VOLUME 101 | SUPPLEMENT 1 | MARCH 2021

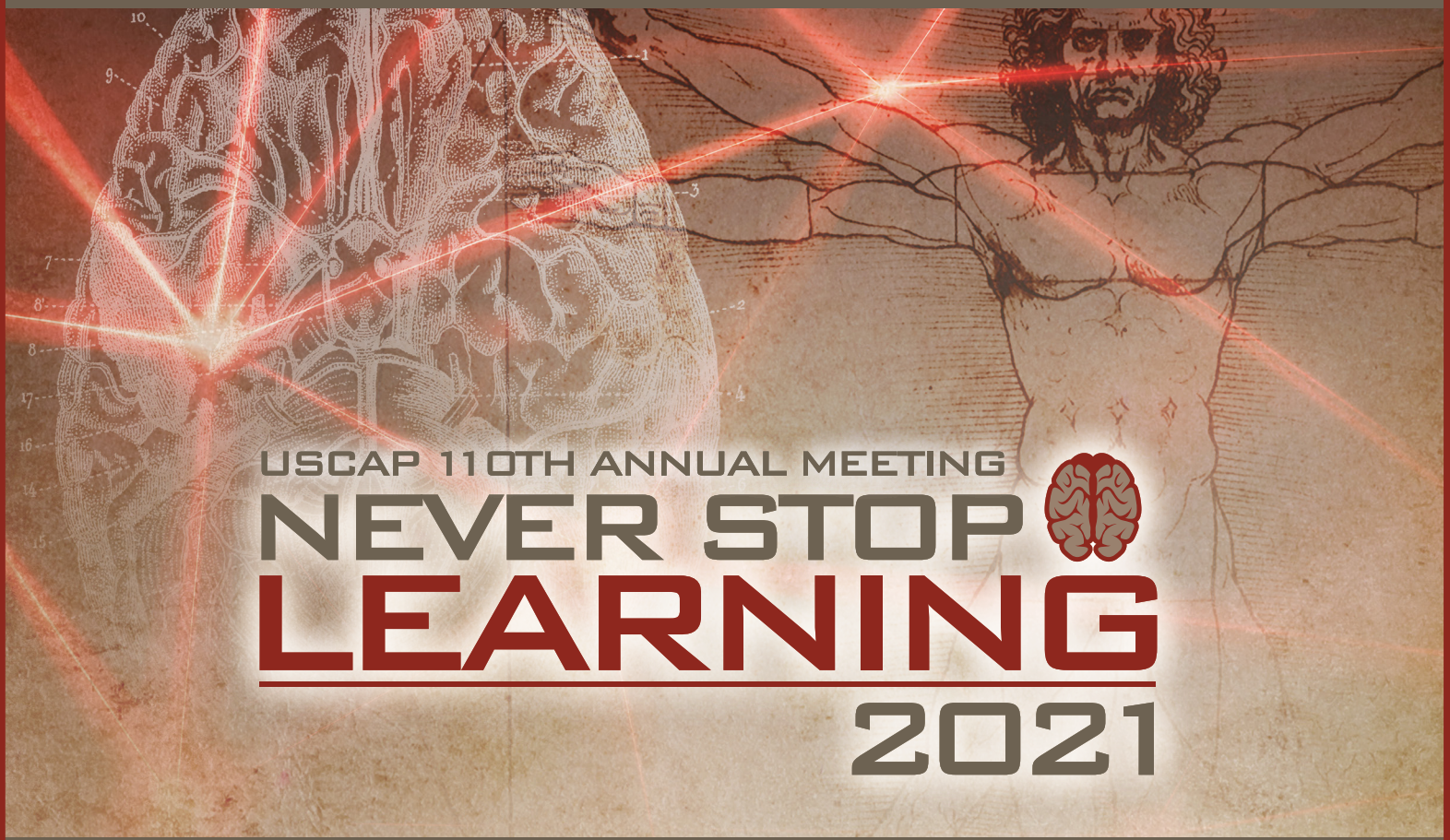
LI

LABORATORY INVESTIGATION

THE BASIC AND TRANSLATIONAL PATHOLOGY RESEARCH JOURNAL

ABSTRACTS

PEDIATRIC AND PERINATAL PATHOLOGY (903-909)



MARCH 13-18, 2021 | VIRTUAL AND INTERACTIVE

Published by
SPRINGER NATURE
www.ModernPathology.org

USCAP
Creating a Better Pathologist
AN OFFICIAL JOURNAL OF THE UNITED STATES AND CANADIAN ACADEMY OF PATHOLOGY

EDUCATION COMMITTEE

Jason L. Hornick
Chair

Rhonda K. Yantiss, Chair
Abstract Review Board and Assignment Committee

Kristin C. Jensen
Chair, CME Subcommittee

Laura C. Collins
Interactive Microscopy Subcommittee

Raja R. Seethala
Short Course Coordinator

Ilan Weinreb
Subcommittee for Unique Live Course Offerings

David B. Kaminsky
(Ex-Officio)
Zubair W. Baloch
Daniel J. Brat
Sarah M. Dry
William C. Faquin
Yuri Fedoriw
Karen Fritchie
Jennifer B. Gordetsky
Melinda Lerwill
Anna Marie Mulligan
Liron Pantanowitz
David Papke,
Pathologist-in-Training
Carlos Parra-Herran
Rajiv M. Patel
Deepa T. Patil
Charles Matthew Quick
Lynette M. Sholl
Olga K. Weinberg
Maria Westerhoff
Nicholas A. Zoumberos,
Pathologist-in-Training

