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OF THE ARKANSAS MEDICAL SOCIETY

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by MAURINE E. LESTER, CDT; CHRISTOPHER SCHACH, MD; ERIC STEWART, MD

A 61-year-old male veterinarian presents with a 2-3-month history of an enlarging well-demarcated plaque with scale, crusting, and oozing over his left second metacarpophalangeal joint. The lesion developed after an abrasion. The patient is currently taking oral trimethoprim-sulfamethoxazole and applying topical mupirocin with minimal response. The plaque has continued to increase in size and tenderness. The patient denies fever, cough, and chest pain.

Provided the clinical image and patient history, what is the most appropriate next step in the diagnosis and treatment of this patient?

A.) Immediate biopsy to confirm the diagnosis of squamous cell carcinoma. Pending pathology results, the lesion should be excised utilizing a standard excision or Mohs surgery. Electrodessication and curettage is not recommended for this location.

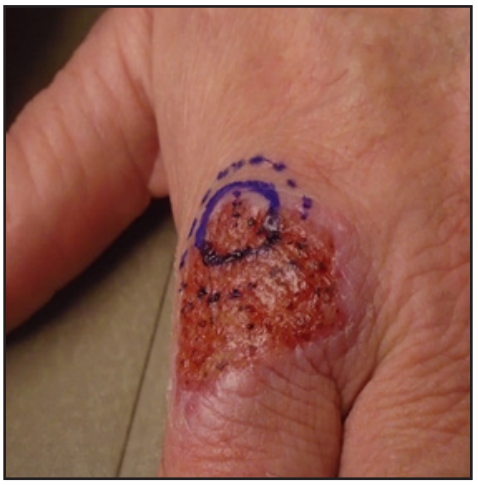
B.) The lesion is consistent with pyoderma gangrenosum. A skin prick test causing a papule, pustule or ulcer is helpful in diagnosis (pathergy phenomenon) in addition to the clinical appearance and pain associated with the lesion. Necrotic tissue should be gently debrided and a Class I topical corticosteroid should be prescribed. Resolution may take several months to a year.

C.) A potassium hydroxide mount (KOH) is needed to assess for the presence of broad-based budding spores which suggests the patient has an infection with Blastomycosis. Skin biopsy for fresh tissue culture and special stains is necessary to confirm the KOH findings. If patient has any respiratory symptoms, a chest x-ray should be obtained to ensure there is not active pulmonary infection.

D.) This eruption represents dermatomyositis. Subsequent laboratory evaluation should include muscle enzymes as well as appropriate autoantibodies. A skin biopsy of the lesion is also necessary and will show a mild interface dermatitis with increased mucin. Prescribe an oral corticosteroid, such as prednisone, to slow down the progression of the disease. This disease state is photosensitive so warn the patient to avoid direct sunlight. Inform the patient that this disease is not curable and will most likely necessitate chronic management.

Answer: C

Blastomycosis is a fungal infection caused by Blastomyces dermatitidis. This dimorphic fungus is most commonly found in the soil in North America, most notably the southeastern states, and is usually contracted by inhalation of the microscopic fungal spores from the air. Due to the method of transmission, blastomycosis most often presents as a pulmonary infection but may occasionally manifest cutaneous symptoms. It is rare for blastomycosis to only occur as a primary cutaneous infection but is possible through direct inoculation. From the patient's history, it is known the lesion began as an injury. Given that the patient is a veterinarian, it is plausible that an animal was the vector for infection in this case and, interestingly, dogs are the most common animal to be infected with blastomycosis.



Typically, cutaneous blastomycosis presents as verrucous plaques and/or papulopustules with overlying erosions. Seropurulent scale/crust may occur around the periphery and the lesion(s) may ulcerate centrally. It is commons for skin lesions to occur on extremities, neck, and face and scarring is possible upon resolution. About half of patients with acute pulmonary blastomycosis will have flu-like symptoms such as fatigue, muscle aches, chest pain, cough, and fever while others may be asymptomatic. Infected patients with compromised immune systems can experience more severe symptoms and are more likely to have the infection disseminate to other organs from the lungs. Symptoms of blastomycosis (both cutaneous and pulmonary) most often occur within a few weeks of inhalation or inoculation.

