PEDIATRIC ALLERGY, IMMUNOLOGY, AND PULMONOLOGY
Volume 35, Number 1, 2022
© Mary Ann Liebert, Inc.
DOI: 10.1089/ped.2021.0070

CASE REPORT

Open camera or QR reader and scan code to access this article and other resources online.

Specific Granule Deficiency Due To Novel Homozygote *SMARCD2* Variant

Zeynep Kihtir, MD,[1] Kıymet Çelik, MD,[1] Funda Tayfun Küpesiz, MD,[2] Osman Alphan Küpesiz, MD,[2] Dilara Fatma Kocacik Uygun, MD,[3] Sema Arayici, MD,[1] Hakan Ongun, MD,[1] İpek Acarbulut, MD,[1] Celal Sağlam, MD,[1] Gülay Ceylaner, MD,[4] and Ayşen Bingöl, MD[3]

Downloaded by REPRINTS DESK INC from www.liebertpub.com at 03/28/22. For personal use only.

Background: Specific granule deficiency (SGD) is a rare immunodeficiency associated with *CCAT/enhancer-binding protein epsilon (CEBPE)* gene variants. It can cause severe recurrent infections and is lethal without successful stem cell transplantation. Few cases with SGD of both type 1 and type 2 have been described in the literature. In this study, we present the first report of a case with a novel homozygous *c.511 C>T (p.Gln171Ter)* mutation in the *SMARCD2* gene of SGD type 2, which was successfully treated with bone marrow transplantation.
Case: A male infant presented to our neonatal intensive care unit on the second day of life with an icteric appearance and mild hypotonia. He was evaluated for immunodeficiency as the cause of delayed cord separation and refractory neutropenia. At 6 weeks of age, SGD type 2 with a new variant was diagnosed and successfully treated by bone marrow transplantation.
Conclusion: SGD is an immunodeficiency disease that is quite rare. However, we believe that SGD diagnosis and associated new variants can be detected more frequently with the widespread use of all whole-exome sequencing techniques.

Keywords: newborn, genetics, immunologic deficiency syndromes, neutrophils, cytoplasmic granules

Introduction

Specific granule deficiency (SGD) is a rare autosomal disorder defined by the absence of the granule proteins that normally fill the lumen of 2 of the 3 granule populations in neutrophils. SGD is caused by a homozygous mutation in the *CCAAT Enhancer Binding Protein Epsilon* (*CEBPE*) gene (type I) on chromosome 14q11.[1] The *CEBPE* gene encodes a transcription factor that is expressed primarily during granulocytic differentiation. Targeted inactivation of the *CEBPE* gene in mice results in macrophage dysfunction, impaired tumor killing, and lymphoproliferative disorders.[2] Yamanaka et al.[3] reported that mice with a ''knockout'' of the *CEBPE* gene developed normally and were fertile, but failed to produce functional neutrophils and eosinophils. Thus, *CEBPE* is essential for terminal differentiation and functional maturation of committed granulocyte progenitor cells.

Witzel et al.[4] reported that the *SWItch/Sucrose Non-Fermentable (*SWI/SNF*)-related, matrix-associated, actin-dependent regulator of chromatin, subfamily d, member 2 (SMARCD2)* mutation is associated with the SGD (type II) fenotype. *SMARCD2* is also reported to interact with the transcription factor *CEBPE* and control the expression of neutrophil proteins stored in the specific granules. Neutrophils of individuals with SGD have atypical bilobed nuclei, lack expression of all secondary and tertiary granule proteins, and have defects in chemotaxis, disaggregation, receptor upregulation, and bactericidal activity, leading to frequent and severe bacterial infections.[5] Frequent and severe bacterial infections are the main findings in SGD patients.[6]

In this study, we report a newborn who had a novel homozygous *SMARCD2* variant leading to SGD and was successfully treated with bone marrow transplantation.

[1]Department of Neonatology, Akdeniz University Medical Faculty, Antalya, Turkey.
[2]Department of Pediatric Hematology and Oncology, Akdeniz University Medical Faculty, Antalya, Turkey.
[3]Department of Pediatric Allergy and Immunology, Akdeniz University Medical Faculty, Antalya, Turkey.
[4]Intergen Genetic and Rare Diseases Diagnosis and Research Center, Ankara, Turkey.

Downloaded by REPRINTS DESK INC from www.liebertpub.com at 03/28/22. For personal use only.

Case Presentation

A male infant with a gestational age of 38 weeks and a birth weight of 3,080 g was born by cesarean delivery to a healthy 31-year-old gravida 2 para 2 mother and a healthy 40-year-old father. There was a consanguineous marriage between parents (cousin marriage). His older sister died of sepsis on the 40th day of life, before the diagnosis was made on investigation of immunodefficiency. He was admitted to our neonatal intensive care unit on the second day of life for direct hyperbilirubinemia. Physical examination revealed an icteric appearance and mild hypotonia. Other physical examination findings and vital signs were normal with no dysmorphic signs.