ABSTRACT REVIEW BOARD

Benjamin Adam
Rouba Ali-Fehmi
Daniela Allende
Ghassan Allo
Isabel Alvarado-Cabrero
Catalina Amador
Tatjana Antic
Roberto Barrios
Rohit Bhargava
Luiz Blanco
Jennifer Boland
Alain Borczuk
Elena Brachtel
Marilyn Bui
Eric Burks
Shelley Caltharp
Wenqing (Wendy) Cao
Barbara Centeno
Joanna Chan
Jennifer Chapman
Yunn-Yi Chen
Hui Chen
Wei Chen
Sarah Chiang
Nicole Cipriani
Beth Clark
Alejandro Contreras
Claudiu Cotta
Jennifer Cotter
Sonika Dahiya
Farbod Darvishian
Jessica Davis
Heather Dawson
Elizabeth Demicco
Katie Dennis
Anand Dighe
Suzanne Dintzis
Michelle Downes
Charles Eberhart
Andrew Evans
Julie Fanburg-Smith
Michael Feely
Dennis Firchau
Gregory Fishbein
Andrew Folpe
Larissa Furtado
Billie Fyfe-Kirschner
Giovanna Giannico
Christopher Giffith
Anthony Gill
Paula Ginter
Tamar Giorgadze
Purva Gopal
Abha Goyal
Rondell Graham
Alejandro Gru
Nilesh Gupta
Mamta Gupta
Gillian Hale
Suntrea Hammer
Malini Harigopal
Douglas Hartman
Kammi Henriksen
John Higgins
Mai Hoang
Aaron Huber
Doina Ivan
Wei Jiang
Vickie Jo
Dan Jones
Kirk Jones
Neerja Kambham
Dipti Karamchandani
Nora Katabi
Darcy Kerr
Francesca Khani
Joseph Khoury
Rebecca King
Veronica Klepeis
Christian Kunder
Steven Lagana
Keith Lai
Michael Lee
Cheng-Han Lee
Madelyn Lew
Faqian Li
Ying Li
Haiyan Liu
Xiuli Liu
Lesley Lomo
Tamara Lotan
Sebastian Lucas
Anthony Magliocco
Kruti Maniar
Brock Martin
Emily Mason
David McClintock
Anne Mills
Richard Mitchell
Neda Moatamed
Sara Monaco
Atis Muehlenbachs
Bita Naini
Dianna Ng
Tony Ng
Michiya Nishino
Scott Owens
Jacqueline Parai
Avani Pendse
Peter Pytel
Stephen Raab
Stanley Radio
Emad Rakha
Robyn Reed
Michelle Reid
Natasha Rekhtman
Jordan Reynolds
Andres Roma
Lisa Rooper
Avi Rosenberg
Esther (Diana) Rossi
Souzan Sanati
Gabriel Sica
Alexa Siddon
Deepika Sirohi
Kalliopi Siziopikou
Maxwell Smith
Adrian Suarez
Sara Szabo
Julie Teruya-Feldstein
Khin Thway
Rashmi Tondon
Jose Torrealba
Gary Tozbikian
Andrew Turk
Evi Vakiani
Christopher VandenBussche
Paul VanderLaan
Hannah Wen
Sara Wobker
Kristy Wolniak
Shaofeng Yan
Huihui Ye
Yunshin Yeh
Anjana Yeldandi
Gloria Young
Lei Zhao
Minghao Zhong
Yaolin Zhou
Hongfa Zhu

To cite abstracts in this publication, please use the following format: **Author A, Author B, Author C, et al. Abstract title (abs#). In "File Title."** ***Laboratory Investigation*** **2021; 101 (suppl 1): page#**

903 Expanding the Concept of Clonal Transient Neoplasms: Nine Cases of Cranial Fasciitis in Children with Peculiar Clinical Course

Jessica Alvarez Lesmes[1], Siraj El Jamal[2], Faizan Malik[3], Anas Bernieh[4], Ali Saad[5]
[1]UMH/JMH, Miami, FL, [2]Icahn School of Medicine at Mount Sinai, New York, NY, [3]The University of Tennessee Health Science Center, Memphis, TN, [4]Cincinnati Children's Hospital Medical Center, Cincinnati, OH, [5]University of Miami Miller School of Medicine, Miami, FL

Disclosures: Jessica Alvarez Lesmes: None; Siraj El Jamal: None; Faizan Malik: None; Anas Bernieh: None; Ali Saad: None

Background: Cranial fasciitis (CF) is a benign myofibroblastic proliferation mostly of children. It presents as a rapidly growing scalp mass and characterized by a peculiar growth cycle displaying a rapid growth phase followed by a relative growth slowdown and even growth arrest. Several examples of tumor regression and resolution have been reported. This growth pattern has been recently explored and coined “transient neoplasia”. We report the clinicopathological and molecular findings of 9 cases of CF in children with emphasis on their peculiar growth pattern.

Design: The files of multiple institutions were searched for CF in children. To evaluate the behavior of the tumor, only patients with subtotal resection (STR) were included. Demographics and clinical history retrieved from medical records and physicians’ notes. The histology and immunophenotype were reviewed for the accuracy of the diagnosis.

Results: The search resulted in 6 males and 3 females (mean age 2.8 years; range 0.8-5 years). All patients presented with scalp mass. The tumor was present for a mean 3.5 months before presentation (range 1-9 months). The tumor was rapidly growing in 4 patients and slow growing in 5. The most common location was the temporal region (4 patients). The tumor size ranged from 3.5-10.6 cm (mean 6.2 cm). The tumor was painful in 2 patients. A history of head trauma was present in 2 patients and one patient reported history of radiation for medulloblastoma. Seven patients showed periosteal invasion. In 2 patients, the tumor was adherent to the dura and in 3 patients it appeared arising from the dura.

USP6 gene rearrangement (performed by fluorescence in situ hybridization) was detected in 6 patients. It was not done in 2 cases and one case was negative.

Follow-up showed the tumor was stable in 5 patients after a mean follow-up of 15.2 months (range 10-23 months). The tumor regressed in 4 patients (mean 16.5 months, range: 8-26 months). In none of the patients, the tumor continued to grow.

Conclusions: This is the first description of the peculiar clinical course of CF in children. This clinical course has been recently described in other lesions such as nodular fasciitis (the extracranial counterpart of CF), aneurysmal bone cyst, and myositis ossificans. Interestingly, these entities share overlapping histomorphology and association with *USP6* rearrangement. Awareness of this clinical course is important to avoid extensive, sometimes disfiguring, surgical excisions.

