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**Safe Sleep Practices of Kansas Birthing Hospitals**

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**ABSTRACT**

**Introduction.** Sleep-related death is tied with congenital anomalies as the leading cause of infant mortality in Kansas, and external risk factors are present in 83% of these deaths. Hospitals can impact caregiver intentions to follow risk-reduction strategies. This project assessed the current practices and policies of Kansas hospitals with regard to safe sleep.

**Methods.** A cross-sectional survey of existing safe sleep practices and policies in Kansas hospitals was performed. Hospitals were categorized based on reported delivery volume and data were compared across hospital sizes.

**Results.** Thirty-one of 73 (42%) contacted hospitals responded. Individual survey respondents represented various hospital departments including newborn/well-baby (68%), neonatal intensive care unit (3%) and other non-nursery departments or administration (29%). Fifty-eight percent of respondents reported staff were trained on infant safe sleep; 44% of those held trainings annually. High volume hospitals tended to have more annual training than low or mid volume birth hospitals. Thirty-nine percent reported a safe sleep policy, though most of these (67%) reported never auditing compliance. The top barrier to safe sleep education, regardless of delivery volume, was conflicting patient and family member beliefs.

**Conclusions.** Hospital promotion of infant safe sleep is being conducted in Kansas to varying degrees. High and mid volume birth hospitals may need to work more on formal auditing of safe sleep practices, while low volume hospitals may need more staff training. Low volume hospitals also may benefit from access to additional caregiver education materials. Finally, it is important to note hospitals should not be solely responsible for safe sleep education.

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**INTRODUCTION**

In 2016, the state of Kansas had an infant mortality rate (IMR) of 5.9 per 1,000 live births.\(^1\) While infant mortality in Kansas has reached the Healthy People 2020 target of 6.0, sudden unexplained infant deaths (SUID) are tied with congenital anomalies for the leading cause of death.\(^1,2\) SUID includes deaths from unknown cause and sleep-related deaths, such as Sudden Infant Death Syndrome (SIDS) and accidental suffocation or strangulation in bed.\(^1\) In 2014, 83% of deaths attributed to SIDS had one or more factors contributing to an unsafe sleep environment.\(^3\)

The American Academy of Pediatrics (AAP) recommendations for a safe infant sleeping environment delineate a number of modifiable factors to reduce the risk of sleep-related infant deaths.\(^4\) Factors include back sleep only, room-sharing without bed-sharing, use of a firm sleep surface, keeping soft bedding and other items out of the crib, and avoiding infant overheating. The AAP also suggested healthcare providers, especially those in hospitals, model safe sleep recommendations.

In concert with AAP efforts, the Collaborative Improvement and Innovation Network to Reduce Infant Mortality (IM CoIIN) began a multiyear national movement in 2014 to reduce infant mortality and improve birth outcomes.\(^5\) One of the six strategic areas of focus includes improving safe sleep practices. The Kansas Infant Death and SIDS Network (www.safekidsks.org), in collaboration with Safe Kids Kansas (www.safekidskansas.org), is leading the Kansas Safe Sleep CoIIN Group’s multi-pronged effort to increase safe sleep practices. Efforts include building on previous work\(^6,7\) with maternal and infant health providers to enhance delivery of anticipatory guidance related to safe sleep.

Postpartum education on infant sleep can be provided in hospital settings through direct education and modeling proper infant safe sleep strategies;\(^8,9\) both impact adherence to safe sleep recommendations at home.\(^10,11\) For some groups at high risk for sleep-related death, such as African Americans,\(^12\) hospitals may be the main or only source of safe sleep education.\(^13,14\) However, hospitals are not consistent in their practice of the AAP infant safe sleep recommendations.\(^15\)

Hospital healthcare providers involved in infant care have a unique opportunity to influence infant safe sleep. The purpose of this project was to assess delivering hospitals’ activities related to safe sleep, specifically with regard to policies and practice.

**METHODS**

This was a cross-sectional survey of Kansas hospitals that were identified as having at least one delivery in the year prior to the study (2015). The Safe Sleep CoIIN group partnered with the Kansas Hospital Association (KHA), which sent the survey to these hospitals across the state.

No validated survey was identified; therefore, an instrument was created to ascertain current hospital practices related to infant safe sleep. Thirteen questions were developed by the CoIIN group. The final survey was reviewed for readability and content validity by the Medical Society of Sedgwick County Safe Sleep Taskforce, a group consisting of physicians, community members, and researchers. Surveys asked about safe sleep training for hospital staff, safe sleep hospital policy, following of AAP recommendations in the hospital, and education provided to patients. For those hospitals that endorsed having a safe sleep policy, respondents were asked about the contents of that policy. Finally, participants were asked about barriers to improving safe sleep practices in their hospitals and additional comments were invited at the end of the survey.

A cover letter was developed requesting the anonymous survey be forwarded to the most appropriate person, as only one response was
allowed per hospital. In January 2016, KHA emailed the cover letter and a SurveyMonkey® link to 74 hospitals. One reminder was sent in the same manner.

Responses were summarized with frequencies and percentages reported. For analysis, hospitals were divided into empirical categories based on reported volume. Differences between results for volume groups were evaluated using chi-squared tests. Number of items provided to parents to support safe sleep were compared across volume groups using the Kruskal-Wallis H-test. Analysis was performed using SPSS 23 [IBM Corp. Armonk, NY].

RESULTS
Thirty-one hospitals provided responses (42% response rate). Different hospital departments responded to the survey with most (68%) coming from the newborn/well-baby unit, and others coming from the neonatal intensive care unit (NICU; 3%) and other non-nursery departments (29%), including general staff and administrators from small rural hospitals. The median number of reported deliveries for surveyed hospitals was 100, but ranged from as high as 1800 to as low as zero. One respondent commented they “do not deliver babies unless they drop on our door step”. Fourteen hospitals (45%) delivered between 0 and 49 infants annually (low volume), 9 (29%) delivered between 50 and 500 (mid volume), and 8 (26%) delivered more than 500 (high volume).

In regard to training, 18 responding hospitals (58%) reported they provided infant safe sleep training to staff. Of these, 8 (44%) reported annual trainings. High volume hospitals (63%) were more likely to hold annual training as compared to low (7%) or mid (22%) volume hospitals ($x^2 (4, N =31) = 13.3; p = 0.001$).

Thirty-nine percent of hospitals (n = 12) reported a safe sleep policy. Of those, 10 (83%) had revised their policy following the 2011 guideline update. Mid (80%) and high volume hospitals (100%) reported keeping their policies up-to-date more frequently than low volume hospitals (50%), though this difference was not statistically significant ($x^2 (2, N =12) = 2.6; p = 0.267$). All hospitals with a policy reported that keeping soft items out of cribs was addressed and the majority addressed avoiding co-sleeping (Table 1). Policies differed by hospital size, with low volume hospitals less likely to encourage rooming in or back sleeping ($x^2 (2, N = 12) = 8.8; p = 0.001$). Avoiding co-sleeping of multiple birth siblings (i.e., twins) was lacking from 50% of policies.

Table 1. Policies and resources devoted to safe sleep for hospitals with a policy (n = 12), n (%).

| Which items are explicitly included in your department's safe sleep policy/guideline? | Birth volume* |
|---|---|---|
| | Low (n = 2) | Mid (n = 5) | High (n = 5) |
| Avoid co-sleeping | 2 (100) | 4 (80) | 5 (100) |
| Avoid co-sleeping of multiple-birth siblings | 1 (50) | 1 (20) | 4 (80) |
| Avoid infant overheating | 2 (100) | 3 (60) | 5 (100) |
| Back sleep only for infants | 1 (50) | 4 (80) | 5 (100) |
| Encourage rooming in | 1 (50) | 4 (80) | 5 (100) |
| Keep soft items out of crib | 2 (100) | 5 (100) | 5 (100) |

*Low: <50 births, Mid: 50 - 500 births, High: >500 births

Across all hospitals, discharge instructions (n = 23, 74%) and printed materials (n = 21, 68%) were the most common resources provided to patients (Table 2). Low volume hospitals had fewer resource provisions overall (median = 1.5) as compared to mid (median = 4) and high volume (median = 5) (H(2) = 9.5, p = 0.009); no low volume hospitals reported offering a safe sleep instructional video viewing and only 4 (29%) reported offering educational classes on newborn care. Notably, more mid volume hospitals (67%) offered viewing of a safe sleep video, than high volume hospitals (38%; $x^2 (2, N = 31) = 12.2; p = 0.002$).

Table 2. Educational/information items devoted to safe sleep by number of births, n (%).

| Which of the following education, information or items devoted solely or primarily to safe sleep practices does your department provide? | Birth volume* |
|---|---|
| | Low (n = 14) | Mid (n = 9) | High (n = 8) |
| Brochures and other printed materials | 7 (50) | 6 (67) | 8 (100) |
| Discharge instructions on safe sleep | 8 (57) | 8 (89) | 7 (88) |
| Educational messages on products (t-shirts, mugs) | 2 (14) | 1 (11) | 1 (13) |
| In-house wearable blanket | 3 (21) | 3 (33) | 5 (63) |
| Newborn classes | 4 (29) | 6 (67) | 5 (63) |
| Offered viewing of safe sleep instructional video | 0 (0) | 6 (67) | 3 (38) |
| Posters | 2 (14) | 3 (33) | 3 (38) |
| Required viewing of safe sleep instructional video | 1 (7) | 2 (22) | 0 (0) |
| Take home wearable blanket | 1 (7) | 1 (11) | 4 (50) |

*Low: <50 births, Mid: 50 - 500 births, High: >500 births

Most hospitals (71%) reported sometimes or always asking parents if they had a safe crib at home. Few (23%) reported referring parents to crib distribution programs. Two-thirds of hospitals (67%) reported never auditing compliance with their infant safe sleep policy. Two (13%) reported auditing weekly and two (13%) reported auditing annually. The remaining site (7%) reported occasional “spot checks”.

Respondents reported how frequently safe sleep recommendations were followed by staff (Table 3). Most hospitals reported always utilizing tight fitting sheets (96%) and back positioning (78%). Only one hospital (7%) reported “never” putting infants in a crib. Low volume hospitals used wearable blankets infrequently (50% reported never using), though most high volume hospitals (88%) used wearable blankets at least sometimes. A majority of hospitals (63%), irrespective of volume, reported always keeping toys and diapers out of cribs. Fewer (22%) reported always keeping blankets out of cribs.
A number of barriers to improving infant safe sleep were reported (Table 4). The top barrier, irrespective of hospital volume, was conflicting patient and family member beliefs. Lower volume hospitals also reported lack of appropriate educational materials, low awareness of infant safe sleep practices among nursing or medical staff, and language barriers as impediments to improving infant safe sleep practices. Mid volume hospitals reported language barriers, competing demands for staff, and nursing staff not always following infant safe sleep practices. High volume hospitals reported language barriers and nursing staff not always following safe sleep practices as their other major impediments.