Laboratory tests revealed total bilirubin of 16.6 mg/dL, direct bilirubin of 9 mg/dL, aspartate aminotransferase of 316 U/L, alanine aminotransferase of 248 U/L, and other biochemical tests results were within the normal range. Total leukocyte count was 5,700/mm^3 and absolute neutrophil, lymphocyte, monocyte, basophil, and eosinophil counts were 1,300/mm^3, 2,370/mm^3, 1,970/mm^3 50/mm^3, and 10/mm,^{3} respectively. Vitamin B12 was measured to be 482 pg/mL. Bone marrow smear showed bilobed abnormal granulocytes without granules (Fig. 1). Dihydrorhodamine test was positive. Blood, urine, and cerebrospinal fluid cultures collected at admission were found to be sterile. Cranial ultrasound and echocardiogram were normal. Ophthalmologic examination showed that the fundus was normal.

Tests were done for etiology of cholestasis, which were toxoplasmosis, rubella, cytomegalovirus and herpes simplex scanning, hepatitis A, B, C for viral hepatitis, brucellosis, and syphilis. The results were all negative. Alpha-1 antitrypsin with a value of 2 g/L and metabolic tests were normal (amino acids in blood and urine, ammonia, reducing substances in urine, galactose-1-phosphate uridylyltransferase), bile acids were slightly above normal at 41.8 μmol/L. The liver, bile duct, and gallbladder were visualized by both ultrasound and magnetic resonance cholangiopancreatography and were considered normal. Liver biopsy showed cholestasis and findings of extramedullary hematopoiesis. The patient was neutropenic from birth on follow-up. Immunodeficiency was considered because of the delay of cord separation, neutropenia during hospitalization, high levels of C-reactive protein (CRP) with febrile and subfebrile episodes of fever, although no pathogen was produced in his cultures.

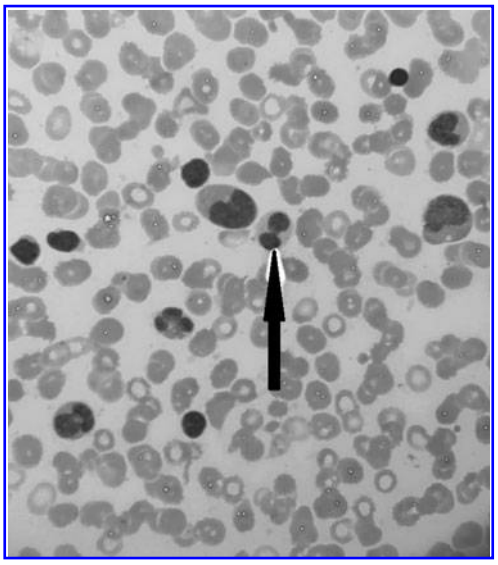


FIG. 1. Neutrophil with a bilobed nucleus in bone marrow smear (*black arrow*).

Immunological tests performed on both peripheral blood and bone marrow showed low CD 19 and CD 16 levels. Other immunological tests such as immunoglobulin levels such as IgG 6.29 g/L, IgA 0.08 g/L, IgM 0.17 g/L, and lymphocyte subgroups were normal. Bone marrow aspiration showed absence of neutrophils and pause in myeloid series with hypocellularity and there were megaloblastic changes in the bone marrow. Trimethoprim-sulfamethoxazole and flucanozole were given prophylactically. The patient with high CRP levels was treated with a broad spectrum antibiotic (vancomycin and meropenem) during his hospitalization.

On the 40th day of life, *Serratia marcences* was isolated from the blood culture specimen and *Aspergillus* antibody positivity was detected, both of which were successfully treated with meropenem, amicasin, and liposomal amphotericin b. He also received a filgastrim infusion due to neutropenia. Separation of the umbilical cord was on the sixth week of his life. The cholestasis regressed at follow-up. Whole-exome sequencing test revealed a homozygous *c.511 C>T (p.Gln171Ter)* mutation in the *SMARCD2* gene causing SGD 2 disease. The heterozygous form of the same mutation was found in the parents.

Hematopoietic stem cell transplantation, which is a curative treatment, was planned for the patient with a fungal infection, bloodstream infection, ulcerated skin lesion, unresponsiveness to filgrastim, and dysplastic changes in bone marrow. The patient, 87 days old, was performed hematopoietic stem cell transplantation from an HLA full-matched mother.