904 Comparative Study Of Placental Pathologic Findings In Early-Onset And Late-Onset Preeclampsia

Rabia Bhalli[1], Jorge Sepulveda[2], Stephanie Barak[1]
[1]George Washington University, Washington, DC, [2]George Washington University Hospital, Washington, DC

Disclosures: Rabia Bhalli: None; Jorge Sepulveda: None

Background: Preeclampsia (PEC) is a common and potentially lethal multisystem disorder of pregnancy defined by new onset of hypertension and proteinuria after 20 weeks of gestation or development of signs of end organ dysfunction (hepatic, renal and cerebral). Its presentation is heterogeneous with early onset form (EO-PEC) arising before 34 weeks of gestation with associated high rate of fetal growth restriction, and late onset form (LO-PEC)

after 34 weeks that can have maternal consequences. The pathophysiology of PEC is poorly understood with unpredictable onset. EO-PEC is thought to be related to poor placentation and abnormal vascular remodeling, whereas LO-PEC may be related to intrinsic processes restricting intervillous perfusion. In this study, we look at the placental pathologic differences between EO-PEC and LO-PEC as potential markers of different pathophysiologic pathways.

Design: We compared the pathologic findings in placentas with EO-PEC (n=13) and LO-PEC (n=86) using the Amsterdam Placental workshop group consensus criteria defined in 2014. We looked at different categories: maternal vascular malperfusion (MVM), implantation site abnormalities, inflammatory-immune processes, villous maldevelopment, fetal vascular malperfusion (FVM), and other miscellaneous findings. We included patients with only true preeclampsia and excluded superimposed chronic hypertension, gestational diabetics, metabolic disorders or twin pregnancies to remove confounding factors. Chi-squared with Bonferroni correction was used for statistical analysis.

Results: The EO-PEC group (n=13) had a higher rate of placentas lesions associated with MVM than the LO-PEC (n= 86) (100% vs 51%, P=0.018) and more implantation site abnormalities (62% vs 19%, P=0.015). Overall, accelerated maturation, placental infarcts and decidual arteriopathy with or without fibrinoid necrosis/atherosis were more commonly seen in EO-PEC (p<0.02). Chronic inflammatory/immune lesions were seen in only 12% of LO-PEC and 23% of EO-PEC.

	Early-onset (n=13)(%)	Late onset (n=86)(%)
Maternal vascular malperfusion	**13 (100%)**	**44 (51%)**
Placental infarct	**10 (77%)**	**24 (16%)**
Distal villous hypoplasia	2 (15%)	14 (16%)
Accelerated maturation	**8 (62%)**	**4 (5%)**
Increased syncytial knots	3 (23%)	5 (6%)
Villous agglutination	2 (15%)	15 (17%)
Implantation site abnormality	**8 (62%)**	**16 (19%)**
Decidual vasculopathy (persistent muscularization of spiral arteries)	**8 (62%)**	**16 (13%)**
Fibrinoid necrosis / Atherosis	**5 (38%)**	**2 (12%)**

Table 1: Pathologic changes associated with Maternal vascular perfusion

And Implantation side abnormality in EO-PEC and LO-PEC

(statistically significant p<0.0002)

Figure 1 - 904

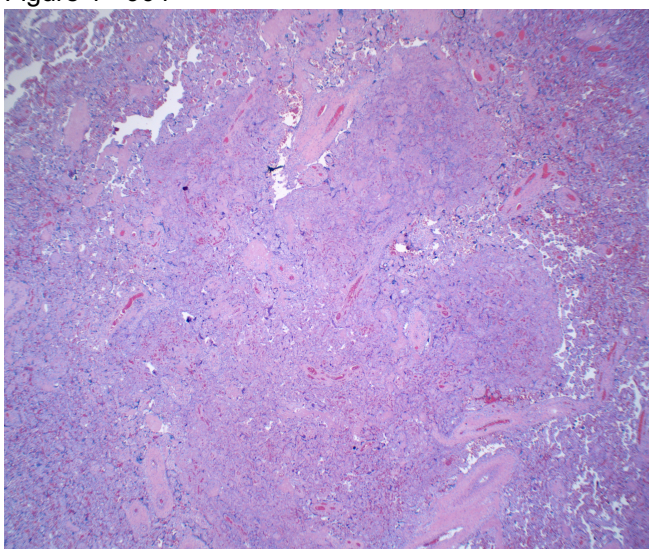
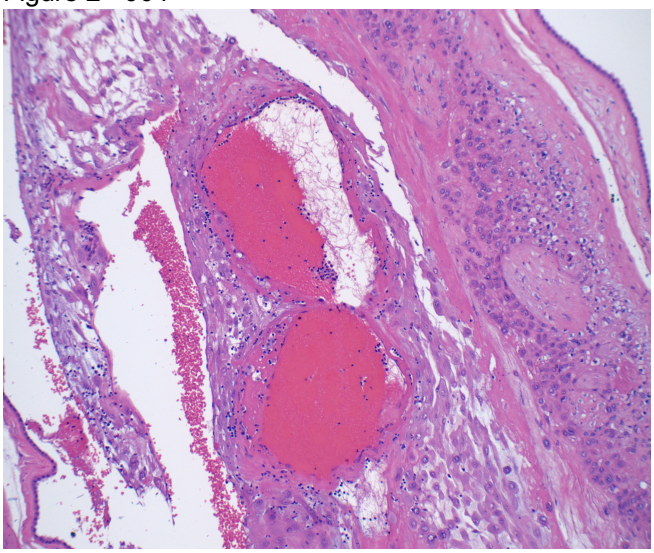


Figure 2 - 904



Conclusions: EO-PEC and LO-PEC have different placental pathology, most likely reflecting different mechanisms. Our findings are in keeping with the literature supporting that EO-PEC may be related to poor placentation with implantation abnormality, resulting in placental malperfusion. There are probably two different pathways for development of EO-PEC and LO-PEC. We did not find a significant relation between chronic inflammatory/immune conditions and LO-PEC. Additional studies are needed to compare the pathologic findings of LO-PEC to a cohort of uncomplicated gestations and identify potential significant findings.

