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Fat embolism in pediatric patients: An autopsy evaluation of incidence and Etiology

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ABSTRACT

Introduction: Little is known about the incidence and etiology of fat embolism in pediatric patients. We sought to determine the incidence, time course, and associated factors of pulmonary fat embolism (PFE), cerebral fat embolism (CFE), and kidney fat embolism (KFE) in trauma and nontrauma pediatric patients at the time of autopsy.
Methods: Retrospectively, a convenience sample of consecutive pediatric patients (age, ≤10 years) who had undergone autopsy between 2008 and 2012 were evaluated for fat embolism. Patients who had no documented cause of death or who were hospital births and died during the same hospitalization were excluded. Formalin-fixed paraffin sections were reviewed by a forensic pathologist for evidence of fat embolism and nuclear elements. Autopsy reports were used to determine cause of death, injuries, resuscitative efforts taken, sex, height, weight, and age.
Results: Sixty-seven decedents were evaluated. The median age was 2.0 years (interquartile range, 0.75-4), median body mass index (BMI) was 18.0 kg/m^2 (interquartile range, 15.7-19.0 kg/m^2), and 55% of the patients were male. Pulmonary fat embolism, CFE, and KFE were present in 30%, 15%, and 3% of all patients, respectively. The incidence of PFE was not significantly different by cause of death (trauma 33%, drowning 36%, burn 14%, medical 28%). Patients with PFE but not CFE had significantly higher age, height, weight, and BMI. Half of the PFE and 57% of the CFE occurred in patients who lived less than 1 hour after beginning of resuscitation. Seventy-one percent of patients with CFE did not have a patent foramen ovale. Multivariate regression revealed an increased odds ratio of PFE based on BMI (1.244 [95% confidence interval, 1.043-1.484], $P = .015$). None of the samples evaluated demonstrated nuclear elements.
Conclusions: Pulmonary fat embolism, CFE, and KFE are common in pediatric trauma and medical deaths. Body mass index is independently associated with the development of PFE. Absence of nuclear elements suggests that fat embolism did not originate from intramedullary fat.

1. Introduction

Fat embolism (FE) can be a serious and potentially fatal event for surgical and trauma patients. It was first described in animals in the 17th century by Lower of Oxford, but was not described in humans until 1862 when Zinker described posttraumatic FE [1]. Although FE has been documented for years, the pathophysiology behind the source of these emboli as well as the physiologic ramifications of their presence remains unclear. The clinical manifestation of FE, FE syndrome (FES), was defined by Gurd in 1970 consisting of major and minor criteria. Major criteria include hypoxia, deteriorating mental status, and petechiae [2]. Minor criteria consist of tachycardia, fever, anemia, and thrombocytopenia. Fat embolism syndrome is thought to occur in approximately 0.9% to 2.2% of patients with long bone fractures; however, FE has been associated with blunt force trauma with an incidence of 47% to 100% [3–7]. Classically, there is an initial asymptomatic period of time until systemic symptoms manifest after 12 to 72 hours [2]. Recent publications have documented clinical decline, after blunt trauma, within the golden hour, demonstrating that FE and FES can occur immediately after injury in adult patients [3,8].

Traumatic FE in children is generally considered to be rare [9]. Little is known about the actual incidence of pulmonary FE (PFE) or cerebral FE (CFE) in pediatrics. Recently, an incidence at an autopsy of CFE was reported to be 2% in adults [3].

Traditionally, FE has been associated with long bone fractures or surgical instrumentation [3,9]. Several theories have been published to attempt to explain the pathophysiology behind FE. The first theory is based on the release of mechanical emboli from fractures or soft tissue injury into the venous system, which are then transported to the lungs [9,17,18,19,20]. A second theory suggests that FES is a biochemical process where inflammatory reactants, including lipoprotein lipase,

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cause the release of fatty acids, thus altering the fat transport mechanisms of the plasma. These changes in homeostasis result in fat droplet aggregation with systemic sequestration in the microvasculature and are inflammatory in nature [8,9,21]. A third less commonly described theory states that freed liquid fat from bone marrow or adipose tissue is forced into gaping venules by local increases in extravascular pressure from hemorrhage or compression [9]. Support for each theory is limited. The first theory remains the most popular because embolic elements can be seen by echocardiography during orthopedic procedures [21]. Cellular bone marrow elements have even been aspirated during, or found at autopsy after, orthopedic procedures [21,22].