A reduced toxicity conditioning regimen, which is less hepatotoxic because the patient has signs of cholestasis, is treosulfan 36 g/[m^2·day] (−7, −6, and −5 days), fludarabine 30 mg/[m^2·day] (−7, −6, −5, and −4 days) was preferred.[7] Cyclosporine was used for graft-versus-host disease prophylaxis. The defibrotide and ursodeoxycholic acid were administered as prophylactic regimens for veno-occlusive disease (VOD). In the pre- and posttransplant period, antibacterial (ciprofloxacin), antiviral (acyclovir), antifungal (amphotesine B), and *Pneumocystis jirovecii* (thrimetophrim- methoxazole*)* prophylaxis were administered.

There was minor ABO group incompatibility between recipient (A RhD negative) and donor (0 RhD negative). Despite the fact that the mother's Anti-A titer was 1/4096, bone marrow stem cell was preferred instead of peripheral stem cells (PBSC) to decrease risk of GVHD. After erythrocyte and plasma depletion, the Anti-A titer of the product was reduced to 1/2048. The CD34 count at harvest was 5.94×10^6/kg and the total nucleated cell (TNC) count was 4.54×10^8/kg. Neutrophil and thrombocyte engraftment could not be achieved and accepted as graft failure.

Since there was no full-match donor candidate other than her mother, a second transplant was planned from the mother.

In the second transplant, myeloablative conditioning regimen (Busulfan 5.1 mg/[kg·day] (−5, −4, −3, and −2 days), fludarabine 40 mg/[m^2·day] (−5, −4, −3, and −2 days), and anti-human globulin 10 mg/[kg·day] (−5, −4, −3, and −2 days) was administered. The anti-A titer of the PBSC harvest was reduced from 1/2048 to 1/512 (CD34 count: 8.38×

10^6/kg, TNC count:10.4×10^8/kg) by apheresis collection. Mesenchymal stem cell was applied (8×10^6) −1 day to reduce engraftment failure. As a GVHD prophylaxis, cyclosporin A and short methotrexate regimen (1, 3, and 6 days) were given. The prophylactic theraphy was administered for VOD.

The second transplant was performed 57th day after the first transplant when the patient was 144 days old. Neutrophil engraftment occurred on day 11 and platelet engraftment on day 12. Stage I acute skin GVHD developed post-27 HSCT and resolved with low-dose metilprednisolone treatment in 2 weeks. The patient followed by full donor chimerism without any complications.

He is still on outpatient follow-up with asyclovir, ciprofloxacin, trimethoprim-sulfamethoxazole prophylaxis, and cyclosporine and methyl prednisolone. The patient has not received any live vaccines. We also know that there is a preposition for malignancy with these patients, but our patient does not have such a problem for now on his follow-up.

Discussion

We present a case with a novel variant related to SGD type 2 that has not yet been identified.

SGD is an immunodeficiency disease that is relatively rare in both type 1 and type 2.[8] The discovered variant is novel. Adenosine triphosphate-dependent chromatin remodeling complexes are molecules found in eukaryotic cells that provide easy access to chromatin structure.[9]

The *SMARCD-2* gene is located at chromosomal position 17q23.3 and encodes a protein from the ATP-dependent chromatin remodeling complex family.[10] *SMARCD2* has been shown to affect the expression of proteins stored in neutrophil granules by interacting with transcription factors and is also effective in the early differentiation of myeloid progenitor cells.[4] *SMARCD2* interacts with the transcription factor *CEBPE*, which plays a role on SGD type 1, and controls the expression of neutrophil proteins stored in specific granules. To date, only 3 variants in the *SMARCD2* gene have been shown to cause SGD2 (*c.1181+1G> A, c.414_438dup, c.401+2T>C*).[4]

The variant determined in our patient *c.511C>T (p.Gln171Ter)* is a novel variant related to SGD type 2 that has not been identified before. Whole-exome sequencing techniques are increasingly being used in the diagnosis of rare diseases in newborns.[11] We could not reach the diagnosis with the laboratory tests performed with respect to common etiologies in our patient and the clinical findings were accompanied by signs of immunodeficiency, so we performed whole-exome sequencing for the diagnoses.

To the best of our knowledge, there are few cases in the literature with SGD both type 1 and type 2. Delayed separation of the umbilical cord has been reported as a common finding in these cases as in our case, and recurrent infections ranging from recurrent skin abscesses to severe invasive bacterial infections or sinopulmonary bacterial infections or even brain abscesses have been noted in almost all of them in the first year of life.[5]

Treatment of neutrophil disorders includes antimicrobial therapy, granulocyte-colony stimulating factor, and allogeneic hematopoietic stem cell transplantation.[7,12] Bakhtiar et al.[13] in their review on syndromic congenital neutropenia, tried to answer important questions such as the clinical course of diseases, susceptibility to myeloid malignancies, indication and time of HSCT, and make recommendations by compiling rare data in the literature. In this article, it was stated that due to the low number of cases for *SMARCD2*, there is not enough data for HCST.