The key to diagnosing blastomycosis is observing broad-based budding spores from a KOH skin prep or biopsy specimen. The scraping and biopsy specimen were both positive for broad-based budding spores. Obtaining a deep tissue culture is ideal for confirming blastomycosis, but this specific fungal infection can be quite difficult to grow in a lab. In this case, it was not possible to do so. However, the clinical diagnosis in combination with the positive scraping and skin biopsy were enough to confirm the diagnosis. In some cases, blood or urine serologies may be used to aid the diagnosis. If blastomycosis manifests as a pulmonary infection, a chest X-ray or CT scan of the thorax can confirm the severity of the infection. In this particular patient's case, a chest radiograph was not considered due to high suspicion of primary cutaneous disease and absence of pulmonary symptoms.

Treatment of blastomycosis is dependent upon extent and severity. In critical situations, Amphotericin B via intravenous administration is necessary. In most cases of cutaneous blastomycosis, orally administered azole antifungals are usually adequate. Oral Itraconazole 200mg twice daily was prescribed for this patient after assessment of hepatic transaminases. The necessary duration of treatment may vary from several months to a year. Itraconazole was discontinued after a total of seven months in this individual progress.

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Crafting a Solution to the Arkansas School Vision Care Follow-Up Gap: Mobile Clinic Funding

Abstract

Each year 21,000 Arkansan children fail their school eye screening and of those two-thirds do not receive follow-up care.[1] There are 529 eye care providers in the state, such that, if each doctor saw 28 of these children, then every child who failed their screening without follow-up would receive care.[2] Yet, the barriers to follow-up researchers have identified (family/social barriers, logistics, timing, and cost) are not readily overcome with present eye care solutions.[3] The purpose of this report is to evaluate, discuss, and propose alternative vision care funding models for a mobile vision clinic to reach underserved Arkansan children.

Problem Statement

1 in 10 children have a vision problem significant enough to impact learning.[4] It is estimated that over 60% of problem learners have undiagnosed vision problems.[5]

Each fall, students receive vision screening from school nurses as required per Arkansas Code §6-18-1501. If a child fails to meet vision standards, they are retested within 30 days and then a note is sent home to their parents requiring that they receive a comprehensive eye exam within 60 days. Unfortunately, 2 out of 3 Arkansas children do not receive a follow-up eye exam after failing multiple vision screenings. This problem is spread across the state with 76% of school districts having a vision follow-up rate of 50% or worse.[6]

The leading cause of pediatric vision impairment – amblyopia – is often fully reversible if detected and treated prior to age 5.[7] However, if this condition is not treated the brain develops without learning how to properly utilize both eyes. After age 7, some amblyogenic vision impairment can be permanent.[8] Time is vision for children with untreated vision deficits. Could a mobile vision clinic initiative provide a sustainable solution of Arkansan children?

Method of Analysis

Evaluation of conflicting evidence on mobile clinic and care outreach sustainability will be the method of analysis. The following viewpoints will be evaluated:

- The entry costs of mobile clinics are more substantial than brick-and-mortar clinics.
- Insurance-based funding models are suitable for ensuring long-term mobile vision outreach fiscal stability.

The entry costs of mobile clinics are more substantial than brick-and-mortar clinics.

Mobile Clinic Cost

Through quotes obtained from Life Line Mobile, the cost of a mobile clinic infrastructure with proper electrical wiring, alarm system, back-up camera system, and durability warranty is $279,793.
Further costs for eye care equipment are $50,699. See Table 1 for a complete cost breakdown.

Brick-and-Mortar Clinic Cost

The cost of a start-up brick-and-mortar eye clinic was evaluated from several independent sources.