905 Cytogenetic Abnormalities in Products of Conception Samples from Cases of Spontaneous Miscarriage: An Institutional Experience

Cameron Felty[1], Christopher Jackson[1], Elizabeth Melchionna[2], Michelle Bickford[1], Mohammad Azim[1], Michael Baker[1], Wahab Khan[1]

[1]Dartmouth-Hitchcock Medical Center, Lebanon, NH, [2]Dartmouth-Hitchcock Medical Center, Lebanon, PA

Disclosures: Cameron Felty: None; Christopher Jackson: None; Elizabeth Melchionna: None; Michelle Bickford: None; Mohammad Azim: None; Michael Baker: None; Wahab Khan: None

Background: Spontaneous miscarriage occurs in up to 15% of clinically recognized pregnancies, and cytogenetic abnormalities are identified in 50% of cases. Risk factors include advanced maternal age, substance use, prior pregnancy loss, obesity, and diabetes. Metaphase chromosome analysis allows for a crude genome-wide view of the ploidy state in products of conception (POC) specimens. We compared the relative and categorical frequency of cytogenetic abnormalities identified in POC samples at our institution as part of a quality assurance initiative. We also examined maternal clinical data to identify risk factors associated with miscarriage.

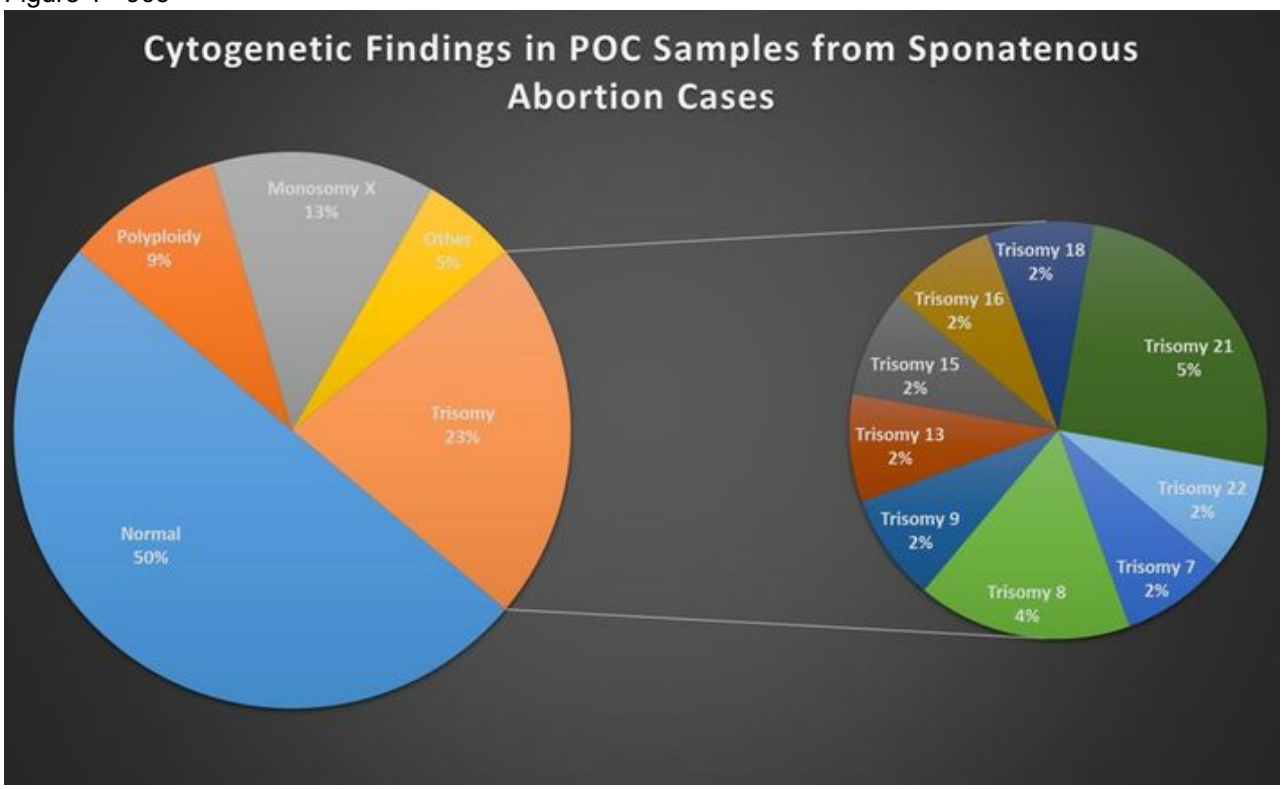
Design: 214 POC specimens underwent karyotype analysis from January 2014-May 2019. Cases not meeting criteria for spontaneous abortion (gestational age >20 weeks and/or induced termination) were excluded. Karyotype, maternal age, body mass index, history of hypertensive disorder and/or diabetes mellitus, history of tobacco and/or drug use, history of recurrent miscarriages, and chromosomal microarray (CMA) data were recorded where available.

Results: 54 cases met inclusion criteria, of which 27 (50%) had a cytogenetic abnormality. Females accounted for 19/27 of cases with an apparently normal karyotype. In cases with an abnormal karyotype, trisomy was the most common finding (23%) followed by monosomy X (13%), polyploidy (9%), and other (5%) (**Figure 1**). The “other” category included one case with multiple gains/losses not related to common aneuploidies, a structural aberration on X, and a case with a pathogenic loss of 15q11.2 identified by CMA. Spontaneous abortion tended to occur at earlier gestation when a cytogenetic abnormality was present (11.2 weeks vs. 14.2 weeks, p=0.01, **Table 1**). A

history of recurrent miscarriage was prevalent in cases with a cytogenetic abnormality, though this did not reach statistical significance (74% versus 48%, p=0.09).