Our case is the sixth case reported and the second case who underwent transplantation in the earliest period (infancy) among the cases reported in the literature. Two of the 5 previously presented cases had died before transplantation could be performed, and the other 3 cases are still alive after HSCT.[14]

In the literature, Witzel et al.[4] described 4 patients with symptoms in the neonatal period in 2017. Three patients were from Pakistan and 2 of them were siblings. Last one was a Lebanese patient. The Pakistani patient who underwent HSCT at the age of 1month lived for 1 year and 1 month, and the other sibling underwent HSCT at the age of 2 and was still being followed. The Lebanese patient is 5 years and 4 months old, and the third Pakistani patient died when he was 1.5 months old before HSCT could be performed. In 2021, Yucel et al.[14] presented the 5th patient who underwent HSCT when she was 10 years old in Turkey.

Patients also have extrahematopoietic findings such as mild to moderate developmental delay and dysmorphic features such as low set ears, posteriorly rotated, with prominent concha, hypoplastic mandible, saddle nose, midface hypoplasia, synophris and asymmetrical face (ear to ear) misaligned, dysplastic teeth, and incomplete amelogenesis imperfecta, brachytelephalangia and longitudinal ridges on the fingernails, sandal gap/increased interdigital space, brachymetatarsia and brittle nails, and severe osteopenia with relative constriction of the diaphysis and flaring of the metaphysis (Erlenmeyer deformity).[4] Our patients dysmorphic findings such as hypotonia, developmental delay, hypoplastic mandible, saddle nose and midface hypoplasia, and sandal gap/increased interdigital space became evident with the growth of our patient.

In this case, our most important limitation is that we could not study Sanger sequencing since the result of whole exome sequencing should be confirmed by a Sanger.

Conclusion

SGD is an extremely rare form of immunodeficiency that affects neutrophil function. The most common finding in this disorder is recurrent pyogenic infections, with symptoms beginning in infancy. SGD should also be kept in mind in the differential diagnosis of immunodeficiency diseases when severe pyogenic infections or cholestasis triggered by systemic infections accompanied by persistent neutrophenia are detected in the neonatal period. We believe that SGD diagnosis and associated new variants can be detected more frequently with the widespread use of all whole-exome sequencing techniques.

Informed Consent

Informed consent was obtained from the patient's parents.

Author Disclosure Statement

No competing financial interests exist.

Funding Information

No funding was received for this article.

Downloaded by REPRINTS DESK INC from www.liebertpub.com at 03/28/22. For personal use only.

References

1. Lang F, ed. Encyclopedia of molecular mechanisms of disease. Springer Berlin Heidelberg, 2009.
2. Tanaka T, Akira S, Yoshida K, et al. Targeted disruption of the NF-IL6 gene discloses its essential role in bacteria killing and tumor cytotoxicity by macrophages. Cell 1995; 80:353–361.
3. Yamanaka R, Barlow C, Lekstrom-Himes J, et al. Impaired granulopoiesis, myelodysplasia, and early lethality in CCAAT/enhancer binding protein -deficient mice. Proc Natl Acad Sci 1997; 94:13187–13192.
4. Witzel M, Petersheim D, Fan Y, et al. Chromatin-remodeling factor SMARCD2 regulates transcriptional networks controlling differentiation of neutrophil granulocytes. Nat Genet 2017; 49:742–752.
5. Leszcynska M, Patel B, Morrow M, et al. Brain abscess as severe presentation of specific granule deficiency. Front Pediatr 2020; 8:117.
6. Gombart AF, Koeffler HP. Neutrophil specific granule deficiency and mutations in the gene encoding transcription factor C/EBP&epsiv. Curr Opin Hematol 2002; 9:36–42.
7. Party EEW. EBMT/ESID Guidelines for haematopoietic stem cell transplantation for primary immunodeficiencies 2017. https://esid.org/content/download/15402/422689/file/ESID%20EBMT%20HSCT%20Guidelines%202017.pdf (accessed March 15, 2022).
8. Dinauer MC. Inherited neutrophil disorders: molecular basis and new therapies. Hematology 2000; 2000:303–318.
9. Clapier CR, Iwasa J, Cairns BR, et al. Mechanisms of action and regulation of ATP-dependent chromatin-remodelling complexes. Nat Rev Mol Cell Biol 2017; 18: 407–422.
10. Ring HZ, Vameghi-Meyers V, Wang W, et al. Five SWI/SNF-related, matrix-associated, actin-dependent regulator of chromatin (SMARC) genes are dispersed in the human genome. Genomics 1998; 51:140–143.
11. Smith LD, Willig LK, Kingsmore SF. Whole-exome sequencing and whole-genome sequencing in critically ill neonates suspected to have single-gene disorders. Cold Spring Harb Perspect Med 2016; 6:a023168.
12. Lankester AC, Albert MH, Booth C, et al. EBMT/ESID inborn errors working party guidelines for hematopoietic stem cell transplantation for inborn errors of immunity. Bone Marrow Transplant 2021; 56:2052–2062.
13. Bakhtiar S, Shadur B, Stepensky P. The evidence for allogeneic hematopoietic stem cell transplantation for congenital neutrophil disorders: A comprehensive review by the inborn Errors Working Party Group of the EBMT. Front Pediatr 2019; 7:436.
14. Yucel E, Karakus IS, Krolo A, et al. Novel frameshift autosomal recessive loss-of-function mutation in SMARCD2 encoding a chromatin remodeling factor mediates granulopoiesis. J Clin Immunol 2021; 41:59–65.