Table 1

Line Items	Cost per Unit	Quantity	Cost
Motor Clinic	$ 279,793.00	1	$ 279,793.00
Phoropters	$ 1,800.00	2	$ 3,600.00
Slit Lamp Microscopes	$ 2,050.00	2	$ 4,100.00
Exam Chairs	$ 4,250.00	2	$ 8,500.00
Electronic Eye Chart	$ 1,600.00	2	$ 3,200.00
Laptops	$ 550.00	2	$ 1,100.00
Welch Allyn Spot Screener	$ 6,999.00	1	$ 6,999.00
Keratometers	$ 800.00	2	$ 1,600.00
Counterbalance Arm	$ 300.00	2	$ 600.00
Safety Camera's throughout clinic	$ 2,000.00	1	$ 2,000.00
Handicap Vehicle Modification	$ 19,000.00	1	$ 19,000.00
			$ 330,492.00

"The total startup costs for a new Pearle Vision EyeCare Center range from $399,439 to $603,904".[9]

"The most common estimates for startup costs for ophthalmologists are between $200,000 to $300,000, with a $100,000 line of credit to cover operating expenses after opening, as it typically takes three to six months to break even".[10]

Summary

The start-up cost of a mobile eye clinic is comparable to the average brick-and-mortar clinic, however there is significant variability and discrepancy in the available data ($200,000-$603,904).[10-11]

Insurance-based funding models are suitable for ensuring long-term mobile vision outreach fiscal stability.

Viewpoint 1

Per Census.gov report, 294.6 million Americans were insured in 2017. Ninety two percent of children under 19 and in poverty are insured. Insurance coverage is widespread and sustainable care models should focus on properly utilizing available insurance reimbursements.

Viewpoint 2

Insurance reimbursement rates are consistently decreasing meanwhile; the costs of operating medical clinics are rising. According to the American Hospital Association 2017 Fact Sheet, 63.9% of hospitals are losing money on Medicare patients and 22.6% of hospitals are losing money overall. In fact, there is an annual $57.8 billion dollar hospital deficit secondary to Medicare and Medicaid underpayment. Reimbursements are available for low-income patients, but the reimbursement rates are less than what it costs to provide quality care. Additionally, there is much uncertainty regarding the willingness of insurance companies to reimburse for mobile health services.

Viewpoint 3:

Avoiding declining insurance reimbursement in favor of receiving Affordable Care Act mandated community benefit allocations might be an amenable solution to sustaining a mobile vision clinic for Arkansas kids. As of 2013, each nonprofit hospital is required to conduct a community needs assessment and then make subsequent allocations to meet these community needs.

The Wills Eye Hospital has leveraged this mandated regulatory requirement to meet the vision care needs of underserved children! The Wills Hospital staff conducted a community needs assessment and subsequently funded and outfitted a screening van specifically for testing pediatric vision and dispensing glasses. All of these activities and costs are covered under mandated sustainable Community Benefit Funds.

Conclusion

The state of insurance coverage and the level of reimbursement to providers is constantly evolving. A paradigm shift is necessary to reach the 2 out of 3 Arkansan children who fail their school vision screening without follow-up. Relying on health insurance for reaching the underprivileged at this time, may be less stable than an ACA Community Benefit Based model.

However, ensuring these funds are allocated to pediatric vision outreach will require personnel dedicated to being Community Benefit Advocates. They must champion the cause of pediatric vision care to non-profit hospitals resulting in Community Benefit Fund allocations to the children's mobile vision clinic.