### Table 3. Frequency of staff following safe sleep recommendations for hospitals with any births (n = 27), n (%).

| Safe Sleep Recommendation                  | Birth volume* |
|-------------------------------------------|---------------|
|                                           | Low (n = 10)  | Mid (n = 9)  | High (n = 8) |
|                                           | Always | Sometimes | Never | Always | Sometimes | Never | Always | Sometimes | Never |
| In a crib                                 | 7 (70) | 2 (20)    | 1 (10) | 9 (100) | 0 (0)     | 0 (0) | 7 (88) | 1 (13)    | 0 (0) |
| On the back/supine position               | 8 (80) | 1 (10)    | 1 (10) | 7 (78)  | 2 (22)    | 0 (0) | 6 (75) | 2 (25)    | 0 (0) |
| Tight fitting sheet                       | 9 (100) | 0 (0)     | 0 (0)  | 8 (89)  | 0 (0)     | 1 (11) | 8 (100) | 0 (0)     | 0 (0) |
| No blankets                               | 1 (10) | 7 (70)    | 2 (20) | 2 (22)  | 6 (67)    | 1 (11) | 3 (38) | 5 (63)    | 0 (0) |
| No toys or extra diapers in the crib      | 7 (78) | 2 (22)    | 0 (0)  | 5 (56)  | 4 (44)    | 0 (0) | 5 (63) | 3 (38)    | 0 (0) |
| Sleepsack/wearable blanket                | 2 (20) | 3 (30)    | 5 (50) | 2 (22)  | 4 (44)    | 3 (33) | 4 (50) | 3 (38)    | 1 (13) |

Note: Responses of “Not Sure” were treated as missing data; percent is calculated as percent of similar sized hospitals.

*Low: <50 births, Mid: 50 - 500 births, High >500 births

### Table 4. Barriers to improving safe sleep, n (%).

| What do you think are strong barriers to improving safe sleep practices in your department? | Birth volume* |
|----------------------------------------------------------------------------------------|---------------|
|                                                                                       | Low (n = 14)  | Mid (n = 9)  | High (n = 8) |
| Patient, family beliefs                                                                | 5 (36)        | 6 (67)       | 5 (63)       |
| Language barriers                                                                      | 2 (14)        | 5 (56)       | 3 (38)       |
| Nursing staff don’t always follow guidelines                                          | 1 (7)         | 3 (33)       | 3 (38)       |
| Competing staff priorities                                                             | 2 (14)        | 4 (44)       | 1 (13)       |
| Lack of appropriate educational materials                                               | 3 (21)        | 2 (22)       | 1 (13)       |
| Low awareness of safe sleep practices                                                  | 3 (21)        | 0 (0)        | 0 (0)        |
| Staff belief that co-sleeping improves bonding                                         | 1 (7)         | 1 (11)       | 1 (13)       |
| Physicians don’t always follow guidelines                                              | 0 (0)         | 2 (22)       | 1 (13)       |
| Not enough time to educate patients during stay                                        | 1 (7)         | 1 (11)       | 0 (0)        |

*Low: <50 births, Mid: 50 - 500 births, High >500 births

### DISCUSSION

As evident by the survey responses, hospital promotion of infant safe sleep is being conducted in Kansas to varying degrees. While the sample size is small, a number of interesting findings emerge. More than half of responding hospitals reported offering safe sleep training for staff, with high volume hospitals more likely to conduct annual trainings than mid or low volume hospitals. In contrast, fewer hospitals reported a safe sleep policy. All reported policies address keeping soft items out of cribs and high volume hospitals’ policies were more complete in terms of addressing the AAP guidelines. However, most hospitals reported never auditing policy compliance. Across all hospitals, discharge instructions and printed materials were the most common resources provided to parents. However, the quality and content of the discharge instructions were not assessed. The new AAP Safe Sleep guidelines and the National Action Partnership to Promote Safe Sleep (NAPPS; www.nappss.org) are advocating for enhanced conversations with parents regarding infant sleep to better address individual barriers. Finally, while most hospitals reported usually asking parents if they had a safety-approved crib available, very few refer parents to crib distribution programs.

Barriers to providing safe sleep education differed somewhat by hospital volume. However, regardless of volume, parent and family member’s beliefs are perceived to be a major barrier to improving infant safe sleep practices across hospitals. There are cultural norms to overcome with regard to infant safe sleep. Further, language barriers often were cited as a barrier by mid and high volume hospitals, while not endorsed as frequently in low volume hospitals. Mid and high volume hospitals also reported nursing staff not always following safe sleep practices. However, unlike low volume hospitals, no mid or high volume hospitals reported a lack of awareness of infant safe sleep practices.

There may be different remedies for improving infant safe sleep practice depending on the hospital birth volume. In reviewing the barriers to safe sleep implementation, low volume hospitals appeared to need more training and educational materials, while mid and high volume hospitals may need to work more on implementation of safe sleep practices. This is concordant with studies aimed at improving infant sleep environments in a hospital setting. One study in a large hospital (more than 6,000 annual deliveries) observed whether infants met the AAP guidelines for safe sleep. Prior to implementing a bundled intervention, only 25% of infants were observed in a safe
sleep environment. Another study reported that while 97% of nurses knew the AAP recommendations for safe sleep, only 67% agreed, and only 29% of the infants were found lying in the supine position, compared with 55% side-lying and 16% in the prone position.\(^7\)

Programs that incentivize hospitals or offer providers continuing education credits may increase the likelihood that safe sleep initiatives are introduced. For example, Cribs for Kids, a national safe sleep initiative, established a certification program to encourage hospitals to promote safe infant sleep.\(^8\) Three levels of certification are available: gold, silver, and bronze. To date, only two Kansas hospitals have received this certification, yet many of the survey respondents indicated activities that would qualify them for at least bronze certification. The Kansas Safe Sleep CoIIN group should explore ways to ensure hospitals are aware of the program and support efforts to obtain or renew certification.

While the results of this study may not be representative of all birthing hospitals in Kansas, the response rate of 42% is indicative of a good cross-section of hospitals, especially from respondents of newborn/well-baby departments. Additionally, while it may be desirable to have more respondents from mid and high volume hospitals, the inclusion of a sizable percentage of low volume hospitals provided unique insight into infant safe sleep practices of small and rural hospitals. This survey provides a solid first step to develop interventions and/or tools for the promotion of infant safe sleep.

Safe sleep education and promotion occur at varying levels in Kansas hospitals and interventions to improve safe sleep promotion appears to be associated with birthing volume. High and mid volume hospitals should adopt more formal auditing of safe sleep practices, implement tools to work across language barriers, and support nurse promotion of AAP guidelines. Low volume hospitals may benefit from staff training and access to additional caregiver education materials, such as showing a safe sleep video prior to discharge. Finally, hospitals should not be solely responsible for safe sleep education. Pre- and postnatal care providers and community programs also should promote consistent infant safe sleep messages to enhance the likelihood families will follow risk reduction recommendations.

**REFERENCES**

1. Bureau of Epidemiology and Public Health Informatics. Infant Mortality Kansas, 2016 Research Brief. Topeka: Kansas Department of Health and Environment, 2017.
2. Healthy People 2020. Washington, DC: US Department of Health and Human Services, Office of Disease Prevention and Health Promotion, 2016. Available at: http://www.healthypeople.gov/.
3. Kansas State Child Death Review Board. 2016 Annual Report (2014 Data). 2016. Available at: http://agks.gov/docs/default-source/documents/2016-scrb-annual-report.pdf?sfvrsn=8.
4. Moon RY, Task Force on Sudden Infant Death Syndrome. SIDS and other sleep-related infant deaths: Evidence base for 2016 updated recommendations for a safe infant sleeping environment. Pediatrics 2016; E88(5:e201602940. PMID: 27940805.
5. National Institute for Children’s Health Quality. Collaborative Improvement and Innovation Network to Reduce Infant Mortality (IM CoIIN). 2017. Available at: http://www.nichq.org/childrens-health/infant-health/coi-to-reduce-infant-mortality.
6. Kuhlmann Z, Kuhlmann S, Schunn C, et al. Collaborating with obstetric providers to promote infant safe sleep guidelines. Sleep Health 2016; 2(3):219-224.
7. Ahlers-Schmidt CR, Kuhlmann S, Kuhlmann Z, Schunn C, Rosell J. To improve safe-sleep practices, more emphasis should be placed on removing unsafe items from the crib. Clin Pediatr (Phila) 2014; 53(13):1285-1287. PMID: 24419267.
8. Mason B, Ahlers-Schmidt CR, Schunn C. Improving safe sleep environments for well newborns in the hospital setting. Clin Pediatr (Phila) 2013; 52(10):969-975. PMID: 23872346.
9. Colson ER, Levenson S, Rybin D, et al. Barriers to following the supine sleep recommendation among mothers at four centers for the Women, Infants, and Children Program. Pediatrics 2016; 118(2):e243-250. PMID: 16882769.
10. Colson ER, Bergman DM, Shapiro E, Leventhal JH. Position for newborn sleep. Associations with parents’ perceptions of their nursery experience. Birth 2001; 28(4):249-253. PMID: 11903213.
11. Thompson DG. Safe sleep practices for hospitalized infants. Pediatr Nurs 2005; 31(5):400-403, 409. PMID: 16295155.
12. Goodstein MH, Bell T, Krugman SD. Improving infant sleep safety through a comprehensive hospital-based program. Clin Pediatr (Phila) 2015; 54(3):212-221. PMID: 25670685.
13. Mathews TJ, MacDorman ME, Thoma ME. Infant mortality statistics from the 2013 period linked birth/infant death data set. National vital statistics reports; Vol 64, No 9. Hyattsville, MD: National Center for Health Statistics, 2015.
14. Nortthington L, Graham J, Winters K, Fletcher A. Assessing knowledge of Sudden Infant Death Syndrome among African American women in two Mississippi communities. J Cult Divers 2011; 18(3):95-100. PMID: 22073527.
15. Pickett KE, Luo Y, Lauderdale DS. Widening social inequalities in risk for sudden infant death syndrome. Am J Public Health 2005; 95(11):1976-1981. PMID: 16254121.
16. Voos KC, Terreros A, Larijmoore P, Leick-Rude MK, Park N. Implementing safe-sleep practices in a neonatal intensive care unit. J Matern Fetal Neonatal Med 2015; 28(14):1637-1640. PMID: 25212974.
17. Preece K, Hershberger CM, Kuehn D, Levett J. Infant sleep position. Nursing practice and knowledge. MCN Am J Matern Child Nurs 1999; 24(6):301-304. PMID: 10565145.
18. Cribs for Kids. Hospital Certification 2017. Available at: http://www.cribsforkids.org/hospitalinitiative/.