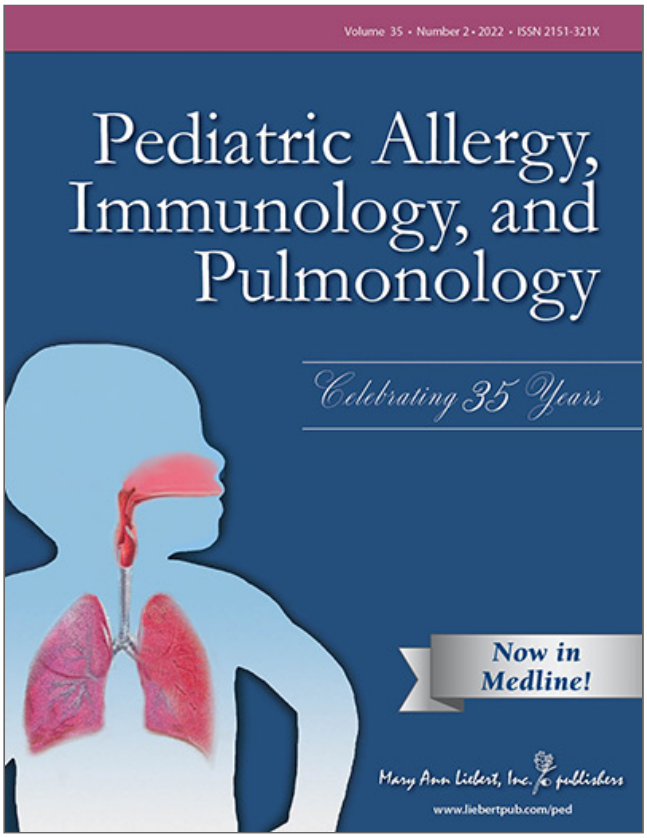
Address correspondence to:
Zeynep Kihtir, MD
Department of Neonatology
Akdeniz University Medical Faculty
Pınarbaşı District Dumlupınar Street
Konyaaltı
Antalya 07070
Turkey

E-mail: zeynepkihtir@gmail.com

Received for publication April 24, 2021; accepted after revision December 9, 2021.

Downloaded by REPRINTS DESK INC from www.liebertpub.com at 03/28/22. For personal use only.

ABOUT THIS JOURNAL >



Pediatric Allergy, Immunology, and Pulmonology

Editor-in-Chief: Mary Cataletto, MD, MMM

ISSN: 2151-321X | Online ISSN: 2151-3228 | Published Quarterly | Current Volume: 35

Impact Factor: 0.885* ⓘ
*2021 Journal Citation Reports™ (Clarivate, 2022)

CiteScore™: 1.8 ⓘ

We use cookies to give you a better experience on liebertpub.com. By continuing to use our site, you are agreeing to the use of cookies as set in our Cookie Policy.

OK

Editorial Board

Editor-in-Chief



Mary Cataletto MD, FAAP, FCCP
Clinical Professor of Pediatrics
NYU Long Island School of Medicine
Mineola, NY, USA
paip@liebertpub.com

Senior Editors

Harold J. Farber, MD, MSPH
Baylor College of Medicine

We use cookies to give you a better experience on liebertpub.com. By continuing to use our site, you are agreeing to the use of cookies as set in our Cookie Policy.