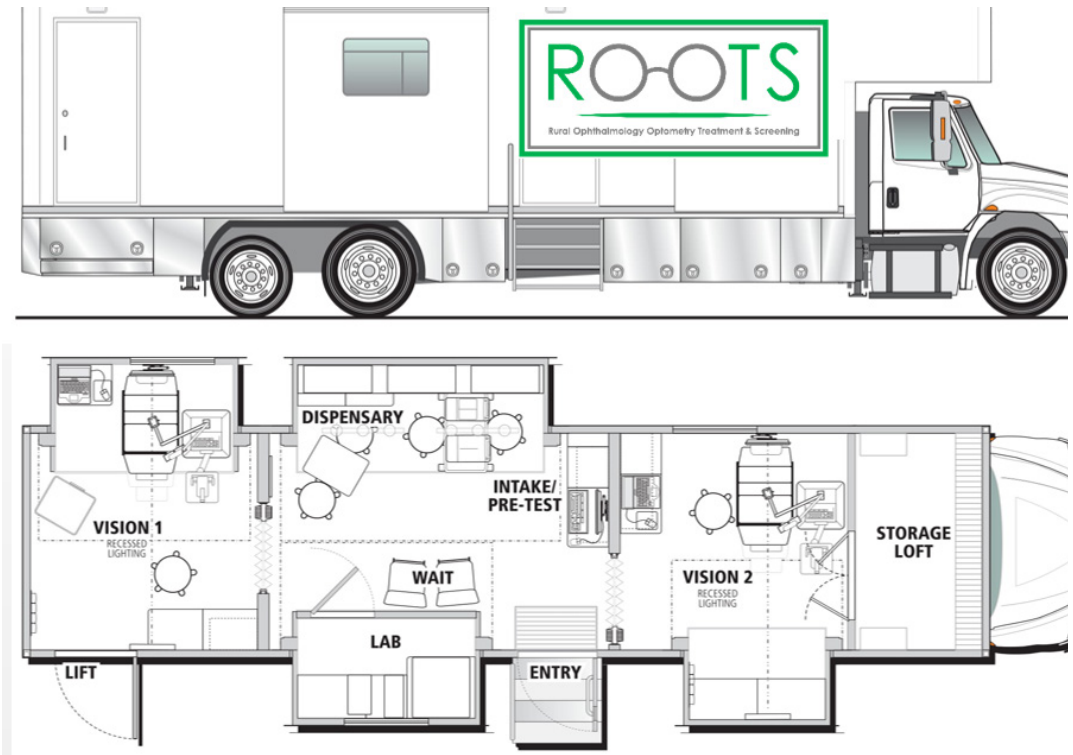

Figure 1. Mobile Vision Clinic Layout, Author: John Musser

Tactically, mobile clinics are ideal to overcome logistical and transportation barriers. A mobile vision clinic that served 15 children per day (5 days per week) could care for all 15,000 Arkansas children currently without care in fewer than 4 years. Interdisciplinary support from community leaders, parents, school nurses, clinicians, and ophthalmology and optometry professional societies will be critical in ensuring this solution is effective in reaching the children in need.

References

For a complete list of references, email ams@arkmed.org.

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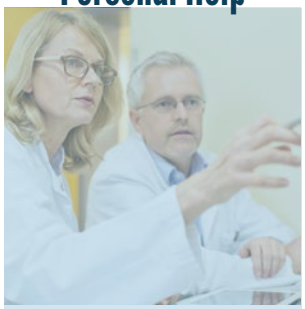

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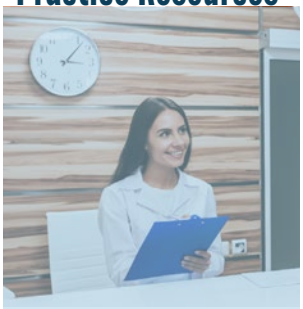

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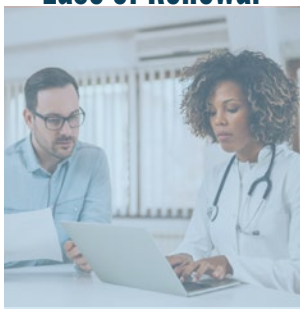

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Food Protein Induced Enterocolitis (FPIES): An Underrecognized and Often Misdiagnosed Pediatric Condition

Case Presentation

A six-month-old previously healthy infant presents to a walk-in clinic with acute onset vomiting that began 6 hours prior to presentation. She is exclusively breast-fed except for occasional oatmeal mixed with breast milk. The patient's mother reports that vomiting began about 2 ½ hours after she introduced a new iron-fortified multigrain cereal containing oat, barley, and wheat. The mother estimates that the child has vomited at least 8 times and expresses no interest in nursing. She has no evidence of urticaria, angioedema, or difficulty breathing. Additionally, the mother notes that the child has been somewhat lethargic since the vomiting episodes started and had an episode of mucous-containing diarrhea on the way to the clinic. Her medical history is significant only for mild eczema. Physical examination reveals a pale, somewhat listless child with mild tachycardia, delayed capillary refill, and no evidence of rash.