	Normal Karyotype (n=27)	Cytogenetic Abnormality (n=27)	p value
Maternal age (years)	31.6	31.4	0.92
Body mass index (kg/m^2)	29.6	28	0.38
Gestational age (weeks)	14.2	11.2	0.01
Tobacco exposure	4/27 (15%)	4/27 (15%)	1.00
Drug exposure	2/26 (8%)	3/24 (13%)	0.66
Hypertensive disorder	3/27 (11%)	3/27 (11%)	1.00
Diabetes mellitus	0/27 (0%)	0/27 (0%)	1.00
Recurrent Miscarriage	13/27 (48%)	20/27 (74%)	0.09
In vitro fertilization (IVF)	1/26 (4%)	1/27 (4%)	1.00

Figure 1 - 905



Conclusions: The relative and categorical frequencies of cytogenetic abnormalities reported in POC specimens at our institution are in agreement with established literature. We found that spontaneous abortions occurred earlier in gestation when cytogenetic abnormalities were present. Though a history of recurrent miscarriage was prevalent in cases with a cytogenetic abnormality, this did not reach statistical significance. This suggests that other underlying maternal or subchromosomal mechanisms could be responsible for most recurrent miscarriages.

906 Comparison of the Autopsy and Placental Findings in Second Versus Third Trimester Pregnancy Loss

Kiran Manjee[1], Linda Ernst[1]
[1]*University of Chicago at NorthShore HealthSystem, Evanston, IL*

Disclosures: Kiran Manjee: None; Linda Ernst: None

Background: Second-trimester pregnancy loss is defined as a pregnancy loss between 14 and 0/7 weeks and 27 and 6/7 weeks gestation. The pathology of second-trimester fetal loss is not well-characterized due to lack of comprehensive autopsy studies. Many cases are attributed to infections or genetic abnormalities. The purpose of this study is to describe the pathology of second-trimester pregnancy loss and compare it with causes of third-trimester pregnancy loss.

Design: In this retrospective study, 68 second-trimester and 54 third-trimester stillborn, intact fetal autopsies performed in-house with complete fetal and placental examination were included. Autopsy reports were reviewed to gather maternal demographics and information about fetal gestational age, gender, body and placental weight, congenital anomalies, and immediate and underlying cause of death. The immediate cause of death was coded 'probable' or 'possible' according to Initial Causes of Fetal Death (INCODE). Statistical analyses were performed using IBM SPSS Statistics22.

Results: At least one probable cause of death was identified in 59/68 (87%) second-trimester and 44/54 (81%) third-trimester fetal autopsy cases. Overall, pathologic placental conditions led to the fetal loss in 70/122 (57%) cases. 42/68 (62%) second-trimester and 28/54 (52%) third-trimester fetuses had probable cause of death secondary to placental pathology. Among placental causes, 29/42 (69%) second-trimester and 14/28 (50%) third-trimester fetal losses were related to compromised fetal microcirculation with umbilical cord abnormality, most commonly umbilical cord hypercoiling (p=0.055). There were no other significant differences between second and third-trimester causes of fetal loss (Table). Pregnancy losses secondary to early amnion rupture sequence, aneuploidy and complications of multiple gestations were only seen in second-trimester.

'Probable' Cause of Death by INCODE Category	Total N=122	Second Trimester N=68	Third Trimester N=54
Maternal Medical Conditions during Pregnancy (INCODE Category 1)	**2 (2%)**	**0 (0%)**	**2 (4%)**
Hypertensive disease with antepartum clinical diagnosis of abruption with retroplacental clot or pathologic confirmation with extensive parenchymal infarction	2 (2%)	0 (0%)	2 (4%)
Obstetric Complications (INCODE Category 2)	**11 (9%)**	**4 (6%)**	**7 (13%)**
Fetal maternal hemorrhage	4 (3%)	1 (1%)	3 (6%)
Abruptio placentae	3 (2%)	0 (0%)	3 (6%)
Complications of multiple gestation	3 (2%)	3 (4%)	0 (0%)
Uteroplacental insufficiency	1 (1%)	0 (0%)	1 (2%)
Maternal or Fetal Hematologic Conditions (INCODE Category 3)	**1 (1%)**	**0 (0%)**	**1 (2%)**
Transient abnormal myelopoiesis	1 (1%)	0 (0%)	1 (2%)
Fetal Genetic, Structural, and Karyotypic Abnormalities (INCODE Category 4)	**13 (11%)**	**9 (13%)**	**4 (7%)**
Aneuploidy	5 (4%)	5 (7%)	0 (0%)
Cardiac anomaly causing hydrops (structural defects/dysrhythmias)	5 (4%)	3 (4%)	2 (4%)
Fetal tumor causing hydrops	1 (1%)	0 (0%)	1 (2%)
Other chromosomal or structural abnormality	2 (2%)	1 (1%)	1 (2%)
Placental and/or Fetal Infection (Excluding Fetal Membranes) (INCODE Category 5)	**6 (5%)**	**4 (6%)**	**2 (4%)**
Fetal infection	5 (4%)	3 (4%)	2 (4%)
Placental infection	1 (1%)	1 (1%)	0 (0%)
Pathologic Placental Conditions (INCODE Category 6)	**70 (57%)**	**42 (62%)**	**28 (52%)**
Early amnion rupture sequence	3 (2%)	3 (4%)	0 (0%)
Compromised maternal circulation: vascular lesions	17 (14%)	7 (10%)	10 (19%)
Compromised fetal microcirculation with umbilical cord abnormality	43 (35%)	29 (43%)	14 (26%)
Compromised fetal microcirculation without umbilical cord abnormality	6 (5%)	2 (3%)	4 (7%)
Other placental abnormalities	1 (1%)	1 (1%)	0 (0%)
No probable Cause of Death by INCODE	**19 (15%)**	**9 (13%)**	**10 (19%)**

Conclusions: The most prevalent cause of second and third-trimester pregnancy loss is pathologic placental conditions. This study stresses the importance of placental examination, particularly examination of the umbilical cord and fetal vasculature for establishing the cause of death in both second and third-trimester fetuses.