Pediatric patients are unique in the fact that their bone marrow is primarily hematopoietic and the fat content is low [10]. Conventional standards of marrow cellularity specify that bone marrow biopsies are nearly 100% cellular at birth and the cellularity decreases approximately 10% in each decade of life [11]. Given this, one would expect that patients in the first decade of life should have a high incidence of hematopoietic cells associated with FE when evaluated at autopsy.

We sought to better characterize FE after traumatic and nontraumatic deaths in children. In addition, we wanted to evaluate the presence of hematopoietic cells as a marker for origin of the embolic particles.

2. Materials and methods

Pediatric forensic autopsy cases were reviewed from May 2008 through April 2012 who underwent autopsy at the Medical University of South Carolina. Institutional review board approval was sought (PRO#17241) but was ultimately determined to be unnecessary because the project met the Not Human Research criteria set forth by the Code of Federal Regulations (45CFR46).

Retrospectively, a convenience sample of consecutive pediatric patients (age, ≤10 years) who had undergone autopsy were evaluated for evidence of pulmonary, kidney, and brain FE. Only autopsies of individuals younger than 10 years were used to focus on young patients. Cases were evaluated for cause of death as well as evidence of resuscitation. Cases were excluded if the cause of death was unknown, patients had significant decomposition limiting full evaluation at autopsy, or patient was declared brain dead and organ donation was performed. Patients were also excluded if they had not been discharged home after birth or were diagnosed as having genetic syndrome or cardiac flow abnormality. This was done in an attempt to capture children with normal physiology and metabolism.

The preexisting hematoxylin and eosin–stained glass slides were retrieved from storage and reviewed by a forensic pathologist blinded to the case category. However, other evidence of trauma may have been apparent on the glass slides. The pathologist reviewed the glass slides in order to identify clear round spaces without evidence of erythrocytes, platelets, or fibrin within vascular lumens consistent with and suggestive of fat emboli. Areas around and within the clear round spaces were also evaluated for nuclear elements consistent with hematopoetic cell lines. In evaluating these sections, the presence of these findings was noted as a dichotomous variable. The pathologist reviewed lung, brain, and kidney sections as available for each case. Demographic data, injury data, and cause of death were extracted from the coroner report for each patient. Medical interventions including the performance of cardiopulmonary resuscitation (CPR) were also documented.

Descriptive statistics were used to report the incidence of PFE. Continuous data were analyzed using Student t test if normally distributed or Mann–Whitney U if skewed. Dichotomous data were compared using Pearson χ^2 of Fisher exact test. All variables with $P <$ .2 were included in a backward Wald multivariate regression to determine independent factors associated with PFE. All statistical analyses were performed using SPSS version 13.0 (Chicago, IL). Statistical significance was determined using a P value less than .05.

3. Results

Over a period of 4 years, 67 decedents were evaluated for PFE, kidney FE (KFE), and CFE. The median age was 2.0 years (interquartile range [IQR], 0.75-4 years), median body mass index (BMI) was 18.0 kg/m^2 (IQR, 15.7-19.0 kg/m^2), and 55% of the patients were male. The time from initial insult to declaration of death was a median of 54 minutes (IQR, 0-1020 minutes). Cardiopulmonary resuscitation was performed on 63% and intraosseous access lines (IO lines) were placed in 39% of patients. Pulmonary FE, CFE, and KFE were present in 30%, 15%, and 3% of all patients, respectively. The cause of death was divided into 4 groups consisting of trauma (21/67; blunt 17, penetrating 4), drowning (14/67), burn (7/67), and medical (25/67).

The incidence of PFE was not significantly different by cause of death (trauma 33%, drowning 36%, burn 14%, medical 28%; $P = .752$). Representative sections of lung with fat emboli are presented in Figs. 1 and 2. Patients with PFE had significantly higher age, height, weight, and BMI (Table 1). Pulmonary FE was not associated with location of death or resuscitation attempts (Table 2). Half (10/20) of the patients with PFE died within 1 hour of insult.

Of the traumatically injured patients, PFE was associated weight and BMI. There was no association between location of death and resuscitation efforts (Table 3). Three patients with PFE died within 1 hour of injury. No specific fracture (Fx) was associated with PFE (Table 4). None of the penetrating trauma patients or automobile vs pedestrian patients had PFE present. All patients involved in motor vehicle collisions (5/5) had PFE, whereas only 30% (2/6) patients had PFE after homicide due to blunt force trauma to the head.