This patient has a clinical presentation consistent with food protein-induced enterocolitis syndrome (FPIES). FPIES is a non-IgE mediated food hypersensitivity that presents with intractable, forceful emesis that begins 1-4 hours after ingesting the offending agent with associated lethargy, pallor and sometimes watery diarrhea – occasionally with mucus or blood.[1,2] On some occasions, volume loss can be significant enough to cause hypotension and mimic shock leading to a potential medical emergency.

Background/Diagnosis

FPIES is pathologically different from IgE-mediated diseases, although some patients with FPIES have concurrent atopic disease as well. Skin prick testing and serum IgE testing for foods are not helpful in diagnosis of FPIES but can help rule out IgE-mediated food allergy if the history is not clear.[2] The delayed onset of symptoms, lack of typical IgE-related symptoms consistent with anaphylaxis, and poor recognition by the medical community can make the diagnosis of FPIES difficult.[1]

Diagnostic criteria were established in 2017 by the American Academy of Allergy, Asthma and Immunology with International Consensus Guidelines.[1] FPIES can be confirmed by patient having one major criterion and three or more minor criteria fulfilled [see Table I][1]. In most cases, history alone is sufficient for diagnosis and there are no laboratory tests or radiographic studies to confirm the diagnosis of FPIES.

FPIES was officially defined in the mid-1970s.[1] Prior to this there were no official uniform diagnostic criteria. Therefore, the prevalence estimates vary widely. Katz et al described CM-induced FPIES with an incidence of 3 per 1000 newborns over 2 years in a prospective birth cohort.[1] However, the incidence seems to be increasing and health providers need to be familiar with the manifestations, diagnostic criteria, and management of this disease[1].

The most commonly reported FPIES triggers in the United States are cow's milk (CM) and soy, though a trend towards solid foods, particularly grains, has been noted in some areas.[1,3,4] CM and soy FPIES typically present at an earlier age than solid food FPIES.[1] The most common solid food offenders for FPIES are rice and oat, likely because they are typically the first solid foods introduced.[1] Approximately 50% of infants who experience CM FPIES will react to soy and vice versa[1]. Up to 80% of children with solid food FPIES react to multiple foods and about 65% of these were previously diagnosed with either CM or soy FPIES.[1] Other implicated solid foods include avocado and banana3. The typical age of presentation for FPIES is 2-7 months of age, when either formula and/or solid foods are first being introduced.[1,2,3,4] Infants that present prior to 2 months of age are more likely to present with bloody stools and failure to thrive.[1] Interestingly, exclusive breastfeeding has been shown to have protective properties against FPIES.[1] It is rare for a child to present with FPIES after 1 year of age, though there are case reports of FPIES developing even into adulthood.[1]

Pathophysiology

The pathophysiology of FPIES is poorly understood. Studies have observed many different inflammatory mechanisms at work during FPIES reactions, indicating that the underlying immune response is quite complex. Some studies have shown that activation of the innate immune system (monocytes, natural killer cells, neutrophils) follows FPIES challenge, while others have pointed to evidence of a T-cell mediated inflammatory response.[1,5,6,7] Whatever the underlying cause of the inflammation, the result is an increase in intestinal permeability leading to significant fluid shifts. Attempts to use readily available laboratory studies to identify FPIES patients have largely failed given the paucity of concrete knowledge regarding the underlying pathophysiology. While

Table I – Diagnostic Criteria for Acute Food Protein Enterocolitis Syndrome (FPIES)