*Keywords: infant mortality, organizational policy, hospital, pediatrics, guidelines*
Neck Trauma and Extra-tracheal Intubation
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INTRODUCTION
Neck trauma can be devastating due to the relatively high probability of life-threatening pathology. Although direct neck injury is uncommon and only accounts for 1:30,000 emergency room visits, airway trauma remains a major cause of early death in trauma. Airway obstruction or associated injury (i.e., retropharyngeal hematoma) is estimated to account for a 30% early mortality rate. Early recognition and security of the airway remains a priority in resuscitating patients with airway compromise. However, there is limited evidence on how to manage traumatic airways. This is likely due to the unpredictable nature of the injuries themselves and the rarity of these events.

CASE REPORT
A 21-year-old male was brought to the emergency room as a level one trauma patient after suffering a suspected brain injury in a forklift accident. Upon arrival, emergency medical services remarked that the patient was initially pulseless and apneic, thus cardiopulmonary resuscitation had been initiated. His cervical spine was secured with a C-collar, and a Combitube™ placed to facilitate ventilation. Return of spontaneous circulation (ROSC) was achieved after ten minutes and two doses of epinephrine.

The patient had a Glasgow Coma Score of 3 and needed a more secure airway to facilitate imaging and possible operative intervention. He was oxygenated with 100% FiO2, and the Combitube™ was removed under visualization utilizing the Glidescope®. The airway was assessed while maintaining in-line cervical stabilization. Blood was seen throughout the oropharynx and through the glottic aperture. An 8.0 endotracheal (ET) tube was placed under visualization; placement was confirmed with positive end-tidal CO2 of 75. SaO2 reached 88% at the lowest point, but promptly improved with recruitment maneuvers. The patient was hyperventilated in an attempt to normalize CO2 and attenuate cerebral vasodilation that could lead a potential increase in intracranial pressure. Figure 1 depicts a chest x-ray showing the ET tube projecting over the trachea, but the tip appeared to be lateral to the tracheal lumen.

A computed tomography (CT) scan of the head showed no acute intracranial abnormality, but demonstrated extensive soft tissue gas throughout the neck bilaterally extending into the right face. A CT angiogram of the neck revealed multiple nondisplaced transverse process fractures and an apparent lack of vertebral artery flow bilaterally. A vascular surgery consultant suspected bilateral vertebral artery dissection with occlusion. A CT of the neck and chest (Figures 2 and 3) showed extensive pneumomediastinum, gas throughout chest, neck, and face, and, most notably, the ET tube was anterior to the trachea.

Figure 1. Chest x-ray depicts the ET tube placement.

Figure 2. Transverse images of a CT of the neck show path of the ET tube (arrow): A) CT Scan showing ET tube passing through glottis; B) ET tube moving inferior, anterior airway noted to have possible disruption; and C) ET tube is noted to be outside of the trachea (arrowhead).

Figure 3. Sagittal images of a CT of the neck show the ET tube location relative to trachea: A) note the location of the trachea and trajectory of the ET tube; B) the ET tube tip lies anterior to the trachea.
The anesthesia team learned of the mechanism of his injury at this time. He had leaned forward under the forklift crossbar, his knee hit a lever on his control board, lowering the forklift, and his neck was caught between the crossbar and a fixed strut. Witnesses reported his body lay on the lever, and they were unable to raise the lift off his neck for five minutes. Large contusions were noted on the anterior and posterior neck with significant subcutaneous emphysema.

The patient was transported emergently to the operating room to undergo a bronchoscopy through the endotracheal tube (ETT) to visualize the tracheal injury and reposition the ETT. The patient’s vital signs remained stable, including O₂ saturation remaining above 95%. Qualitative end-tidal CO₂ was positive. The patient was at high risk for loss of the airway, thus the surgical team was present during the entire procedure to provide a surgical one, if necessary. Bronchoscopy noted friable and erythematous tissue at the distal end of the ETT, and unknown particulate matter also was seen. The ETT was withdrawn until a slit area was noted in the right visual field; the bronchoscope was advanced into this opening. Tracheal rings were appreciated and the ETT was advanced toward the carina. A probable linear tear was visualized in the anterior trachea/larynx as the ETT was withdrawn. A Glidescope™ was inserted to enable visualization of the ETT superior to the glottis. The bronchoscope was reinserted anterior to the ETT, and bubbles of blood were noted distal to the glottis coinciding with an area of suspected tracheal perforation. Initial ETT cuff inflation likely served to tamponade local bleeding. The tracheal discontinuity could not be well-visualized due to the surrounding hemorrhage. Ventilation improved, oxygenation remained stable, and there was no hemodynamic collapse from known pneumomediastinum.

A follow-up CT of the neck was performed to reassess the patient’s cervical spine. It also showed continued tracheal placement of the ETT. Unfortunately, the patient sustained a severe anoxic brain injury. An electroencephalogram showed severe diffuse encephalopathy and findings consistent with coma. A magnetic resonance image (MRI) of the brain, 48 hours post-trauma, showed findings consistent with global hypoxic ischemic injury without evidence of herniation, hydrocephalus, or midline shift. An MRI of the cervical spine showed a transected cervical spinal cord injury. No improvements in neurological status were observed. His family pursued comfort care measures and organ donation according to the patient’s wishes.

**DISCUSSION**

Injuries to the airway are rare and account for less than 1% of emergency room visits. However, there remains limited evidence on how to manage a traumatized airway, and effectively securing it can be fraught with unforeseen challenges. This is likely due to the unpredictable nature of the injuries themselves and the rarity of these events. Early recognition and security of the traumatic airway remain vital in resuscitating patients.

Head and neck injuries can be complex, and typically, the neck can be divided into three zones to help with a differential diagnosis. Zone I is below the cricoid and has a relatively significant mortality rate of 12% due to high likelihood of injury to the great vessels. Angiography usually is recommended over manual exploration if the patient is stable. Zone II lies between the cricoid and mandible and consists of 60 - 75% of neck injuries. Zone III is the area superior to the mandible, primarily consisting of bony facial structures at risk of fracture and displacement.

Recognizing laryngeal injury early is vital in ensuring the proper treatment to sustain both life and phonation. In general, neck trauma is subcategorized into blunt and penetrating trauma. Laryngeal-tracheal separation can occur with both types and even following endotracheal tube placement. A completely transected cervical trachea has the risk of retracting into the mediastinum, likely necessitating immediate surgical intervention.

In our case, recognition of airway injury was delayed due to initial lack of information regarding mechanism of injury as well as the presence of a cervical collar that obscured evidence of neck trauma. In retrospect, bronchoscopy immediately following intubation could have been considered.

Neck injuries introduce a myriad of complications to airway security, including hemorrhage, aspiration, retropharyngeal hematoma, skull base fracture, and temporomandibular joint injury limiting mouth opening. Cervicothoracic vascular injuries are reported to occur in up to 25% of neck trauma. Cervical spine injury at C4-5 increases the risk of apnea secondary to loss of diaphragmatic innervation and neurogenic shock. Only a few other cases of pneumopericardium or pneumoperitoneum from hypopharyngeal perforation have been reported previously, which can increase the risk of cardiovascular collapse. Due to its infrequency, however, this risk may not warrant an extensive workup. Injury to the esophagus is more common in penetrating trauma than blunt trauma.

A current proposed airway strategy for trauma cases include: 1) the larynx and trachea must be clearly intact and in continuity versus partial separation or avulsions of these structures, 2) the airway should be visible to inspection by endoscopy in the emergency department or operating room, and 3) intubation requires a highly experienced physician. If available, fiberoptic laryngoscopy should be utilized to assess the airway patency, vocal fold mobility, and integrity of the pharynx and larynx.

Use of video laryngoscopy or lighted stylet reduce cervical neck motion when compared with direct laryngoscopy with a Macintosh blade and should be considered in trauma cases. If a definitive airway is unable to be established, an emergency cricothyroidotomy or surgical tracheostomy is warranted.

After securing the airway, ongoing debate continues for obligatory exploration versus selective exploration, some arguing for a more conservative approach with the latter. Ongoing case reports and series will build a foundation of literature pertaining to airway trauma. In time, sufficient information may be gathered to form a consensus with how to best approach these uncommon presentations to maximize the likelihood of meaningful survival.
REFERENCES

1. Schaefer SD. The acute management of external laryngeal trauma. A 27-year experience. Arch Otolaryngol Head Neck Surg 1992; 118(6):598-604. PMID: 1637537.

2. Jain U, McCunn M, Smith CE, Pittet JF. Management of the traumatized airway. Anesthesiology 2016; 124(1):199-206. PMID: 26517857.

3. Kummer C, Netto FS, Rizoli S, Yee D. A review of traumatic airway injuries: Potential implications for airway assessment and management. Injury 2007; 38(1):27-33. PMID: 17078954.

4. Bhojani RA, Rosenbaum DH, Dikken E, et al. Contemporary assessment of laryngotraheal trauma. J Thorac Cardiovasc Surg 2005; 130(2):426-432. PMID: 16077408.

5. Mandavia DP, Qualls S, Rokos I. Emergency airway management in penetrating neck injury. Ann Emerg Med 2000; 35(3):221-225. PMID: 10692187.

6. Paik JH, Choi JS, Han SB, Jung HM, Kim JH. Complete cervical tracheal transection caused by blunt neck trauma: Case report. Ulus Travma Acil Cerrahi Derg 2014; 20(6):459-462. PMID: 25541928.

7. Seims AD, Shellengerger TD, Parrish GA, Lube MW. Cervical tracheal transection after scarf entanglement in a go-kart engine. J Emerg Med 2013; 45(1):e13-16. PMID: 23473892.

8. Noorizan Y, Chew YK, Khir A, Brito-Mutanayagam S. A complete transection of larynx. Med J Malaysia 2009; 64(2):179-180. PMID: 20058586.

9. Senel AC, Gunduz AK. Retropharyngeal hematoma secondary to minor blunt neck trauma: Case report. Rev Bras Anestesiol 2012; 62(5):731-735. PMID: 22999465.

10. Omar HR, Helal E, Mangar D, Camporesi EM. Diaphragmatic paralysis following blunt neck trauma. Intern Emerg Med 2013; 8(4):359-360. PMID: 23400674.

