB	Interferon	Alive	-	55	T2b	II A
15.7	Male	White	Unknown	Leg	Spitzoid	3	Yes	3	Negative SLNB	None	Alive	-	26	T3b	II B
15.7	Female	White	42	Scalp	Unclassified	NR	NR	NR	SLNB not performed	Interleukin-2	Dead	2 & 23	-	Not completely staged	Not completely staged
15.8	Female	White	5	Face	Spitzoid	7.25	No	2	SLNB not performed	None	Alive	-	25	T4a	II B
16.1	Male	White	12	Scalp	Unclassified	3	Yes	5	SLNB positive after lymphadenectomy (3 nodes positive)	BRAF inhibitor	Alive	-	48	T3b	IIIB
16.3	Male	NR	24	Chest	Superficial spreading	1.1	No	2	SLNB not performed	None	Alive	-	9	T2a	IB
17.2	Male	White	3	Back	Nodular	3.75	No	4	SLNB positive after lymphadenectomy (1 node positive)	Interferon, radiation	Dead	23 & 34	-	T3a	III A
17.4	Female	White	Unknown	Arm	Superficial spreading	0.7	No	1	SLNB not performed	None	Alive	-	43	T1b	IB
17.7	Male	White	Unknown	Back	In situ	NR	NR	NR	SLNB not performed	Unknown	Alive	-	44	-	0
18.1	Male	White	Unknown	Brain	Unclassified	NR	NR	Abundant	SLNB not performed	Chemotherapy, radiation, immune checkpoint inhibitor	Dead	0 & 8.27	-	-	IIIC
18.5	Female	White	60	Face	In situ	NR	NR	NR	SLNB not performed	None	Alive	-	26	-	0
19.3	Female	White	12	Face	Superficial spreading	0.95	No	1	Negative SLNB	None	Alive	-	16	T1b	IB
19.5	Female	White	6.5	Leg	Superficial spreading	0.53	No	1	SLNB not performed	None	Alive	-	15	T1b	IB

NR, not recorded; SLNB, sentinel lymph node biopsy.

TABLE 2

Clinical features of cohort

Characteristic	Childhood melanomas, n = 12 n/N (%)	Adolescent melanomas, n = 20	Fisher exact test <i>P</i> -value
Sex			
Male	5 (41.7)	8 (40.0)	>.99
Female	7 (58.3)	12 (60.0)	
Race			
White	8 (66.7)	18 (90.)	.17
Other	2 (16.7)	1 (5.0)	.54
Not recorded	2 (16.7)	1 (5.0)	.54
Fitzpatrick skin phototype			
I/II	2 (16.7)	7 (35.)	.42
III/IV	3 (25.0)	2 (10.0)	.34
V/VI	1 (8.3)	0 (0.0)	.38
Not recorded	6 (50.0)	11 (55.0)	>.99
Tumor site			
Head, neck	3 (25.0)	5 (25.0)	>.99
Torso ^a	2 (16.7)	4 (20.0)	>.99
Extremities	7 (58.3)	10 (50.0)	.73
Noncutaneous	0 (0.0)	1 (5.0)	>.99
Other clinical features			
Lesion evolution before biopsy	9/9 (100.0)	14/14 (100.0)	>.99
First-degree family history of melanoma	0/9 (0.0)	1/19 (5.3)	>.99
Blistering sunburns	0/2 (0.0)	4/5 (80.0)	.27
Indoor tanning	0/4 (0.0)	2/7 (28.6)	.52
Significant medical comorbidity ^b	1/10 (10.0)	0/19 (0.0)	.35
History of predisposing skin lesions	1/10 (10.0) ^c	4/19 (21.1) ^d	.37

^aChest, abdomen, back, buttocks, and anogenital area.

^qIncludes diagnoses that would have been predisposing to melanoma, such as other malignancy, immunosuppression, or congenital syndrome. One child had stage IV neuroblastoma treated with chemotherapy, radiation, and hematopoietic stem cell transplantation.

^cMelanoma in one child developed in a medium congenital melanocytic nevus.

^dMelanomas developed in two adolescents in small congenital melanocytic nevi, one adolescent had an unrelated medium congenital melanocytic nevus, and one adolescent had a nevus of Ota.

TABLE 3

Comparison of histopathologic features in childhood and adolescent melanomas

Histopathologic features	Childhood melanomas	Adolescent melanomas	P-value
Breslow thickness, mm, median (range)	3.5 (0.9–85)	1.5 (0.5–36)	.20 ^a
Mitotic index, figures/mm ² , median (range)	5 (1–15)	2 (0–10)	.07 ^a
Ulceration, n/N with reported features (%)	3/9 (33.3)	3/12 (25)	>.99 ^b
Lymphovascular invasion, n/N with reported features (%)	1/8 (12.5)	1/9 (11.1)	>.99 ^b
Neural invasion, n/N with reported features (%)	2/6 (33.3)	0/9 (0)	.14 ^b

Results reported for all melanomas for which Breslow thickness, mitotic index, ulceration, lymphovascular invasion, and neural invasion were reported.

^a Mann-Whitney U-test.^b Fisher exact test.