Major Criterion	Minor Criteria
Vomiting in the 1- to 4-h period after ingestion of the suspect food and absence of classic IgE-mediated allergic skin or respiratory symptoms.	1. A second (or more) episode of repetitive vomiting after eating the same suspect food.
	2. Repetitive vomiting episode 1-4 h after eating a different food.
	3. Extreme lethargy with any suspected reaction.
	4. Marked pallor with any suspected reaction.
	5. Need for emergency department visit with any suspected reaction.
	6. Need for intravenous fluid support with any suspected reaction.
	7. Diarrhea in 24 h (usually 5-10 h).
	8. Hypotension.
	9. Hypothermia
The diagnosis of FPIES requires that a patient meets the major criterion and >_3 minor criteria. If only a single episode has occurred, a diagnostic OFC should be strongly considered to confirm the diagnosis, especially because viral gastroenteritis is so common in this age group. Furthermore, although not a criteria for diagnosis, it is important to recognize that acute FPIES reactions will typically completely resolve over a matter of hours compared with the usual several-day time course of gastroenteritis. The patient should be asymptomatic and growing normally when the offending food is eliminated from the diet.	

not useful in acute diagnosis of FPIES (because many similar conditions share this finding), increases in the peripheral neutrophil count from baseline have been noted in patients who react during FPIES challenges.[9,10]

Management

Knowledge of the initial recognition and management of infants presenting with acute FPIES is of considerable importance. FPIES can mimic many other pediatric conditions from simple viral gastroenteritis in mild cases to overwhelming sepsis in more severe cases. It is not uncommon for an infant with a severe FPIES presentation to be admitted for a "sepsis rule-out", which can lead to unnecessary invasive studies and antibiotics. While the practitioner should keep all causes of shock in mind, a familiarity with the presentation of FPIES can lead to better identification of this group of patients.

The initial management of FPIES involves stabilization of the patient, assessment of the volume status, and prevention of further volume loss.[1,9] Because FPIES is not a histamine-mediated process, epinephrine and antihistamines have no role in treatment.[1,9] In severe cases, aggressive IV fluid resuscitation should be the first step in treatment followed by the administration of a non-sedating antiemetic like ondansetron to prevent further volume loss through emesis.[1,9,10.] A single dose of IV methylprednisolone (1mg/kg, 60mg maximum) has been theorized to provide benefit by stabilizing the inflammatory response; however, this has not been rigorously studied.[1,9,10] In the setting of severe or refractory hypotension, vasopressors and Intensive Care Unit admission may be indicated.[1,9,10] Mild to moderate FPIES reactions with a few episodes of emesis and no significant lethargy can be controlled at home at home or in clinic, as long as the child is willing/able to rehydrate with breastfeeding or clear liquids.[1,9,10] While the use of ondansetron has been shown to be beneficial in FPIES reactions in the hospital setting, there have not been large-scale studies to support its use in the home.[1,8,9,10] It has, however, become the practice of many practicing allergists to prescribe orally dissolving ondansetron tablets for use at the first sign of vomiting following an accidental ingestion. More studies are needed before FPIES management guidelines are changed to reflect this.[8]

The cornerstone of long term FPIES management is avoidance of the trigger food. As few large-scale prospective studies exist on FPIES, the majority of the management decisions regarding introduction of non-implicated foods, optimal times to challenge, etc. have been based on expert opinion. Fortunately, research into the natural history of FPIES is providing new information to better guide long-term management. For milk/soy FPIES patients, initial management involves removal of both milk and soy from the diet. Milk/soy formula should be replaced with a hypoallergenic formula.[1,9] While FPIES is rare in breastfed babies, when it does occur, exclusion of the culprit food from the maternal diet is not recommended if the infant is growing well and remains asymptomatic.[1] Solid food FPIES can present more of a challenge in terms of which foods to introduce and which to empirically avoid. This has become an area of further concern as studies like the LEAP study have promoted the benefits of early food introduction to prevent IgE-mediated food allergy. Confirmatory Oral Food Challenge (OFC) performed by an Allergist in a hospital setting are the gold standard for diagnosis but is not required for initial diagnosis in most cases, and should be conducted with caution since up to 50% of positive OFC results in IV rehydration treatment.[9,10] OFC should be reserved for those patients where history is unclear, though this is rarely performed following the initial presentation. FPIES OFC is more commonly performed to determine whether a patient has "outgrown" their reactivity to a specific food. These are performed at different ages for different FPIES triggers, because outgrowth occurs at different ages for different foods. Most allergists delay performing an FPIES challenge until after age 2, though occasionally milk/soy challenges are performed prior to this.[9,10] FPIES challenges are performed in a hospital setting, generally in a setting akin to a "day medicine unit." Intravenous access is established prior to commencing the challenge. The challenge food in question is ingested in a full serving at the beginning