11. Uscategui-Florez T, Martinez-Devesa P, Gupta D. Mucosal tear in the oropharynx leading to pneumopericardium and pneumomediastinum: An unusual complication of blunt trauma to the face and neck. Surgeon 2006; 4(3):179-182. PMID: 16764205.

12. Chouliaras K, Bench E, Talving P, et al. Pneumomediastinum following blunt trauma: Worth an exhaustive workup? J Trauma Acute Care Surg 2015; 79(2):188-192. discussion 192-183. PMID: 26218684.

13. Assimakopoulos D, Tsirves G. Blunt trauma of the larynx and pneumomediastinum. Otolaryngol Head Neck Surg 2009; 141(6):788-789. PMID: 19932857.

14. Schaefer SD. Management of acute blunt and penetrating external laryngeal trauma. Laryngoscope 2014; 124(1):233-244. PMID: 23804493.

15. Schaefer SD. The treatment of acute external laryngeal injuries. ‘State of the art’. Arch Otolaryngol Head Neck Surg 1991; 117(1):35-39. PMID: 1986758.

16. Turkstra TP, Craen RA, Pelz DM, Gelb AW. Cervical spine motion: A fluoroscopic comparison during intubation with lighted stylet, GlideScope, and Macintosh laryngoscope. Anesth Analg 2005; 101(3):910-915, table of contents. PMID: 16160183.

17. Turkstra TP, Pelz DM, Jones PM. Cervical spine motion: A fluoroscopic comparison of the AirTraq Laryngoscope versus the Macintosh laryngoscope. Anesthesiology 2009; 111(1):97-101. PMID: 19512871.

18. Turkstra TP, Pelz DM, Shaikh AA, Craen RA. Cervical spine motion: A fluoroscopic comparison of Shikani Optical Stylet vs Macintosh laryngoscope. Can J Anaesth 2007; 54(6):441-447. PMID: 17541072.

Keywords: wounds and injuries, neck injuries, airway management, intubation
Strongyloides Duodenitis in an Immunosuppressed Patient with Lupus Nephritis
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INTRODUCTION
Strongyloides stercoralis is an intestinal nematode acquired in the tropics or subtropics.1-3 In an immunocompetent host, infection with S. stercoralis produces a negligible or minimally symptomatic chronic disease. However, when immunosuppression impairs the host’s regulatory function, infection with S. stercoralis can lead to hyperinfection syndrome and disseminated strongyloidiasis, which can be fatal. Diagnosing strongyloidiasis early is important because the case fatality rate of hyperinfection syndrome in immunosuppressed patients is between 50 and 86%.3,5 We report the case of a woman from Laos with recently diagnosed lupus nephritis who presented with complaints of abdominal pain, vomiting, and diarrhea.

CASE REPORT
A 57-year-old Laotian female with a past medical history of systemic lupus erythematosus presented as a transfer from an outside facility for further management of a lupus nephritis flare. She was admitted at the outside facility five days before transfer due to complaints of shortness of breath on exertion, leg swelling, intermittent non-bloody watery diarrhea, nausea, vomiting, abdominal pain, bloating, and worsening proteinuria over two weeks. She had a history of lupus for the past few years. Due to worsening renal function, she had a renal biopsy three months prior to presentation which was consistent with lupus nephritis (stage V membranous nephropathy). She was placed on prednisone taper and mycophenolate mofetil two months prior at the outside facility. At presentation, the patient reported four days of melena but denied bloody stools. She did not report any recent travel outside the United States, any sick contacts, or recent antibiotic use.

On examination, the patient demonstrated pallor and alopecia. Cardiovascular and respiratory examinations were unremarkable, and her abdomen was soft without any tenderness or abdominal distension. Bilateral knee swelling and pedal edema were noted. Skin examination was normal. She had normal vital signs on admission at our hospital. Labs done on admission demonstrated normocytic anemia with hemoglobin of 9.8 gm/dl (reference range 12 - 15 gm/dl), leukocytosis with white blood cell count of 13.3 K/UL (reference range 4.5 - 11.0 K/UL, differential count not checked), elevated ESR of 41 mm/hr (reference range 0 - 30 mm/hr), elevated CRP of 1.64 mg/dl (reference range < 1.0 mg/dl), and normal renal and liver function tests. Urine culture was positive for Escherichia Coli, which was treated with appropriate antibiotics.

Due to melena and a two-gram drop in hemoglobin, upper and lower endoscopy was performed. Esophagogastroduodenoscopy (EGD) showed a large gastric submucosal mass with the appearance of cystic lesion in the cardia, which was not biopsied, and erythematous mucosa in the stomach and normal appearing duodenum (Figure 1), which was biopsied due to history of watery diarrhea. Duodenal biopsies showed marked duodenitis with parasitic organisms favoring duodenal strongyloidiasis (Figures 2, 3, and 4). Colonoscopy demonstrated a 4 mm cecal polyp, consistent with tubular adenoma, and 7 mm sigmoid colon polyp, which were removed. Colonoscopy also revealed normal ileum and colonic mucosa.

Following the diagnosis of duodenal strongyloidiasis by biopsy, the patient’s absolute eosinophil count was elevated at 1137/UL (reference range 0 - 450/UL). Strongyloides antibody (IgG) and Immunoglobulin E (IgE) were positive, Stool examination was positive for Strongyloides stercoralis. Sputum examination was negative, as was the CT scan of the chest, providing no definite evidence of disseminated disease. We did not find the source of melena but hemoglobin stabilized during the hospital stay. The patient was treated with 9 mg ivermectin for seven days and finally discharged on 9 mg ivermectin monthly for six months.

Two weeks following discharge, the patient was readmitted with complaints of melena, fatigue, and abdominal pain. Stool examination was negative for S. stercoralis, and peripheral eosinophilia had resolved. She was evaluated further with EGD for melena and noted to have gastric cardia submucosal mass and otherwise normal stomach. Duodenal biopsy obtained at that time did not show evidence of parasitic organisms.
STRONGYLOIDES DUODENITIS IN A PATIENT WITH LUPUS NEPHRITIS continued.

DISCUSSION

Roughly 100 million people are infected by *Strongyloides stercoralis* in tropical and subtropical areas. It is the fourth most common nematode infection in the world and is prevalent in Africa, Asia, Southeast Asia, Central America, South America, and parts of the eastern United States. Humans acquire strongyloidiasis when larvae penetrate the skin and migrate to reach the duodenum and upper jejunum to mature. The rhabditiform larvae develop into filariform larvae within the intestines, which may penetrate the colonic wall or perianal skin and complete an internal cycle (auto-infection). This phenomenon of autoinfection is responsible for the persistence of infection virtually indefinitely in infected hosts.

Chronic infection with *S stercoralis* is most often asymptomatic in an immunocompetent host. Hyperinfection syndrome manifesting as locally destructive bowel or lung disease and disseminated strongyloidiasis can occur in patients with impaired cell-mediated immunity (such as patients with transplant, patients receiving...
steroids or immunosuppressive therapy). Gastrointestinal and pulmonary symptoms are common but non-specific, and include abdominal pain, diarrhea, vomiting, dynamic ileus, small bowel obstruction (SBO) and protein-losing enteropathy, as well as pneumonia.3,10 Endoscopic findings of the duodenum in strongyloidiasis as reported by several prior case reports, includes normal mucosa, edema, erythema, erosion, swollen folds, fine granule, tiny ulcer, polyps, hemorrhage, megaduodenum, deformity, and stenosis.12

Definitive diagnosis of strongyloidiasis usually is made by detecting larvae in the stool, sputum, or tissue biopsy. A single stool examination is unable to detect larvae in up to 70% of cases, but repeat stool examinations have been shown to improve diagnostic sensitivity.13,14 Due to the need for multiple stool samples to detect *S. stercoralis* larvae, it is important to recognize that not identifying larvae in the stool does not imply an absence of infection. Upper and lower endoscopy also can establish the diagnosis of strongyloidiasis, as larvae may be seen on biopsies of the affected mucosa as noted in our patient.15 Thompson et al.16 showed that a minimum of six biopsies obtained from duodenal lesions resulted in a 100% histopathologic yield in diagnosing duodenal strongyloidiasis. Larvae can be demonstrated on normal appearing duodenal mucosa as shown in our patients and the endoscopist should consider biopsy of normal appearing duodenum if there is clinical suspicion of Strongyloides infection.

Ivermectin is the drug of choice as per World Health Organization.13 In disseminated disease, hyperinfection syndrome, and the immunocompromised patients, ivermectin is given daily until symptoms cease and stool samples are negative for *S. stercoralis* larvae for at least two weeks (1 autoinfection cycle). Diagnosing strongyloidiasis early is important, as almost all deaths due to helminths in the United States are due to *S. stercoralis* hyperinfection.17 It appears that our patient, an immigrant from an endemic area, was a chronic carrier of *S. stercoralis* and the recent treatment with steroids precipitated hyperinfection syndrome leading to marked duodenitis. Timely diagnosis and providing therapy without delay in our patient most likely decreased her mortality as case fatality rate of hyperinfection syndrome in patients with diminished cellular immunity is between 50 and 86%.13,5

In summary, we presented a case of hyperinfection syndrome with Strongyloides stercoralis with improved clinical outcome due to early intervention and therapy. Clinicians must have a high suspicion of *S. stercoralis* infection, especially in immunocompromised patients, as strongyloidiasis is difficult to diagnose and delaying therapy can have fatal consequences.