OK

Winthrop University Hospital
Mineola, NY
mdavislorton@winthrop.org

Pathology & Laboratory Medicine
Megan Dishop, MD
Children's Hospital Colorado
Aurora, CO
Megan.Dishop@childrenscolorado.org

Pediatric Radiology
Edward Y. Lee, MD, MPH
Boston Children's Hospital & Harvard Medical School
Boston, MA
Edward.Lee@childrens.harvard.edu

My Corner
Nemr S. Eid, MD
University of Louisville School of Medicine
Louisville, KY
nseid@louisville.edu

International Editors

Myrna B. Dolovich, PEng
McMaster University
Hamilton, Ontario, Canada

Harb A. Harfi, MD, ABD, ABAI
National Center of Allergy, Asthma & Immunology (NCAAI)
Riyadh, Saudi Arabia

Giovanni B. Pajno, MD, FAAAAI
University of Messina-Italy
Messina, Italy

Associate Editors

Joseph A. Bellanti, MD

We use cookies to give you a better experience on liebertpub.com. By continuing to use our site, you are agreeing to the use of cookies as set in our Cookie Policy.

OK

Kathryn Blake, PharmD
Nemours Children's Clinic
Jacksonville, FL

Hengameh Raissy, PharmD
University of New Mexico School of Medicine
Albuquerque, NM

Editorial Board

Joann Blessing-Moore, MD
Stanford University Hospital
Palo Alto, CA

Jean-Marie Bruzzese, PhD
NYU Child Study Center
New York, NY

Jose A. Castro-Rodriguez, MD, PhD
Pontificia Universidad Catolica de Chile
Santiago, Chile

Timothy Craig, DO
Penn State University
Hershey, PA

Jane Davies, MD
Imperial College
London, United Kingdom

Marianne Frieri, MD, PhD
Nassau University Medical Center
East Meadow, NY

LeRoy M. Graham, MD
Morehouse School of Medicine
Atlanta, GA

We use cookies to give you a better experience on liebertpub.com. By continuing to use our site, you are agreeing to the use of cookies as set in our Cookie Policy.

OK

University of Nebraska Medical Center
Omaha, NE

Catherine Kier, MD
State University of New York at Stony Brook
Stony Brook, NY

Alan P. Knutsen, MD
Cardinal Glennon Children's Hospital-St. Louis University
St. Louis, MO

Jason E. Lang, MD
Nemours Children's Hospital
Orlando, FL

George B. Mallory, MD
Baylor College of Medicine, Texas Children's Hospital
Houston, TX

Stephen J. McGeady, MD
Thomas Jefferson University
Philadelphia, PA

Henry Milgrom, MD
National Jewish Medical and Research Center
Denver, CO

Susan Millard, MD
DeVos Children's Hospital
Grand Rapids, MI

Ronald Morton, MD
University of Louisville School of Medicine
Louisville, KY

Eliezer Nussbaum, MD
Miller Children's and Women's Hospital of Long Beach
Long Beach, CA

Nicholas Osborne, PhD

We use cookies to give you a better experience on liebertpub.com. By continuing to use our site, you are agreeing to the use of cookies as set in our Cookie Policy.

OK

The University of Sydney
Westmead, Australia

Kunling Shen, MD
Capital Medical University
Beijing, China

Narong Simakajornboon, MD
Cincinnati Children's Hospital Medical Center
Cincinnati, OH

David Skoner, MD
Allegheny General Hospital
Pittsburgh, PA

Brian A. Smart, MD
Asthma & Allergy Center of DuPage Medical Group
Glen Ellyn, IL

Marianna Martin Sockrider, MD, DrPH, AE-C
Baylor College of Medicine
Houston, TX

Russell Steele, MD
Oschner Children's Health Center
New Orleans, LA

Lynn M. Taussig, MD
University of Denver
Denver, CO

Michael J. Welch, MD
University of California
San Diego, CA

Maryam Zarei, MD
Family Allergy, Asthma, Immunology & Sinus Center
Poway, CA

Social Media Editor

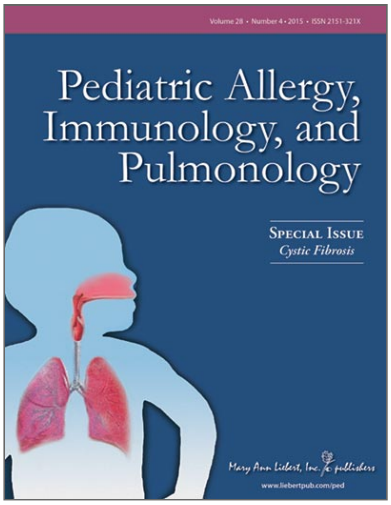
We use cookies to give you a better experience on liebertpub.com. By continuing to use our site, you are agreeing to the use of cookies as set in our Cookie Policy.