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of the challenge and the child is monitored for up to 6-8 hours afterwards.[1,10] Many centers obtain blood samples throughout the challenge looking for a rise in the absolute neutrophil count which can signal an impending FPIES reaction. If the child does not experience FPIES symptoms by the conclusion of the challenge, he or she may then include the challenge food in the diet moving forward. If symptoms are experienced, management of the reaction is performed as above.

FPIES is a relatively recently described condition that is often misdiagnosed in the pediatric population. There are many reasons for this, including confusion with Ig-E mediated reactions or gastroesophageal reflux, and the frequency of viral gastroenteritis causing similar symptoms in this population. Therefore, it is important for the family to have an FPIES action plan, both to inform treating physicians of their condition as well as to provide treatment recommendations. It should be noted that this should serve a guide, but not exclude other diagnoses if the treating physician feels that other diagnoses are more likely. Increased awareness and recognition of this disease by emergency medicine and primary care practitioners is imperative both for acute symptom management and for appropriate referral to allergists for long term management, leading to increased quality of life for patients and their families.

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by AUSTIN MORGAN; CHRISTOPHER SCHACH, MD

Case

A 33-year-old Caucasian male presents to clinic with complaints of red, burning skin and pimple-like bumps on his forehead that have appeared within the past year. The patient states that his symptoms seem to wax and wane in severity, and he notices that they get noticeably worse after he eats spicy food or has been in the sunlight for an extended period of time. His past medical history is otherwise non-contributory.

On physical exam, the patient is found to have multiple erythematous papules and pustules distributed symmetrically across the forehead, glabellum, nose and cheeks. The attending physician decides to perform a scraping of the lesion with mineral oil preparation to ascertain a possible etiology. The mineral oil scraping is subsequently examined under the microscope and reveals the following organism.

Which of the following is the most likely diagnosis and associated organism?

A.) This is likely periorificial scabies; the culprit organism is Sarcoptes scabiei.

B.) These lesions are representative of rosacea; the associated organism is Demodex folliculorum.

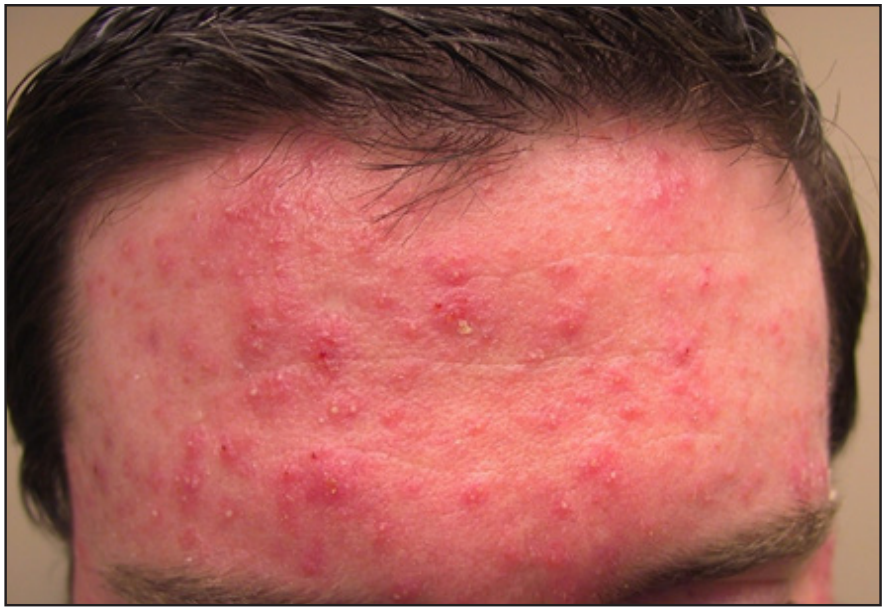
C.) This patient has extensive seborrheic dermatitis; the organism is Malassezia species.