### REFERENCES

1. Liu LX, Weller PF. Strongyloidiasis and other intestinal nematode infections. Infect Dis Clin North Am 1993; 7(3):655–682. PMID: 8254165.
2. Genta RM. Global prevalence of strongyloidiasis: Critical review with epidemiologic insights into the prevention of disseminated disease. Rev Infect Dis 1989; 11(5):755–767. PMID: 2682948.
3. Berk SL, Verghese A, Alvarez S, Hall K, Smith B. Clinical and epidemiologic features of strongyloidiasis. A prospective study in rural Tennessee. Arch Intern Med 1987; 147(7):1257–1261. PMID: 3606282.
4. Lim S, Katz K, Krajden S, Fuku M, Keystone JS, Kain KC. Complicated and fatal Strongyloides infection in Canadians: Risk factors, diagnosis and management. CMAJ 2004; 171(5):479–484. PMID: 15337730.
5. de Silva T, Raychaudhuri M, Poulton M. HIV infection associated with Strongyloides stercoralis colitis resulting in Streptococcus bovis bacteremia and meningitis. Sex Transm Infect 2005; 81(3):276–277. PMID: 15923302.
6. Segarra-Newnham M. Manifestations, diagnosis and treatment of Strongyloides stercoralis infection. Ann Pharmacother 2007; 41(12):1992–2001. PMID: 17940124.
7. Siddiqui AA, Genta RM, Maguilnik I, et al. Strongyloidiasis. In: Guerrant RL, Walker DH, Weller PF (Eds). Tropical infectious diseases: Principles, pathogens & practice. Philadelphia: Saunders, 2011:805–812.
8. Schaeffer MW, Buell JF, Gupta M, et al. Strongyloides hyperinfection syndrome after heart transplantation: Case report and review of the literature. J Heart Lung Transplant 2004; 23(7):905–911. PMID: 15261189.
9. Grover IS, Davila R, Subramony C, Daram SR. Strongyloides infection in a cardiac transplant recipient: Making a case for pretransplantation screening and treatment. Gastroenterol Hepatol (N Y) 2011; 7(11):763–766. PMID: 22298974.
10. McNeeley DJ, Inouye T, Tam PY, Ripley SD. Acute respiratory failure due to strongyloidiasis in polymyositis. J Rheumatol 1980; 7(5):745–750. PMID: 7441665.
11. Pelletier LL, Gabre-Kidan T. Chronic strongyloidiasis in Vietnam veterans. Am J Med 1983; 78(1):139–140. PMID: 2966477.
12. Kishimoto K, Hokama A, Hirata T, et al. Endoscopic and histopathological study on the duodenum of Strongyloides stercoralis hyperinfection. World J Gastroenterol 2008; 14(11):1768–1773. PMID: 18550608.
13. Siddiqui AA, Berk SL. Diagnosis of Strongyloides stercoralis infection. Clin Infect Dis 2001; 33(7):1040–1047. PMID: 11528578.
14. Pelletier LL Jr. Chronic strongyloidiasis in World War II Far East prisoners of war. Am J Trop Med Hyg 1984; 33(1):55–61. PMID: 6696184.
15. Overstreet K, Chen J, Rodriguez JW, Wiener G. Endoscopic and histopathologic findings of Strongyloides infection in a patient with AIDS. Gastrointest Endosc 2003; 58(6):928–931. PMID: 14652568.
16. Thompson BF, Fry LC, Wells CD, et al. The spectrum of GI strongyloidiasis: An endoscopic-pathologic study. Gastrointest Endosc 2004; 59(7):906–910. PMID: 15173813.
17. Muenning P, Pallin D, Sell RL, Chan MS. The cost effectiveness of strategies for the treatment of intestinal parasites in immigrants. N Engl J Med 1999; 340(10):773–779. PMID: 10072443.

Keywords: Strongyloides stercoralis, strongyloidiasis, immunocompromised patients, lupus nephritis, esophagogastroduodenoscopy.
Arcanobacterium Brain Abscesses, Subdural Emphyema, and Bacteremia Complicating Epstein-Barr Virus Mononucleosis
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INTRODUCTION

Infectious mononucleosis is common among adolescents and young adults.1 Although most cases resolve spontaneously, several life-threatening manifestations have been recognized. No guidelines for management of mononucleosis have been published. However, the American Academy of Pediatrics recommends that a short course of corticosteroid therapy may be considered for certain serious complications such as impending airway obstruction, massive splenomegaly, hemolytic anemia, hemophagocytic lymphohistiocytosis, or myocarditis.2 Although some physicians prescribe prednisone for symptomatic treatment of other manifestations of mononucleosis, such as sore throat without airway obstruction, a recent Cochrane review identified no apparent benefit of steroid therapy for uncomplicated mononucleosis;3 such treatment is not recommended despite its widespread use.

Sinusitis is a well-recognized complication of viral infections, including mononucleosis. Corticosteroid therapy, even when only prescribed for brief periods, increases the risk of a variety of bacterial, viral, mycobacterial, and parasitic infections by 60%.4 Fardet et al. reported a 5.84-fold increased relative hazard of lower respiratory tract infections among patients who received systemic steroid treatment for any reason (the prevalence of upper respiratory tract infections including sinusitis was not studied).5

Arcanobacterium haemolyticum is a slowly growing, facultative anaerobic gram-positive bacillus.6 Originally named Corynebacterium haemolyticum when recognized as a cause of exudative pharyngitis in US soldiers and natives of the South Pacific in 1946, the organism’s name was changed to Arcanobacterium haemolyticum in 1982 after cell wall components and chemotaxonomic features were noted to differ from Corynebacterium spp. Humans are the primary reservoir for A. haemolyticum, which has two biotypes, rough and smooth. The smooth biotype is a cause of pharyngitis in healthy adolescents whereas the rough biotype is associated with a variety of invasive infections which are more commonly seen in immunocompromised adults.7 The aptly-named Arcanobacterium (“secret bacterium”) is difficult to cultivate with conventional culture techniques (i.e., incubation of specimens on sheep’s blood agar for 24 hours). One study reported that yield was highest when horse blood agar was utilized and specimens were incubated with supplemental 5% CO2 incubation for 48 hours.7 However, community laboratories do not typically use horse blood agar; thus the true prevalence of Arcanobacterium infections is likely higher than has been reported.

We report a case of Epstein-Barr Virus (EBV) mononucleosis treated with prednisone complicated by secondary bacterial sinusitis with intracranial extension leading to brain abscesses and subdural empyema, associated with Arcanobacterium bacteremia. To our knowledge this is the first report of EBV-associated Arcanobacterium haemolyticum brain abscess and subdural empyema.

CASE REPORT

A previously healthy 20-year-old African American male college football player developed sore throat, fatigue, and painful cervical lymphadenopathy. The diagnosis of infectious mononucleosis was established on the basis of a positive heterophile antibody test. One week later he had worsening sore throat and headache. Prednisone was prescribed for 10 days. Over the following two weeks he had recurrent fevers, chills, malaise, anorexia, and insomnia, but delayed seeking further medical attention because he was busy taking final examinations. His symptoms progressively worsened.

Three days prior to hospitalization he developed global headache, copious green rhinorrhea, recurrent rigors, and severe anorexia. He presented at a hospital emergency department where he appeared acutely ill. Posterior cervical lymph nodes were swollen and tender. No pharyngeal erythema or exudate was noted. No focal deficits were identified on neurologic examination.

His white blood cell count (WBC) was 16,300/µL (64% neutrophils, 24% bands, 7% monocytes, 5% lymphocytes). Total bilirubin was 5.7 µL (direct 4.0 µL), AST 410 U/L, ALT 290 U/L, and alkaline phosphatase 331 U/L. Epstein Barr virus viral capsid IgM serology was positive; viral capsid IgG serology was negative.

Computerized tomography scan of the head without contrast showed pansinusitis without intracranial masses, edema, or midline shift. Cerebrospinal fluid WBC count was 2,800/µL (46% neutrophils, 44% lymphocytes), glucose 44 mg/dL, and protein 100 mg/dL. Gram’s stain showed no organisms; culture, which was submitted prior to initiation of antibiotic therapy, was negative. Cerebrospinal fluid EBV PCR was positive (not quantified); Igs RNA was negative.

The patient was hospitalized and treated with ceftriaxone, doxycycline, and vancomycin. Doxycycline was discontinued after four days. Two of two sets of blood cultures grew Arcanobacterium haemolyticum; identification of the organism was confirmed by Matrix-assisted laser desorption/ionization (MALDI TOF) testing. Over the following four days the patient complained of worsening headache. MRI scan of the brain, with gadolinium, on hospital day five demonstrated...
multilocular fluid collections with enhancing margins in the left temporal lobe and posterior inferior left frontal lobe, consistent with abscesses, and a subdural fluid collection in the anterior and inferior left cranial fossa (Figure 1). Metronidazole was added to his treatment regimen.

The patient underwent image-guided pterional craniotomy with evacuation of frontal and temporal brain abscesses and removal of subdural empyema. Purulent fluid was encountered. Gram's stain showed few polymorphonuclear leukocytes and rare gram positive bacilli with morphology characteristic of Arcanobacterium; culture was negative. He also underwent bilateral maxillary antrostomy, total ethmoidectomy, frontal sinusotomy, sphenoidotomy, sinus irrigation, and submucosal reduction of the inferior turbinates. Thick mucopurulent fluid was encountered. Culture was negative.

Following evacuation of the brain abscesses and subdural empyema, the patient's clinical status improved substantially. His fever and headaches abated. He received a nine-week course of intravenous ceftriaxone and metronidazole. Repeat imaging at the completion of his treatment demonstrated resolution of intracranial abscesses. His infection has not recurred during a two-year follow-up period.

DISCUSSION

We report a case of EBV mononucleosis treated with prednisone complicated by secondary bacterial sinusitis with intracranial extension leading to brain abscesses and subdural empyema, associated with Arcanobacterium bacteremia. The dominant causative organism of the brain abscess and subdural empyema likely was Arcanobacterium: although abscess culture was negative following several days of antibiotic therapy, abscess fluid Gram's stain showed only gram positive bacilli with an appearance consistent with Arcanobacterium. We postulate that EBV infection caused disruption of upper respiratory tract epithelium, triggering secondary bacterial sinusitis with subsequent contiguous spread of infection to the brain and subdural space in the setting of transient immunosuppression precipitated by EBV and exacerbated by prednisone therapy. To our knowledge, this is the first report of EBV-associated Arcanobacterium haemolyticum brain abscess and subdural empyema.

Pharyngitis is the most commonly recognized form of A. haemolyticum infection. In half of cases a prominent erythematous rash is seen. Arcanobacterium accounts for 2% of cases of pharyngitis in 15 - 18 year old healthy males. Several other forms of Arcanobacterium infection have been reported, including sinusitis, pneumonia, bacteremia, meningitis, and brain abscess. Bacteremia is rare. Two cases were identified during a 15-year period at the Karolinska Institute. A similar experience was reported at Ben Taub Hospital in Houston, where among 280,000 blood cultures from 2000 - 2015, only two grew Arcanobacterium. Skin and soft tissue infections typically are seen in older men who either are immunocompromised or have an underlying disease such as diabetes mellitus.

The association of life-threatening Arcanobacterium infection with a concomitant Epstein Barr virus infection was of interest in our case. In invasive infections such as brain abscess, bacteremia, pneumonia, and Lemierre's syndrome, A. haemolyticum typically acts as a co-pathogen with other bacteria or viruses, for reasons that remain unclear. Skin and soft tissue infections typically are seen in older men who either are immunocompromised or have an underlying disease such as diabetes mellitus.