907 Importance of Histopathological Changes of Recovery in Resected Intestine in Neonates with Surgical Necrotizing Enterocolitis

Charles Middleton[1], Neha Varshney[2], Ashish Kurundkar[3], Jaslyn Paschal[1], Akhil Maheshwari[4], Parvesh Garg[1]

[1]University of Mississippi Medical Center, Jackson, MS, [2]University of Mississippi, Jackson, MS, [3]Medical University of South Carolina, Charleston, SC, [4]Johns Hopkins University School of Medicine, Baltimore, MD

Disclosures: Charles Middleton: None; Neha Varshney: None; Ashish Kurundkar: None; Jaslyn Paschal: None; Parvesh Garg: None

Background: Necrotizing enterocolitis (NEC) is a leading cause of mortality in preterm newborns. It is characterized by loss of intestinal barrier, necrosis of mucosa and bowel wall, and acute and chronic inflammation. In some infants, the resected intestine shows histopathological changes of scarring and recovery from necrosis and inflammation. In this study, we sought to determine the clinical importance of these changes of histopathological recovery in the resected intestine in surgical NEC.

Design: We studied 86 neonates with surgical NEC. 245 H&E-stained sections were examined for necrosis, inflammation, and reparative changes. Reparative changes included presence of fibroblasts/myofibroblasts, neovascularization, and regeneration of the epithelium. We compared the clinical outcomes in neonates with and without healing changes.

Results: Twenty-one of our 86 (24.4%) infants showed focal healing changes in the submucosa with abundant fibroblasts/myofibroblasts and neovascularization. Epithelial regeneration was evident in 1 infant. The two groups were similar in terms of inflammation with macrophage and lymphocyte infiltration [15/21 with reparative changes (71.4%) and 54/65 (83%; p = 0.22) in those with no healing changes]. Infants with healing changes had lower birth weights (868 ± 452 vs.1057 ± 461 grams; p=0.03), shorter lengths of time elapsed between the onset of clinical signs and laparotomy (134 vs. 145 hours; p= 0.04), and had shorter bowel resections (17.6 vs. 34.3 cm; p = 0.003). The two groups did not differ in gestational age, age of NEC onset, parenteral nutrition days, length of hospital stay or mortality.

Figure 1 - 907

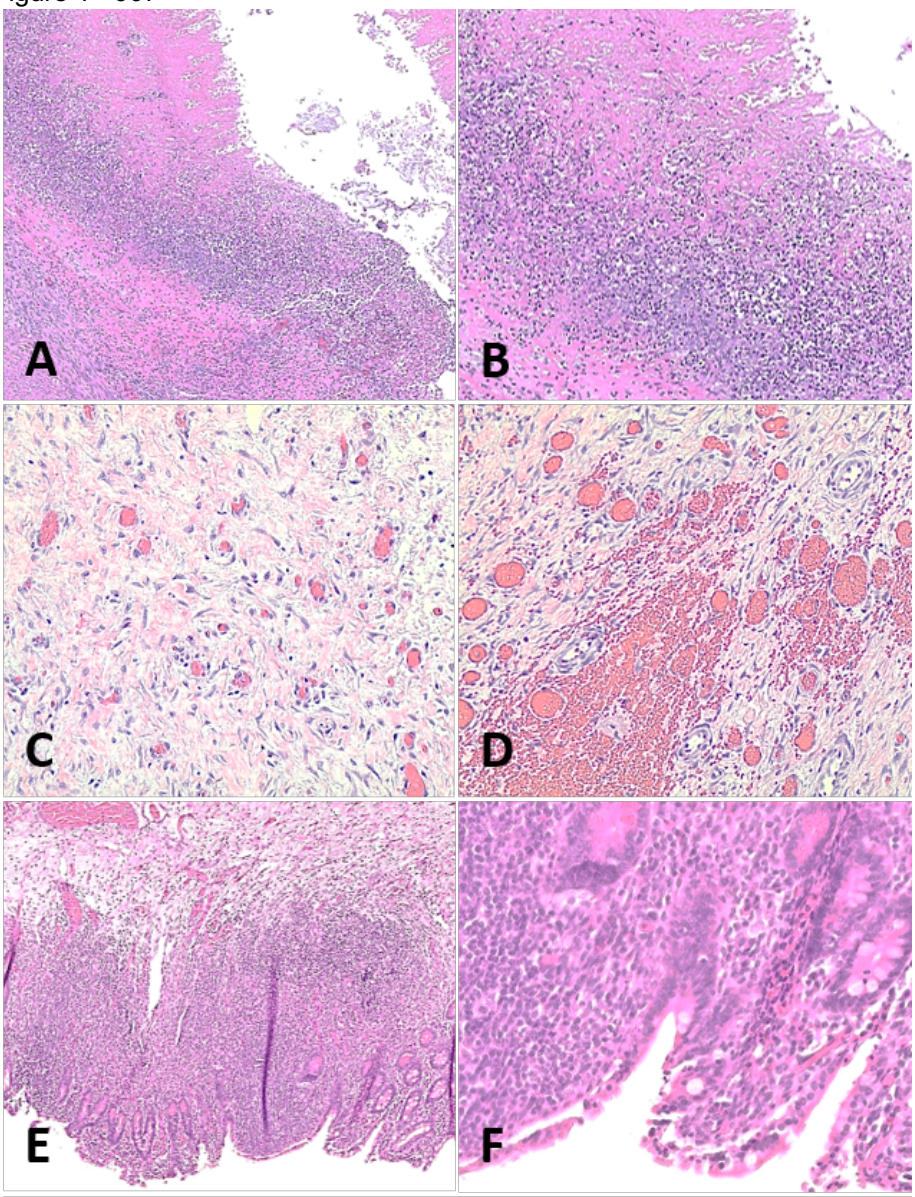


Figure 1: Histologic changes seen in necrotizing enterocolitis. (A) Necrosis and inflammation with no evidence of reparative change (5x). (B) Necrosis and inflammation with no evidence of reparative change (10x). (C) Moderate degree of increased fibroblasts and neovascularization within the submucosa (10x). (D) High degree of increased fibroblasts and neovascularization within the submucosa (10x). (E) Epithelial regeneration of the mucosal surface (5x). (F) Epithelial regeneration of the mucosal surface (20x).