D.) This is gamasoidosis (bird mite dermatitis); the causative organism is Dermanyssus gallinae.

Answer: B. Rosacea; Demodex folliculorum

Discussion

Acne rosacea, or simply rosacea, is an inflammatory skin condition that traditionally presents with erythematous papules and pustules, flushing, telangiectasias, burning, stinging and dryness distributed on the central third of the face. Those afflicted may also experience an irritated, gritty sensation of the eyes, or an enlarged, bulbous nose (rhinophyma). It is usually seen in middle aged individuals ranging from 30 to 60 years of age and can be sub-divided into four main types based upon the predominate symptomology: erythematotelangectactic, papulopustular, phymatous, and ocular. Classically, patients with rosacea will experience worsening of their symptoms with certain triggers, which include alcohol, caffeine, spicy foods, exposure to sunlight, stress and heat exposure.

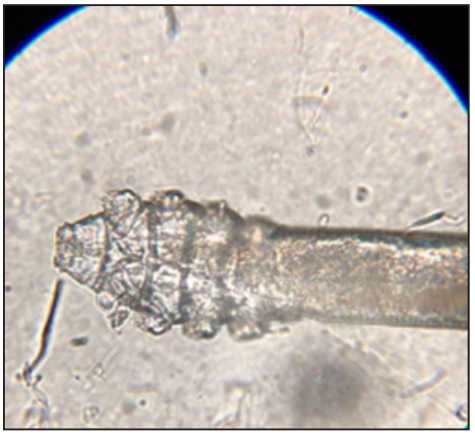


There are many postulated causes of rosacea, and the disease seems to arise from a complicated interplay between genetic predisposition, combined with vascular anomalies, environmental triggers and inflammatory factors. Of note, studies as early as 1932 (Ayres and Ayres) have suggested a possible influence of Demodex folliculorum on the development of rosacea, and subsequent studies have been performed that have corroborated this hypothesis.

The demodex mite is a normal inhabitant of the skin fauna but is often seen in higher concentrations in the pilosebaceous units and hair follicles in conditions such as rosacea. Specifically, multiple studies have shown that >5 mites per follicle or 5 mites/cm2 might play a role in the development of rosacea lesions. As of yet, it is unclear what impact this higher concentration of mites specifically has on the skin of affected individuals, but it is thought that the higher mite burden might trigger inflammatory cascades, either through physical blockage of the follicles, acting as vectors for bacteria that drive the disease process, or by some other process yet to be elucidated. In addition to rosacea, it has also been postulated that Demodex may contribute to the production of other, similar disease processes, such as acne vulgaris, pityriasis folliculorum, pustular folliculitis, and perioral dermatitis.

Treatment of rosacea can be difficult, and one of the most effective measures is simple avoidance of triggers. Other therapies include both topical and oral therapy, such as topical Metronidazole cream and oral doxycycline, depending on the severity of the disease process, as well as the subjective physical and emotional discomfort experienced by the patient. Demodex is now being specifically utilized as a therapeutic target. In fact, recent clinical trials have shown promising results with newer agents specifically targeted at the elimination or reconstitution of normal levels of Demodex on the skin surface of rosacea

patients. These acaricidal agents, namely ivermectin, have shown promising results in treating patients with mild-to-moderate papulopustular rosacea in particular. These topical preparations of Ivermectin are available as both ivermectin 0.5 % lotion (Sklice) or 1% cream (Soolantra), and are applied once daily to clean, dry skin. In clinical trials, once daily 1% Ivermectin has shown greater reduction in inflammatory lesions than twice daily topical metronidazole 0.75% cream. Even with the mounting evidence supporting this Mysterious interplay between the Demodex species, rosacea and other related disease processes, there is still much to be ascertained about the relationship between these entities.



Please welcome the following physicians who joined AMS in September and October 2020.

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McGehee

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Fayetteville

Emily Beckett, DO
Little Rock

Debbie Becton, MD
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