In a retrospective study, all six patients with Arcanobacterium bacteremia infection have been reported, including sinusitis, pneumonia, meningitis, and brain abscess. Three cases of A. haemolyticum pharyngitis are encountered most commonly in persons between the ages of 15 - 24, concomitant infection has been recognized infrequently. Three cases of A. haemolyticum bacteremia with primary EBV infection have been reported, one of which had secondary bacterial sinusitis and polymicrobial bacteremia. However, this association may be more common than has been widely recognized. A study of 13 patients with A. haemolyticum bacteremia detected heterophile antibody positivity in five cases.

Secondary bacterial infections (most commonly caused by beta hemolytic streptococci) are known to be associated with EBV, likely due to either temporary immune suppression or co-pathogenicity. Thus, transient humoral and cell mediated immunosuppression induced by EBV may enhance A. haemolyticum's virulence.

Figure 1. MRI scan of the brain, with gadolinium, demonstrated multilocular fluid collections with enhancing margins in the left temporal lobe and posterior inferior left frontal lobe, and a subdural fluid collection in the anterior and inferior left cranial fossa.
Steroid-induced immunosuppression would be expected to exacerbate the severity of EBV-Arcanobacterium coinfection.

Brain abscess can be caused by bacteria, mycobacteria, fungi, or parasites, and most commonly arises from either contiguous spread of infection, hematogenous seeding, penetrating trauma, or neurosurgical procedures.23 A variety of bacteria can cause brain abscesses; the implicated organisms vary based on the initial source of infection. Brain abscesses secondary to sinusitis, as in this case, usually are caused by anaerobic and aerobic streptococci, Bacteroides, E. coli, Enterobacter, Klebsiella, Proteus, S. aureus or Haemophilus.23 However in 13 - 25% of cases, brain abscess cultures are negative, likely secondary to antecedent antibiotic exposure or infection with non-cultivable microorganisms.23,25 Arcanobacterium haemolyticum is a rare cause of brain abscess; only six cases have been reported previously. Two of these cases were presumed odontogenic infections (one occurred post-dental extraction; a second patient had severe dental caries;17 a third occurred after penetrating skull trauma in a patient with acute sinusitis.15 The sources of the other three infections were not identified.14,24,25

The optimal treatment regimen for invasive A. haemolyticum infections has not been determined. Susceptibility testing is not standardized. In vitro data suggest that A. haemolyticum is susceptible to most classes of antibiotics, including penicillins, cephalosporins, clindamycin, carbapenems, macrolides, fluoroquinolones, tetracyclines, rifampin, and vancomycin, and resistant to sulfonamides.26 First line therapy with either penicillin or a cephalosporin is recommended. In β-lactam allergic patients, alternative options include clindamycin, doxycycline, fluoroquinolones, or vancomycin. Our patient had an excellent response to ceftriaxone therapy.

CONCLUSION

Arcanobacterium haemolyticum bacteremia, brain abscesses, and subdural empyema developed in a young man with concomitant EBV mononucleosis complicated by sinusitis who received prednisone therapy. The association between EBV and Arcanobacterium may be more common than previously recognized, as Arcanobacterium is a slowly growing organism that does not propagate well on commonly utilized sheep’s blood agar. Treatment with prednisone may increase the risk for serious bacterial complications, including brain abscess.

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REFERENCES

1. Luzuriaga K, Sullivan J. Infectious mononucleosis. N Engl J Med 2010; 362(21):1993-2000. PMID: 20605771.
2. Jackson MA, Long SS, Kanikante AC, de DW, Brady MT. (Eds.) Epstein-Barr Virus Infections (Infectious Mononucleosis). In: Red Book 2015; Report of the Committee on Infectious Diseases, 30th edition. Elk Grove Village, IL: American Academy of Pediatrics; 2015, pp.336-339. ISBN: 13-978-1-58110-926-9.
3. Rezk E, Nofal YH, Hamzeh A, Aboujaib MF, AlKheder MA, Al Hammad MF. Steroids for symptom control in infectious mononucleosis. Cochrane Database Syst Rev 2015; (11):CD004402. PMID:26538642.
4. Roostain L, Malvezzi P. Steroid-based therapy and risk of infectious complications. PLoS Med 2016; 13(5):e1002025. PMID:27218466.
5. Fardet L, Petersen I, Nazareth I. Common infections in patients prescribed systemic glucocorticoids in primary care: A population-based cohort study. PLoS Med 2016; 13(5):e1002024. PMID:27218256.
6. Carlson P, Lounatmaa K, Konttinen S. Biotypes of Arcanobacterium haemolyticum. J Clin Microbiol 1994; 32(7):1654-1657. PMID: 7929753.
7. García-de-la Fuente C, Campo-Esquisabel AB, Unja F, Ruiz de Alegria C, Benito N, Martín-Martínez L. Comparison of different culture media and growth conditions for recognition of Arcanobacterium haemolyticum. Diagn Microbiol Infect Dis 2008; 61(2):223-234. PMID:18325709.
8. MacKenzie A, Fuite LA, Chan FTI, et al. Incidence and pathogenicity of Arcanobacterium haemolyticum during a 2-year study in Ottawa. Clin Infect Dis 1995; 21(1):177-181. PMID:7578727.
9. Volante M, Corina L, Contucci A, Calò L, Artuso A. Arcanobacterium haemolyticum: Two case reports. Acta Otorhinolaryngol Ital 2008; 28(3):144-146. PMID: 18646577.
10. Therriault BL, Daniels LM, Carter YL, Raasch RH. Severe sepsis caused by Arcanobacterium haemolyticum: A case report and review of the literature. Ann Pharmacother 2008; 42(11):1697-1702. PMID: 18812563.
11. Stacey A, Bradlow A. Arcanobacterium haemolyticum and Mycoplasma pneumoniae co-infection. J Infect 1999; 38(1):41-42. PMID: 10090506.
12. Tan TY, Ng SY, Thomas H, Chan BK. Arcanobacterium haemolyticum bacteraemia and soft tissue infections: Case report and review of the literature. J Infect 2006; 53(2):e69-74. PMID:16316687.
13. Cortés-Perfield N, Kohli A, Weatherhead J, Ellsahly H. Arcanobacterium haemolyticum central nervous system abscess and bacteremia following head trauma: A case report and literature review. Infect Dis Clin Pract 2017; 25(3):e9-e11.
14. Chhung WH, Ayagari A, Sharma BS, Kak VK. Arcanobacterium haemolyticum brain abscess in a child (a case report). Indian J Pathol Microbiol 1999; 41(4):145-148. PMID: 1752643.
15. Ouriemichi W, Jedd D, El Quessar A, Benouda A. [Arcanobacterium haemolyticum brain abscess mimicking a brain tumor]. Med Mal Infect 2010; 41(7):397-399. PMID: 21440388.
16. Hedman K, Brauner A. Septicaemia caused by Arcanobacterium haemolyticum smooth type in an immunocompetent patient. J Med Microbiol 2012; 61(Pt 9):1328-1329. PMID: 2284353.
17. Lundblom K, Jung K, Kalin M. Lemierre syndrome caused by co-infection by Arcanobacterium haemolyticum and Fusobacterium necrophorum. Infection 2010; 38(5):427-429. PMID:20661621.
18. Skow RL, Sanden AK, Danchell VH, Robertson K, Ejlertsen T. Systemic and deep-seated infections caused by Arcanobacterium haemolyticum. Eur J Clin Microbiol Infect Dis 1998; 17(8):578-582. PMID: 9796659.
19. Goudswaard J, Van De Merwe DW, van der Sluys P, Doorn H. Corynebacterium haemolyticum septicaemia in a girl with mononucleosis infectiosa. Scand J Infect Dis1998; 20(3):339-340. PMID: 3406673.
20. Ginver LB, McGehee D, Taber LH, Stein F, Sumaya CV. Sinusitis, orbital cellulitis and microbacterial blood stream infection in a patient with primary Epstein-Barr virus infection. Pediatr Infect Dis 1984; 3(3):254-256. PMID: 6739327.
21. Bolis V, Karadodos C, Chiotis I, Chaliasos N, Tsabouri S. Atypical manifestations of Epstein-Barr virus in children: A diagnostic challenge. J Pediatr (Rio J) 2016; 92(2):113-121. PMID: 26802473.
22. Yonias F, Chua A, Tortora G, Jimenez VE. Lemierre’s disease caused by co-infection of Arcanobacterium haemolyticum and Fusobacterium necrophorum: A case report. J Infect 2002; 45(2):114-117. PMID: 12217717.
Brouwer MC, Tunkel AR, McKhann GM 2nd, van de Beek D. Brain abscesses. N Engl J Med 2014; 371(5):447-456. PMID: 25075836.

Altmann G, Bogokovsky B. Brain abscess due to Corynebacterium haemolyticum. Lancet 1973; 301(7799):378-379. PMID: 412979.

Washington JA, Martin WJ, Spiekerman RE. Brain abscess with Corynebacterium hemolyticum: Report of a case. Am J Clin Pathol 1971; 56(2):212-215. PMID: 5567728.

Carlson P, Korpela J, Waldor M, Nyman M. Antimicrobial susceptibilities and biotypes of Arcanobacterium haemolyticum blood isolates. Eur J Clin Microbiol Infect Dis 1999; 18(12):915-917. PMID: 10691208.

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Transgender Competent Provider: Identifying Transgender Health Needs, Health Disparities, and Health Coverage

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INTRODUCTION

Transgender individuals have gender identity, gender expression, or behavior that differs from their biological sex assigned at birth. It is estimated that 0.3% of US adults identify as transgender, which is nearly 1 million people. While much is unknown in the field of gender incongruence, “a growing and persuasive body of evidence suggests that biological factors have a substantial role in predisposing some people towards gender incongruence”. The earliest recognition of gender nonconformity in Western medicine was in the 1920’s and was labeled as a mental pathology. Gender Identity Disorder (GID) became a diagnosis in the American Psychiatric Association’s third edition of the Diagnostic Manual of Mental Disorders (DSM) in the 1980s. GID was a mental pathology until the DSM-5 was released in 2012, when GID was dropped as a diagnostic condition. Now, the DSM-5 has only an overarching diagnosis of gender dysphoria. This newly defined diagnosis does not pathologize being transgender, instead it can occur when an individual who is transgender has distress related to the incongruence between his/her experienced and expressed gender. Psychological treatment targets coexisting emotional and mental morbidities. Thus, the trend has shifted from attempting to treat gender nonconforming individuals to being more accepting of them.

To that end, this overview addresses what it takes to be a competent transgender provider, including knowing health needs, health disparities, and health coverage. Next, medical biases toward transgender patients are examined and current hormone treatment guidelines are outlined for both transgender men and women. Also, treatment options for transgender children and adolescents, along with aging patients, are discussed. The discussion concludes with trends, movements, and expectations for medical curricula.