The cause of death in the medical group was divided between asthma (5/25), bacterial infection (10/25), viral infection (5/25), central nervous system insult (4/25), and cardiac (1/25). Pulmonary FE was divided among all of the groups, except bacterial infection. The incidence of PFE in each groups was as follows: asthma 60% (3/5), bacterial infection 0% (0/10), viral infection 20% (1/5), central nervous system insult 50% (2/4), and cardiac 100% (1/1). Pulmonary FE was associated with height and weight but not with location of death or resuscitation efforts (Table 5). Three patients with PFE died within 1 hour of initial insult.

In patients after drowning, we found PFE to be present in 36% of patients and 14% of burn patients. Only 1 patient who drowned with PFE was noted to have a cerebral contusion and no other evidence of trauma and did not receive CPR. All the other patients with PFE had CPR performed, and 1 had evidence of contusions on the body. The only patient who was burned with PFE was also noted to have contusions of the scalp, thorax, and extremities.

Multivariate regression performed on the entire cohort revealed an increased odds ratio of PFE in patients based on BMI (1.244 [95%

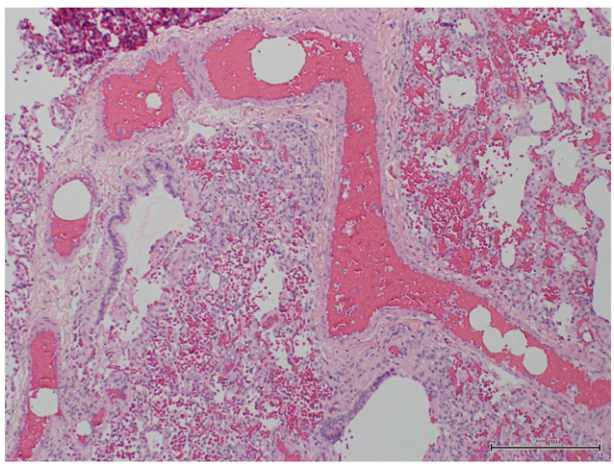

Fig. 1. Large FE within pulmonary artery in the lung.

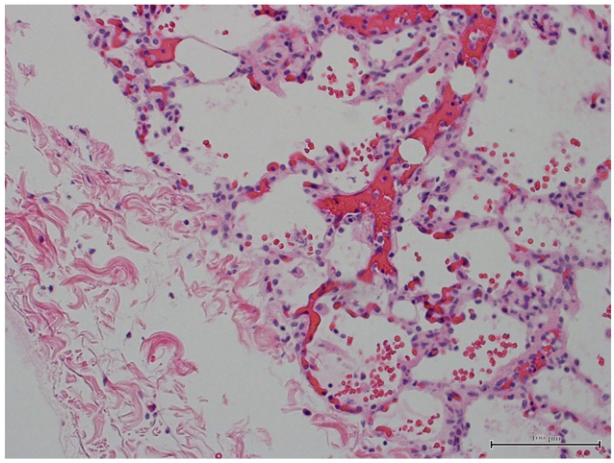

Fig. 2. Fat embolism within capillary in the lung.

confidence interval {CI}, 1.043-1.484], $P = .015$) only. A regression performed on only trauma patients revealed BMI with an increased odds ratio as well (3.791 [95% CI, 1.152-12.476], $P = .028$). A regression performed on medical patients only revealed height to have a significant odds ratio (1.202 [95% CI, 1.009-1.432], $P = .039$).

Cerebral FE occurred less than PFE. Representative sections of brain with fat emboli are presented in Fig. 3. There was no difference in the incidence of CFE based on type of death: trauma 10% (2/19), drowning 30% (3/10), burn 0% (0/2), and medical 12% (2/17). Not all cases had brain slides available for review because they were not created as part of the autopsy. Cerebral FE was not associated with any patient specific factors: sex, age, height, weight, BMI, time from insult to death, location of death, CPR, or IO lines. Most individuals with CFE 57% (4/7) lived less than 1 hour after beginning of resuscitation.