Conclusions: Changes of intramural healing and scarring were seen in the submucosa in about a fourth of all infants with surgical NEC. These patches of reparative changes showed abundant fibroblasts/myofibroblasts and neovascularization. We had anticipated these changes of recovery to be seen in more mature infants, but these findings were surprisingly more prominent in infants who had lower birth weights and underwent laparotomy at an earlier chronological age.

908 Clinical, Histopathologic, and Molecular Findings in Pediatric Non-Tuberculous Mycobacterial Lymphadenitis

Jennifer Mingrino[1], Katelyn Dannheim[2], Sonja Chen[3]
[1]Brown University, Rhode Island Hospital, Providence, RI, [2]Rhode Island and Hasbro Children's Hospitals, Providence, RI, [3]Rhode Island Hospital, Providence, RI

Disclosures: Jennifer Mingrino: None; Katelyn Dannheim: *Consultant*, K.C.D. has received consulting fees from PathAI for work unrelated to this manuscript; Sonja Chen: None

Background: Pediatric nontuberculous mycobacterial lymphadenitis (NTM) is a common phenomenon in healthy children less than 5 years of age, presenting with antibiotic-refractory chronic, unilateral, non-tender, hypoechogenic cervical lymphadenopathy by imaging. We sought to describe the clinicopathologic characteristics of this disease.

Design: Between 2010-2020, we identified archival pediatric cases with NTM and reviewed their available pathology material including special stains, immunohistochemistry, molecular microbiology; and imaging and follow-up data.

Results: We identified fourteen patients (age range: 0.4-4y; 4M, 10F) with clinical lymphadenitis involving the head and neck (11) and groin (3), of which 86% (12/14) were unilateral at presentation and 79% (11/14) were given empiric antibiotics, with mean symptom duration of 2.2 ± 1.5 months (range: 0.4-5m). 57% (8/14) had a positive tuberculin skin test. 71% (10/14) had hypoechoic lymphadenopathy by imaging. 57% had excisional biopsies, whereas 58% had dissection ± parotid resection. The average number of submitted and affected lymph nodes was 11 (range: 1-71) and 6 (range: 1-50) respectively. 93% (13/14) had necrotizing granulomas in at least one lymph node at excision. Granulomas were ill-defined in 79% (11/14), had Langhans giant cells in 71% (10/14) and neutrophil-rich necrosis in 50% (7/14). In 57% (8/14) acid fast bacilli were identified by Kinyoun stain (AFB-K), with 62.5% (5/8) having neutrophil-rich necrosis. 79% (11/14) were cultured with 55% (6/11) having a positive culture and of these 33% (2/6) had positive molecular findings. 7% (1/14) had negative AFB-K with positive culture. All cultured specimens grew mycobacterium avium complex. One patient with neutrophil-rich necrosis was diagnosed with chronic granulomatous disease by dihydrorhodamine testing and had negative tuberculin skin test, stains and culture. 50% (7/14) received anti-mycobacterial medications with symptomatic improvement in 80% (4/5). 50% (7/14) received no further therapy. 71% had improved symptoms with an average follow up time of 42 ± 39 days. Three patients were lost to follow-up. See Table 1 and Figure 1.

Table 1. Pediatric Atypical Non-tuberculous Mycobacteria Lymphadenitis: Epidemiology, Clinical Data, & Histopathology by Case

ID	Age (y) /Sex	Lymphadenopathy		Imaging	Prior ABX	TST	Surg	Granuloma	Microbiology			NTM therapy	Outcome
		Laterality & Location	Time* (mo)	(US/MRI)				(NG/NNG)	AFB Stain	AFB Cx	Mol		
NTM 1	1/F	R submandibular	0.5	Hypoechoic LAD	Y	+	Ex	NG/NNG	+	MAC	MAC	Y	Improved
NTM 2	2/F	R inguinal	1	Hypoechoic LAD	Y	NP	Ex	NG only	+	NP	NP	NR	Lost to followup
NTM 3	4/F	L inguinal	3	Hypoechoic LAD	NR	NP	Ex	NG/NNG	EQ	–	–	NR	Lost to followup
NTM 4	2/M	R submandibular	3	Hypoechoic LAD	Y	–	Ex	NG/NNG	+	–	NP	NR	Lost to followup
NTM 5	0.9/F	Cervical	3.5	LAD, calcification	Y	+	ND	NG/NNG	–	NP	–	–	Improved
NTM 6	2/M	Cervical	4	LAD, drainage	Y	+	ND	NG/NNG	+	MAC	–	Y	Persistence
NTM 7	1/F	R cervical	5	LAD, calcification	N	–	ND	NG/NNG	+	–	–	Y	Improved
NTM 8	1/M	L cervical	3	Hypoechoic LAD, calcification	Y	+	ND	–	–	MAC	–	Y	Improved
NTM 9	0.4/M	R inguinal	1	Hypoechoic LAD	Y	–	Ex	NG only	–	–	NP	N	Improved
NTM 10	2.5/F	R preauricular	0.7	Hypoechoic LAD	Y	+	Ex	NG only	–	–	NP	–	Improved
NTM 11	1/F	L cervical	0.4	Hypoechoic LAD	Y	+	ND	NG/NNG	+	MAC	–	–	Improved

NTM 12	1/F	R cervical	1	Matted LAD with necrosis	Y	+	ND	NG/NNG	+	MAC	MAC	Y	Improved
NTM 13	2/F	R submandibular	3	Hypoechoic LAD	Y	+	Ex	NG/NNG	+	MAC	–	Y	Improved
NTM 14	2/F	L parotid	1.8	Hypoechoic LAD	N	np	Ex	NG/NNG	–	–	NP	Y	Improved

*Time: Duration in months from symptom to definitive therapy

y, years; mo, months; US: Ultrasound; MRI: Magnetic resonance imaging; ABX: Antibiotics; Tuberculin skin test (TST); Surg: surgical procedure; Ex, excision; ND: neck dissection; NG, necrotizing granuloma; NNG, non-necrotizing granuloma; AFB, acid fast bacilli; Cx: culture; Mol, Molecular microbiology; NTM- Non-tuberculous Mycobacteria; LAD – lymphadenopathy/lymphadenitis; NP, not performed, NR, not reported; EQ, equivocal; MAC, mycobacterium avium complex.