OK

Managing Editor

Linda Vranic
lvranic@liebertpub.com

The views, opinions, findings, conclusions and recommendations set forth in any Journal article are solely those of the authors of those articles and do not necessarily reflect the views, policy or position of the Journal, its Publisher, its editorial staff or any affiliated Societies and should not be attributed to any of them.



2015 December

Special Issue:
Cystic Fibrosis

READ MORE

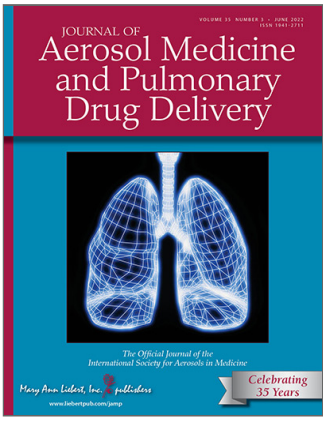
More Special Issues...

We use cookies to give you a better experience on liebertpub.com. By continuing to use our site, you are agreeing to the use of cookies as set in our Cookie Policy.

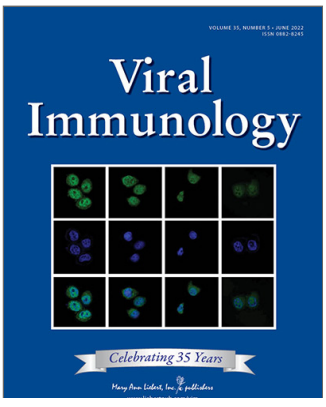
OK

ABOUT THIS JOURNAL >

Recommended Publications



Journal of Aerosol Medicine and Pulmonary Drug Delivery



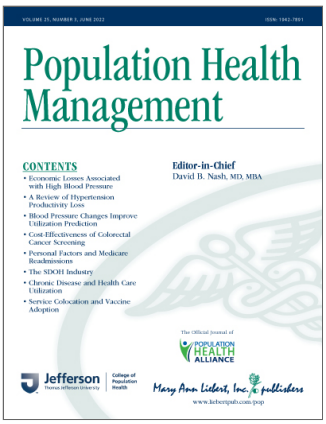
We use cookies to give you a better experience on liebertpub.com. By continuing to use our site, you are agreeing to the use of cookies as set in our Cookie Policy.

OK

ABOUT THIS JOURNAL >



Breastfeeding Medicine



Population Health Management

Call us toll free (800) M-LIEBERT

Publications
For Authors
Librarians
Open Access
Corporate Capabilities
Advertising
Company
Customer Support
Contact Us
Privacy Policy

© 2022 Mary Ann Liebert, Inc., publishers. All rights reserved, USA and worldwide.
Call us toll free at (800) M-LIEBERT (800-654-3237).

We use cookies to give you a better experience on liebertpub.com. By continuing to use our site, you are agreeing to the use of cookies as set in our Cookie Policy.

OK

NEW The power of the Web of Science™ on your mobile device, wherever inspiration strikes. Dismiss Learn More

Already have a manuscript?

Use our Manuscript Matcher to find the best relevant journals!