Seventy-one percent of patients with CFE did not have a patent foramen ovale. One individual had PFE and CFE; however, 6 individuals had CFE without PFE and 15 individuals had PFE without CFE. In addition, none of the samples evaluated in any organ system demonstrated nuclear elements suggestive of being bone marrow origin.

4. Discussion

Fat embolism is common after both traumatic and nontraumatic deaths in children; however, identifying the source of the embolic fat has been difficult to determine. Traditional teaching maintains that FE originates from medullary fat after fracture particularly of long bones [2,9,10]. Recent reports have questioned the origin of this fat [3]. Because we failed to note any cellular elements from bone marrow, in our young population, this increases the evidence that actual marrow embolization is not the source of FE noted without orthopedic surgical instrumentation. An analogy can be made between these findings and the results of a rabbit femur crush study to produce FE. In this study, FE could be reliably created in older animals but not in younger animals [12]. Further studies are warranted to better define FE resulting from injury as well as surgical intervention.

Table 1
Characteristics of patients with PFE

	PFE, yes	PFE, no	%	*P*
Total	20	47	30	
Sex (male)	50% (10)	43% (20)		.575
Age (y)	3 (1.6-5)	2 (0.5-4)		.045
Height (cm)	95.9 (83.0-115.1)	84 (66-101.6)		.048
Weight (kg)	17.9 (12.3-22.7)	13.6 (7.3-18.1)		.016
BMI (kg/cm^2)	19.5 (17.1-21.0)	17.2 (15.3-18.9)		.008
Time (min)	66.5 (10-1142)	53 (0-1020)		.556

Table 2
PFE, location of death, and resuscitation interventions

	PFE, yes (n = 20)	PFE, no (n = 47)	*P*
CPR (n = 42)	60% (12)	64% (30)	.767
IO line (n = 26)	45% (9)	36% (17)	.497
Dead on arrival (n = 19)	25% (5)	30% (14)	.774
Died in field (n = 13)	20% (4)	19% (9)	.936
Died in emergency department (n = 32)	45% (9)	49% (23)	.796
Died in hospital (n = 21)	35% (7)	30% (14)	.674

In our pediatric patients, the incidence of FE in both traumatic and medical deaths is less than has been described in adults. After traumatic injury, 33% of patients were noted to have evidence of PFE. This is much less than was noted in a similar autopsy study of adult patients which showed that 83% of trauma patients had PFE [3]. Our findings were consistent with 2 recent publications finding no single injury or that fracture was associated with PFE [3,16]. Mechanism of injury had not been considered extensively in the published literature on FE. Given the lack of hematopoietic elements associated with the FE noted in this study, the fat of origin after traumatic injury may be subcutaneous or fatty acid in origin.

Similarly to the trauma cases, PFE occurred in 28% of the medical cases reviewed. This is less than the 63% reported from adult autopsy evaluation [3]. Almost every cause of death evaluated showed PFE in some cases. In the study by Eriksson et al [3], 88% of adult medical deaths evaluated at autopsy who received CPR also had PFE. Of the patients who received CPR, 3 of 8 had evidence of rib fractures and 1 of 8 was noted to have a fractured sternum [3]. We found no association with CPR in this pediatric population. One potential explanation of this difference is the adequacy of regaining circulation in the 2 groups. If circulation is not recovered after insult, embolization cannot occur. Often for many reasons, CPR is performed on children until they reach the hospital to give them any chance for survival, whereas this may not hold true in adults.

In addition to the differences between pediatric patients and adult patients, this is the first case series of burns and drownings to describe FE and PFE. Burns have been referenced as a potential population that could have FE but only based on limited case reports [14]. In patients after drowning, we found PFE to be present in 36% of patients and 14% of burn patients. Only 1 patient who drowned with PFE was noted to have a cerebral contusion; however, all the other patients with PFE had CPR performed. The only patient who was burned with PFE was also noted to have contusions of the scalp, thorax, and extremities. A further evaluation of this patient population is warranted to further describe this finding.