Figure 1 - 908

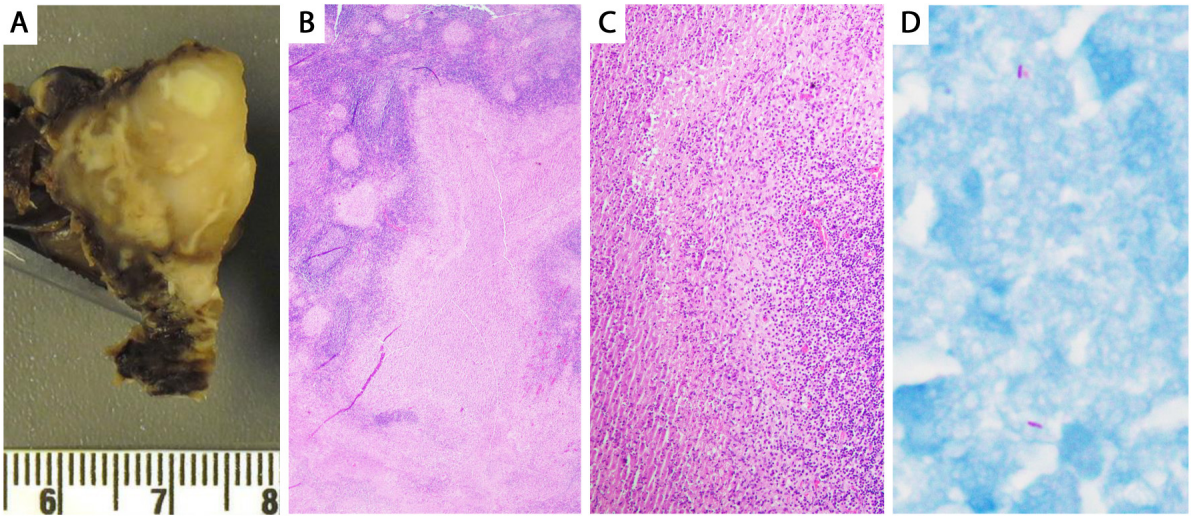


Figure 1. (A) Gross specimen, lymph node with foci of necrosis, (B) Hematoxylin & Eosin, ill-defined necrotizing granulomas (2x), (C) Hematoxylin & Eosin, neutrophil-rich necrotizing granuloma (10x), (D) Kinyoun Stain, acid fast bacilli (100x)

Conclusions: We show that chronic granulomatous disease is a clinical mimicker and while not distinctive histologically, NTM lymphadenitis should be sampled thoroughly and may demonstrate neutrophil-rich necrosis. One case had false negative histology and positive culture, suggesting that multiple modes of identification should always be employed to improve disease identification.

909 Clinicopathologic and Genetic Findings of Infantile Nodular Fasciitis

Yan Qiu[1], Xue Hu[1], Wenjing Zeng[1], Min Chen[1], Huijiao Chen[1], Zhang Zhang[1], Xin He[1], Hongying Zhang[2]
[1]West China Hospital, Sichuan University, Chengdu, China, [2]West China Hospital, Sichuan University

Disclosures: Yan Qiu: None; Xue Hu: None; Wenjing Zeng: None; Min Chen: None; Huijiao Chen: None; Zhang Zhang: None; Xin He: None; Hongying Zhang: None

Background: NFs is a clonally proliferative lesion that usually arises in the adulthood during the second to fourth decades, but not frequently occurs in the childhood, especially in infantile groups. It is prone to be mixed up with soft tissue sarcomas in diagnosis-making process due to the presence of rapid growth rate, high cellularity and brisk mitosis. As far as we know, the clinical, pathological, and genetic features have not been reported in series cases of infantile groups.

Design: Cases with infantile nodular fasciitis (younger than 24 months old) were extracted by a SNOMED search of the hospital surgical pathology files from January 2008 to July 2020. The final diagnoses were reviewed by

senior tissue pathologists. Fluorescence in situ hybridization (FISH) was performed to detect the rearrangement of *USP6*, and reverse transcription polymerase chain reaction assay (RT-PCR) was applied to identify the common type of *MHY9-USP6* fusion in the infantile group.

Results: Ten infants with pathologically confirmed nodular fasciitis were identified among 603 patients (1.7%), including 6 boys and 4 girls. Patients generally presented with firm, enlarged and round soft-tissue masses. The most common disease locations were the head and neck (50%), followed by trunk (40%) and extremities (10%). The median age of patients at diagnosis was 11 months (4 to 23 months). All patients underwent surgical excision. The resected tumors were in largest dimension from 1.4 to 4cm (median 1.9 cm). The duration periods were from 0.5 to 12 months (median 3 month). Histologically, lesions of most patients (6/10) presented as hypercellularity and brisk mitosis. Genetically, FISH revealed that 8 of 10 (80%) cases were positive for *USP6* rearrangement. Tissues of 7 cases with *USP6* rearrangement were retrievable, among which *MHY9-USP6* fusion was observed within only 1(14.2%) case via RT-PCR. Follow-up data was available in 8 patients and the mean (range) follow-up was 53.3 (8-121) months, and one patient suffered from recurrence at 5 months after surgery.

Conclusions: Infantile nodular fasciitis might demonstrate some special characteristics. Clinically, its primarily involved location was head and neck instead of upper extremities. Histologically, lesions of most patients showed higher cellularity. Genetically, lower rate of *MHY9-USP6* fusion pattern was observed. Complete surgical excision is the optimal treatment. The findings of our study indicated that this cohort may presents with some special features, but larger cohort and further investigation are required.