Find a Match

Refine Your Search Results

PEDIATRIC ALLERGY IMMUNOLOGY AND PULI Search

Sort By: Relevancy

Search Results

Found 532 results (Page 1) Share These Results

Filters Clear All

Web of Science Coverage

Open Access

Category

Country / Region

Language

Frequency

Journal Citation Reports

Exact Match Found

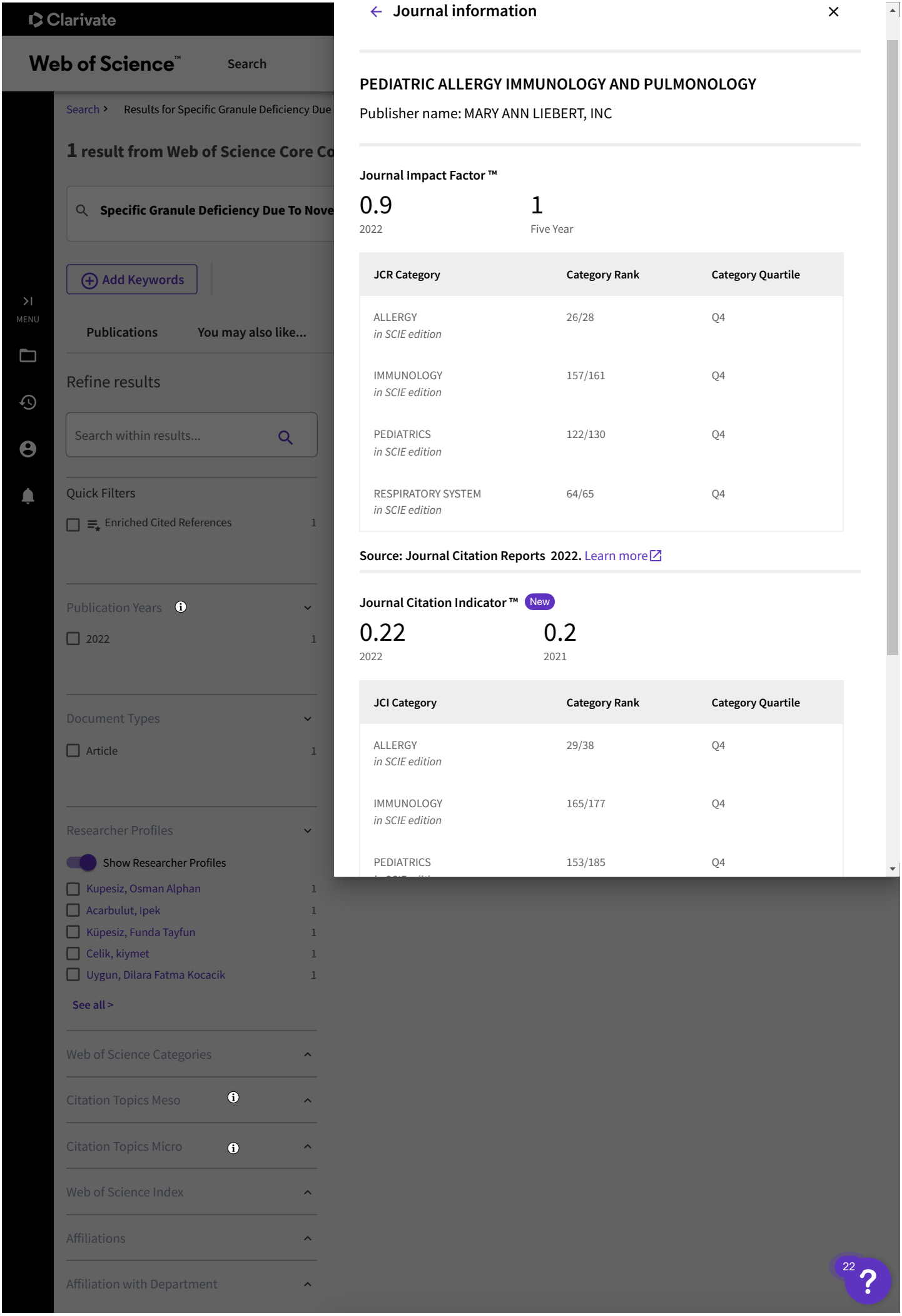
PEDIATRIC ALLERGY IMMUNOLOGY AND PULMONOLOGY

Publisher:	MARY ANN LIEBERT, INC , 140 HUGUENOT STREET, 3RD FL, NEW ROCHELLE, USA, NY, 10801
ISSN / eISSN:	2151-321X / 2151-3228
Web of Science Core Collection:	Science Citation Index Expanded
Additional *Web of Science* Indexes:	Biological Abstracts | BIOSIS Previews | Current Contents Clinical Medicine | Essential Science Indicators

Share This Journal

View profile page

Other Possible Matches


Clarivate
Web of Science
Search
Search > Results for Specific Granule Deficiency Due
1 result from Web of Science Core Co
Specific Granule Deficiency Due To Nove
Add Keywords
MENU
Publications
You may also like...
Refine results
Search within results...
Quick Filters
Enriched Cited References 1
Publication Years
2022 1
Document Types
Article 1
Researcher Profiles
Show Researcher Profiles
Kupesiz, Osman Alphan 1
Acarbulut, Ipek 1
Küpesiz, Funda Tayfun 1
Celik, kiymet 1
Uygun, Dilara Fatma Kocacik 1
See all >
Web of Science Categories
Citation Topics Meso
Citation Topics Micro
Web of Science Index
Affiliations
Affiliation with Department
Journal information
PEDIATRIC ALLERGY IMMUNOLOGY AND PULMONOLOGY
Publisher name: MARY ANN LIEBERT, INC
Journal Impact Factor ™
0.9
2022
1
Five Year
JCR Category | Category Rank | Category Quartile
ALLERGY in SCIE edition | 26/28 | Q4
IMMUNOLOGY in SCIE edition | 157/161 | Q4
PEDIATRICS in SCIE edition | 122/130 | Q4
RESPIRATORY SYSTEM in SCIE edition | 64/65 | Q4
Source: Journal Citation Reports 2022. Learn more
Journal Citation Indicator ™ New
0.22
2022
0.2
2021
JCI Category | Category Rank | Category Quartile
ALLERGY in SCIE edition | 29/38 | Q4
IMMUNOLOGY in SCIE edition | 165/177 | Q4
PEDIATRICS | 153/185 | Q4
22