Health Needs

Besides being a social minority, transgender individuals are a health minority who face myriad challenges as outlined in the National Transgender Discrimination Survey. The challenges outlined in the survey include facing serious acts of discrimination, such as loss of job, eviction, bullying, physical or sexual assault, denial of medical services, and incarceration. Barriers faced by transgender people in society and in the medical system make this group more likely to experience mental illness, discrimination, and poor health outcomes. For example, prevalence of psychiatric diagnoses appears to be high, especially for lifetime depressive episode estimated at 35.4%, and suicidality for transgender women at 20.2%.

Along with traditional health care, transgender individuals have unique needs related to gender transitioning, specifically mental health, hormonal treatment and side effects, and adequate referral to surgery, which can “create an undesired and unavoidable dependency on the medical system.” Procedures may include facial feminization/masculinization surgery, voice, breast, or chest surgery, hysterectomy, and genital reconstruction. Patients who experience obstacles in obtaining health services may experience short- and long-term negative health consequences. Of particular concern, one in four transgender women self-prescribe cross-sex hormones and obtain them illegally (most commonly via the internet), a practice that may be hazardous to health. This practice rarely was observed for transgender men.

Health Disparities

Healthy People 2020 outlines the need to research factors contributing to health disparities in the Lesbian, Gay, Bisexual, and Transgender (LGBT) community. A survey was conducted in 2008 by the National Center for Transgender Equality and the National Gay and Lesbian Task Force to understand these health disparities. Participants included 7,500 individuals. Results showed that 19% of respondents reported being denied healthcare based on their gender identity, and 28% reported verbal harassment in a medical setting. A third of respondents avoided preventive care, while 28% reported postponing any medical care due to discrimination and disrespect. Findings showed a higher than average likelihood of human immunodeficiency virus (HIV) among transgender patients. Respondents also were more likely to depend on drugs and alcohol to cope with negative experiences, while a higher likelihood of lifetime suicide attempts was revealed: 41% compared with 1.6% in the general population. This percentage was for both male and female respondents. Racial minorities who are transgender appeared to experience the highest risk for negative health outcomes and risk-taking behaviors.

Almost ten years later, the National Transgender Discrimination Survey was conducted with 6,450 transgender and gender non-conforming participants. Experiences of discrimination where equal treatment was denied was reported by 24% of participants in doctor’s offices or hospitals, 13% in emergency rooms, 11% in mental health clinics, and 5% for ambulance or emergency medical services. Participants also reported being harassed or disrespected at even higher rates for these same locations.

Results of these studies, among others, led the Association of American Medical Colleges (AAMC) to conclude that transgender health needs are not addressed adequately, and education for medical providers and staff about transgender health and cultural competencies is necessary. Thus, they convened an advisory committee in 2012 to develop a set of competencies for undergraduate medical education.

Health Coverage

In 2011, the Veterans Health Administration in the US Department of Veterans Affairs (VA) issued directives to support covering medical care and hormonal treatment for transgender patients,
Fifty percent of transgender patients reported having to educate their providers about transgender health in a 2014 survey. Medical school and residency curricula are lacking in transgender health. Culturally competent language and sensitive approaches to transgender individuals are lacking from most medical training. In 2014, only one third of US medical schools had any teaching or training in transgender health, and the ones that did may not be adequate to ensure enough knowledge to make providers comfortable to treat transgender patients. One theory for the discrepancy between patient need and medical availability is the belief among medical providers that transgender patients suffer from a psychological disorder. Although the DSM-54 has changed the terminology around transgenderism, it has kept it as a diagnosable condition that can occur when an individual who is transgender has distress related to the incongruence between his/her experienced and expressed gender.

Standards of Care

Standards of care (SOC) have been developed by the World Professional Association for Transgender Health (WPATH). WPATH standards include primary care, gynecologic and urologic care, reproductive options, voice and communication therapy, mental health services, along with hormonal and surgical treatments. A WPATH Mobile App is available at Google Play and iOS at the iTunes Store.

Hormonal Regimen for Transgender Men

Transgender males are assigned female sex at birth, but self-identify as male. Their hormonal treatment consists of administering testosterone to achieve maximum virilization. The levels are increased by pharmacological administration (intramuscular or patch) until the measured testosterone levels are within the normal male range (300 - 1000 ng/dl). Testosterone can be administered orally, however, that preparation is not available in the US. The most commonly used form in the US is intramuscular with doses of 50 - 200 mg weekly. Patients can go to bi-weekly dosing and administer higher doses (100 - 200 mg intramuscular) themselves, but the levels of testosterone fluctuate more with a bi-weekly regimen. Transdermal preparations (patch of 2.5 - 7.5 mg daily), testosterone 1% gel (2.5 - 20g/day), or intramuscular testosterone work, but may cause skin irritation and virilizing effects on quality of life. However, a scoping review observed that many published articles on the sexual health of transgender men come from limited data with potentially biased samples, citing controversial recruitment practices and high dropout rates, which may inflate the positive effects of therapy. In addition, a systematic review stated a need for new self-assessment tools to evaluate functional, psycho-relational, cosmetic, and quality of life of patients who undergo transsexual surgery.

Despite increased efforts to support transgender health, linking medical care to transition care remains the most common cause of coverage denial. Perhaps insurers will agree to enhance coverage after more research is conducted (with less biased samples and new evaluation tools) that verify the benefit of SRS.

Medical Biases toward Transgender Patients

Transgender individuals experience many barriers to healthcare. Among these were: 1) fear of being seen as different (with associated stigma and violence), 2) lack of access to caring and competent professionals, 3) difficulty in identifying sources of information about gender dysphoria and hormone therapies, and 4) inadequate access to safe prescribing and monitoring of hormone therapy. One of the most commonly reported barriers to healthcare identified by transgender individuals is their ability to find a knowledgeable provider. Fifty percent of transgender patients reported having to educate their providers about transgender health in a 2014 survey.

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side effects to family members contaminated with the gel or cream if patients are not careful.

Transgender men must be followed initially every three months to check for virilizing and side effects. The main effects observed are loss of periods (amenorrhea), increased facial and body hair, increased muscle mass with changed fat distribution to male pattern, increased acne, and increased libido. Deepening of the voice, enlarged clitoris (clitoromegaly), and male pattern hair loss occur to varying extent in different individuals and occur over the first year. Transgender men starting hormonal treatment after the age of 40 may see less virilizing effects.

Follow-up and Side Effects

Transgender males require follow-up every three months over the first year, then every six months the following year and yearly, if lab tests are within acceptable limits. Testosterone levels are obtained at every visit; the dose can be modified to achieve a level in the male range (300 - 1000 ng/dl). Hematocrit and lipid levels are monitored. A bone scan is obtained at the beginning of treatment if patients are at risk of osteoporosis or not achieving adequate levels, otherwise the bone scan screening starts at age 60. Transgender males with breast tissue and cervixes need appropriate screening.

Transgender males on testosterone will have reduced fertility and are less likely to get pregnant. Those effects are permanent even with stopping testosterone. Pregnancy is possible and should be explained to transgender males who engage in sexual activity with partners with sperm (fertile non-transgender men or fertile transgender women). Transgender men are also at the risk of contracting sexually transmitted infections with unprotected sex.

Hormonal Regimen for Transgender Women

Transgender women born with male biologic sex require a blockage of testosterone action (anti-androgen) besides increasing estrogen levels. Treating transgender women aims at decreasing testosterone to the female range (30 - 100 ng/dl) and obtaining an estrogen level not exceeding the physiological female range (< 200 pg/ml). Treating with anti-androgens, such as spironolactone, allows for lower doses of estrogen. Spironolactone is used orally in a dose of 100 - 200 mg daily, but may be used up to 400 mg if needed. Spironolactone is a mild potassium sparing diuretic, hence the need to monitor levels of potassium. Estrogen is administered orally (2.5 - 7.5 mg of estrogen or 17-beta estradiol at 2 - 6 mg) daily or intramuscularly (estradiol valerate 2 - 10 mg once a week or 5 - 20 mg every two weeks). Patches can be used (estradiol patch 0.1 - 0.4 mg, two times a week) if the transgender woman is at increased risk of thromboembolic disease.

Changes

Body hair decreased, skin was less oily, muscle mass decreased with redistribution of body fat in a female pattern, spontaneous erections decreased, and libido and breast development decreased within three to six months. Breasts reached peak size typically after two years of hormonal treatment.

Monitoring and Side Effects

Transgender women on hormonal treatment must be monitored with testosterone levels (suppressed to 30 - 100 ng/dl) and estradiol levels increased but remaining below 200 pg/ml. They also must be monitored for potassium levels due to the risk of having high levels with testosterone-blocking spironolactone. The levels of prolactin and triglycerides need checked with labs, since estrogen treatment may cause hyperlipidemia.

Transgender Children and Adolescents

Many pre-pubertal patients with varying features of gender dysphoria will become transgender teenagers. Most transgender teenagers experience gender identity conflicts as children. Children who identify with a gender different from one assigned at birth often become gay and lesbian adults and do not become transgender adolescents. The child's reaction to beginning puberty is often diagnostic. The guidelines recommend allowing puberty to start without medical intervention, however, if not desired, puberty can be blocked early on (at Tanner stages 2 and 3), and hormonal treatment started when the patient is deemed ready (age 18 or 16 with parental consent). Puberty suppression is done for patients who are non-transgender with precocious puberty to allow permanent short stature. Some medical professionals are familiar with puberty blockade, however, not for transgender patients. The trend for transgender health awareness and education targets this gap area to keep transgender teenagers from going through the “wrong puberty”.

Puberty suppression for transgender individuals reduces the risk of emotional and behavioral problems and increases functioning. A study of a simple one-hour transgender curriculum at a Boston University Medical School showed improved student willingness to treat transgender patients, and increased perceived knowledge and comfort level.

Assessment of a Teenager with Gender Dysphoria

Whereas the majority of preadolescent individuals seeking medical attention for gender dysphoria are born with male gender and identify as girls, the ratio in adolescence is close to a ratio of 1:1. One explanation for transgender girls attracting parental and medical attention is that society is more accepting and less alarmed about female-born individuals who dress and act in a masculine fashion. “Tomboy” is a term used for those individuals with no equivalent for their male-born counterparts. Regardless of when gender dysphoria manifests, the consistency, persistence, and insistence of a teenager with an identified gender that is not the one assigned at birth should be taken seriously. Suicidal ideation, depression, and self-medication increase remarkably at this age.

Ideally, a multidisciplinary team with a psychologist, social worker, and physician are available for those patients and families. It often is not the case and many teenage transgender patients avoid medical care and some will attempt conforming to their birth gender, typically without success, and many secondary emotional consequences.