Many more pediatric patients were found to have CFE than has been described previously in adults. A recent adult autopsy evaluation found

Table 3
PFE in trauma

	PFE, yes (n = 7)	PFE, no (n = 14)	*P*
Total	33%	67%	
Sex (male)	43% (3)	57% (8)	.575
Age (y)	3 (1.5-5)	1.8 (0.6-3.3)	.197
Height (cm)	95.3 (83-116)	84.6 (74-100)	.322
Weight (kg)	18.1 (14.0-22.7)	13.3 (8.3-16.7)	.067
BMI (kg/cm^2)	20.0 (19.13-20.5)	17.1 (15.5-18.6)	.002
Time (min)	153 (0-210)	40 (0-1440)	.913
Blunt	100% (7)	71% (10)	.255
CPR	43% (3)	43% (6)	1.000
IO line	28% (2)	28% (4)	1.000
Dead on arrival	43% (3)	43% (6)	1.000
Died in field	43% (3)	21% (3)	.354
Died in emergency department	14% (1)	36% (5)	.613
Died in hospital	42% (3)	42% (6)	1.000

Table 4
Associated traumatic injuries with PFE

	YES PFE (n = 7)	No PFE (n = 14)	*P*
Traumatic brain injury	71% (5)	100% (14)	.100
Facial Fx	14% (1)	14% (2)	1.000
Rib Fx	14% (1)	29% (2)	.624
Pulmonary contusion	57% (4)	21% (3)	.156
Spleen	29% (2)	7% (1)	.247
Kidney	29% (2)	7% (1)	.247
Any fracture	71% (5)	50% (7)	.642

*All of the following had less than 2 occurrences in the data set: skull Fx, C-spine Fx, T-spine Fx, L-spine Fx, sternal Fx, cardiac injury, liver injury, bowel injury, pelvic injury, bladder injury, clavicle Fx, scapula Fx, humerus Fx, radius Fx, hand Fx, femur Fx, tibia Fx, and foot Fx.

that 2% of patients had evidence of CFE [3]. Several authors have noted that FE can occur and pass through the pulmonary circulation and be noted in the systemic circulation [3,8,9]. A high clinical suspicion is warranted in pediatric patients. The reason for a higher incidence in children will require further evaluation.

Height, weight, and BMI were found to be associated with PFE. In this evaluation, taller, heavier, and higher BMI increased the risk of finding FE. This is contrary to the findings in adults where lower weight patients (in kilograms) were more likely to have PFE (86.5 ± 17.5 vs 108.8 ± 28.6) [3]. This apparent difference likely has several factors contributing to this finding. As has been described young rabbits, they are less likely to produce FE than older rabbits [12]. This may be a physiologic reason, associated with age, like degree of hematopoiesis in bones or degree of fat in different storage locations. Older animals and people have more subcutaneous fat in general. If the fat in FE comes from subcutaneous stores entering venules from either external compression of liquid fat or direct entry from adipose injury, there may be an optimal subcutaneous fat deposition for each patient that places them at the highest risk for development of FE. The sheer strain between skin and underlying muscle and skeleton would be more diffuse in obese adult patients. Most of the children in this study were not overweight (median BMI, 18 kg/m^2). Further studies should include evaluations of body composition and degree of subcutaneous adiposity as risk factors for FE.

Physiologically, these FEs may be of great consequence in the treatment for these patients. Half of the patients with PFE and 57% of patients with CFE died within 1 hour of initial insult. This provides additional evidence that FE occurs early in the resuscitative phase for these critically injured patients. This is similar to a recent autopsy report of trauma patients that noted that 80% had evidence of FE within the "golden hour" of trauma [3]. The early presence of this embolic load poses serious potential physiologic changes. Theoretically, the emboli increase the perfusion pressures of the lungs, which results in right heart strain without adequate preload [13]. This is particularly dangerous in trauma patients who may also be experiencing hemorrhagic shock.

Table 5
PFE in medical cases

	PFE, yes (n = 7)	PFE, no (n = 18)	*P*
Total	28%	72%	
Sex (male)	57% (4)	50% (9)	1.000
Age (y)	3.5 (1.2-7)	1.25 (0.2-2.6)	.055
Height (cm)	108 (79-132)	70.0 (51.9-95.4)	.017
Weight (kg)	19.5 (12.2-43.5)	9.4 (3.5-18.1)	.021
BMI (kg/cm^2)	29.5 (18.0-24.9)	17.5 (14.6-19.8)	.158
Time (min)	74 (44-1440)	57 (44-1125)	.745
CPR	71% (5)	78% (14)	1.000
IO line	57% 4)	33% (6)	.378
Dead on arrival	14% (1)	11% (2)	1.000
Died in field	0% (0)	6% (1)	1.000
Died in emergency department	71% (5)	61% (11)	1.000
Died in hospital	29% (2)	28% (5)	1.000

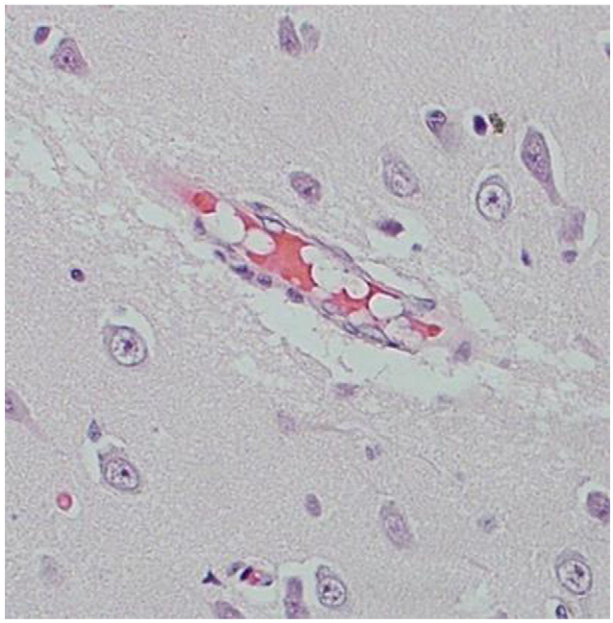

Fig. 3. Fat embolism within a capillary in the brain.

Several limitations must be considered when evaluating these data. First of all, not every death receives an autopsy, so this may represent a unique population with a different rate from that of the general population. Given the small sample size and large number of patients with PFE, one cannot infer causality between individual injuries and the development of PFE. In addition, a single forensic pathologist was used to evaluate and read the tissue slides which may contribute to some observer bias. A review of routinely processed histologic slides to identify and quantify FE is uniquely hindered by the fact that such processing removes the fat. Thus, the pathologist infers the presence of FE by the identification of clear round spaces within vessel lumens. This method of detecting FE, however, has been evaluated in the past. In evaluating for FE during the Korean conflict, Scully [15] found that the incidence of FE by hematoxylin and eosin was 79% compared with slides stained with Oil Red O (93%). The incidence we found may be lower than the actual incidence based on the report from Korea. Future prospective studies may be designed to eliminate the first issue either by using fresh-frozen tissue or by using an alternative processing regimen that retains fat. As for the identification of the fat identified, electron microscopic imaging may lend information with resolving power to better than 50 pm (magnification up to approximately ×10 000 000) as opposed to the 200-nm resolution possible with light microscopy (up to approximately ×2000). The performance of CPR was determined by medical record documentation and may not include bystander CPR for patients declared at home. Similarly, the effectiveness of CPR could not be determined from the records, and restoration of circulation could not be evaluated. Last, the injuries noted were determined form the autopsy record and minor contusions or minor injuries may not be evident in the reports.

5. Conclusions

Pulmonary FE, CFE, and KFE are common in pediatric traumatic and medical deaths regardless of cause of death. In this series, BMI was independently associated with the development of PFE. The absence of nuclear elements and the presence of emboli in both circulations suggest that FE did not originate from intramedullary fat. This study contributes to the understanding of PFE, CFE, and KFE, raising questions about the origin of this pathologic entity. In addition, this case series highlights several potential differences in the incidence of FE in pediatric and adult patients after traumatic and medical deaths. Additional studies

are warranted to determine the origin and additional risk factors associated with PFE, CFE, and KFE to determine when fat emboli occur and their contribution to deranged physiology.

Author contributions

Evert Eriksson initiated the study proposal, submitted the institutional review board proposal, conducted the chart review, completed the statistical analysis, and coordinated the writing and editing of the manuscript.

Joshua Rickey helped develop the study aims and assisted with the chart review and critical review of the manuscript.

Stuart Leon helped develop the study aims, assisted with chart review, and assisted with data analysis and critical review of the manuscript.

Christian Minshall helped develop the study aims and assisted with data analysis and critical review of the manuscript.

Samir M. Fakhry helped develop the study aims and assisted with data analysis and critical review of the manuscript.

Cynthia Schandl helped develop the study aims, reviewed all pathology slides, and assisted in writing and editing of the manuscript.

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