A medical provider lacking the sensitivity and cultural competence to engage a transgender patient, especially a teenager, will miss signs of gender dysphoria and potentially cause harm by saying gender stereotypical things that alienate the patient further. Medical providers interacting with teenagers should establish a safe space and inform
patients about confidentiality in all matters, excluding homicidal and suicidal intentions. Once patients feel safe to discuss private matters, they likely will be willing to talk about any gender struggles they may be experiencing.

Competent clinicians who provide care for teenage patients are expected to screen for emotional and social stressors as part of an interview during their yearly well check. If an adolescent discloses concerns about gender, the clinician should be able to screen for gender dysphoria and be prepared to suggest resources to help the patient and their family. A mental health clinician is often the first place to obtain a diagnosis and suggest future steps. Gender dysphoria screening is not recommended in every teenage patient who is not gender conforming, but should be screened for well-being and personal struggles.

Once a clear and consistent gender identity is established and mental health providers, family, and patient agree on treatment regimen, hormonal replacement can start. Ideally, puberty would have been blocked and minimal secondary sexual characteristics of the birth gender are present. Puberty blockage usually is done with gonadotropin-releasing hormone (GnRH) analogues. Patients must be monitored with bone scans, as height and bone density growth slows during treatment with GnRH analogues. Puberty blockage allows for the need to use fewer hormones to establish the desired secondary sexual characteristics (facial hair, deep voice, and male body habitus in transgender females; breast development and female body habitus in male transgender) and has been correlated with less psychosocial negative outcomes in transgender teens. Progestins are used in the transgender male teenager to avoid menstruation and cyproterone can suppress erections and nocturnal emissions in female transgender teenagers. However, the latter medication can cause breast tissue development and is not recommended without ensuring that the transgender identity is well established for the teenager.

Parental or legal guardian consent is necessary in treatment for transgender teenagers. Consent is a problem for many teenagers with unsupportive families and has been associated with maladaptive behaviors, such as running away from home, and risky, self-destructive behaviors. Visibly gender non-conforming teenagers are vulnerable to harassment and hate crimes. The options of teenage transgender individuals are limited by their financial dependence on their families and abilities to become financially independent. Homeless transgender teenagers have a challenge in finding housing options, as they often are placed based on the gender of their birth.

**Aging Transgender Guidelines for Screening and Preventative Health**

Hormonal replacement has no age limit. However, transgender males who stop testosterone lose their facial and body hair, muscle mass, suffer from decreased libido, and develop hot flashes. It is not recommended to stop hormones if the patient has had resection of their birth gonads (post-gonadectomy) because both men and women are at risk of bone loss and symptoms similar to post menopause. Osteoporosis has been reported in both transgender men and women with poor hormone regimen. Surgical confirming procedures carry a higher risk for older patients, as do most major surgical procedures, and those risks should be discussed with the surgeon (surgical team) involved with gender confirmation surgery.

Transgender females on feminizing treatment should be screened for breast cancer if they have been on estrogen for 30 years and are older than 50. If those women have a strong family history of breast or ovarian cancer, screening should begin earlier. Screening is done with mammography. Yearly breast exams and self-exams for transgender women without family history of breast cancer are not recommended. Breast augmentation does not increase transgender women's risk of breast cancer, but may reduce the accuracy of mammography. Transgender women with prostate tissue should be screened with an exam for enlarged prostates, if they develop symptoms. Prostate-specific antigen is not a useful marker for transgender women on estrogen. Transgender women with prostates must be screened for prostate cancer and breast cancer. Feminizing hormones increase the risk of venous thromboembolic disease, cardiovascular disease, hypertension, and prolactinomas (which is when an adenoma, or a non-cancerous tumor, of the pituitary gland overproduces the hormone prolactin). More studies are necessary to clarify these increased risks.

Transgender males with a cervix need pelvic exams every one to three years after the age of 40. Transgender men may not have engaged in vaginal penetrative intercourse and examining the vagina, cervix, and uterus may be traumatic. It is recommended to delay those parts of the exam until a good connection is established between the patient and the examiner and the patient indicates readiness. Sedation can ease the exam and make it less painful. If the patient cannot tolerate pelvic exams, hysterectomy and oophorectomy are recommended. Transgender males over the age of 60 and taking testosterone for over five years need bone scans. If transgender males have been on testosterone over five years and they have other risks of osteoporosis, they should test at age 50. Calcium and vitamin D are recommended for transgender males due to the unknown effect of testosterone on bone density.

All transgender individuals should be screened for cardiovascular health. Cardiovascular risk factors should be decreased to a minimum before masculinizing or feminizing hormonal therapies. If a patient is at a high risk for cardiovascular disease, a stress test is indicated before treatment and when hormonal treatment is initiated, the patient should begin a low dose aspirin regimen. Patients should be screened for hypertension the same as the non-transgender patient population and blood pressure should be optimized to a goal of 130 mmHg systolic or less and 90 mmHg diastolic or less. Blood pressure should be monitored every one to three months after the onset of testosterone in the first year in transgender men on hormones, especially in ones with Polycystic Ovary Syndrome (PCOS). Transgender men and transgender women should have a lipid profile check annually. High cholesterol should be treated to a level of 3.5 mmol/L in patients...
with no risk factors and 2.5 mmol/L in those with additional cardiac risk factors. Patients taking estrogen have a higher risk of type 2 diabetes if a family history of diabetes occurs or a weight gain of over 11 pounds. Transgender women should be screened annually with fasting blood sugar. For transgender men, screening is indicated with history of PCOS, otherwise diabetes screening guidelines are the same as those of the general population.

All transgender individuals with risk behaviors for sexual transmitted infections or blood transmitted infections (unprotected penile-vaginal or penile-rectal intercourse, sharing needles) should be screened for HIV and Hepatitis B and C every six months.\(^5\) For transgender patients without those risk behaviors, screening once in a lifetime is recommended. All transgender patients need Hepatitis B vaccinations. All transgender patients should be screened for depression (e.g., Patient Health Questionnaire) and, if the screen is positive, referred to a transgender competent mental health provider.

**Trends, Movements, and Expectations**

Despite the growing evidence supporting the value of treating transgender patients in a mindful, supportive setting, the transgender health curriculum in most medical provider curricula remains unchanged. The AAMC has incorporated transgender educational material on MedEdPORTAL (https://www.mededportal.org/collections) with over 100 articles, presentations, and tools. Hospitals lack transgender policies and the medical environment remains a haunting experience for many transgender individuals. Health care provider attitudes toward transgender patients show bias and lack of perceived knowledge to treat them.

The transgender health movement is young, yet showing positive changes in health care learners when proper exposure and education take place.\(^2\) Although there remains a lack of medical education programs for transgender health, research supports introducing this topic early during clinical education of clinicians.\(^3\) Until transgender health care and cultural competence are mandated in medical curricula, the multiple significant obstacles remain facts in the health of over one million transgender people.\(^5\)

**REFERENCES**

1. Gallup, Inc. In US, More Adults Identifying as LGBT. Social Issues. http://www.gallup.com/poll/19731/lgbt-identification-rises.aspx. Accessed May 19, 2017.
2. Winter S, Diamond M, Green J, et al. Transgender people: Health at the margins of society. Lancet 2016; 388(10042):590-400. PMID: 27323925.
3. Stromsoa D. The state of transgender health care: Policy, law, and medical frameworks. Am J Public Health 2010; 104(3):e31-8. PMID: 24432926.
4. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, 5th Edition: DSM-5. Washington, DC: American Psychiatric Publishing, 2013. ISBN: 0890425538.
5. Grant JM, Mottet LA, Tanis J, Harrison J, Herman JL, Keisling M. Injustice at every turn: A report of the national transgender discrimination survey. Washington, DC: National Center for Transgender Equality and National Gay and Lesbian Task Force, 2011.
6. Reisner SL, Biello KB, Huhto JM, et al. Psychiatric diagnoses and comorbidities in a diverse, multiency cohort of young transgender women: Baseline findings from project LifeSkills. JAMA Pediatr 2016; 170(5):481-486. PMID: 26999485.
7. Colebunders B, Brondeel S, D’Arpa S, Hoebeke P, Monstrey S. An update on the surgical treatment for transgender patients. Sex Med Rev 2017; 5(1):103-109. PMID: 27623991.
8. Mepham N, Bouman W, Arcelus J, Hayter M, Wylie K. People with gender dysphoria who self-prescribe cross-sex hormones: Prevalence, sources, and side effects knowledge. J Sex Med 2014; 11(12):2995-3001. PMID: 25723018.
9. US Department of Health and Human Services. Healthy People 2020. Lesbian, gay, bisexual, and transgender health. https://www.healthypeople.gov/2020/topics-objectives/topic/lesbian-gay-bisexual-and-transgender-health. Accessed May 19, 2017.
10. St. Cloud T. Implementing curricular and institutional climate changes to improve health care for individuals who are LGBT, gender nonconforming, or born with DSD: A resource for medical educators. Washington DC: Association of American Medical Colleges, 2014. http://members.aamc.org/eweb/upload/Executive%20LGBT%20 FINAL.pdf. Accessed May 23, 2017.
11. US Department of Veterans Affairs. Providing health care for transgender and intersex veterans. https://www.va.gov/vhapublications/ViewPublication.asp?pub_ID=2863. Accessed May 24, 2017.
12. US Centers for Medicare & Medicaid Services. Proposed decision memo for gender Dysphoria and Gender Reassignment Surgery (CAG-00446N). https://www.cms.gov/medicare-coverage-database/details/nci-proposed-decision-memo-ncsi-cag-00446n. Accessed May 24, 2017.
13. US Center for Medicare & Medicaid Services. Chapter I: Coverage determinations, part 2: Section 1403: Transsexual surgery. In: Medicare National Coverage Determination (NCD) Manual, Baltimore, MD: US Department of Health and Human Services, Centers for Medicare and Medicaid Services, 2012.
14. Rametti G, Carrillo B, Gomez-Gil E, et al. White matter microstructure in female to male transsexuals before cross-sex hormonal treatment. A diffusion tensor imaging study. J Psych Res 2011; 45(2):199-204. PMID: 20562024.
15. Deutsch MB. Guidelines for the primary and gender-affirming care of transgender and gender nonbinary people. 2nd edition. Center of Excellence for Transgender Health, www.transhealth.ucsf.edu/guidelines. Accessed May 16, 2017.
16. Bartlett NH, Vasey PL, Bukowski WM. Is Gender Identity Disorder in children a mental disorder? Sex Roles 2000; 43(11-12):753-785.
17. De Vries AL, Steensma TD, Doreleijers TA, Cohen-Kettenis PT. Puberty suppression in adolescents with gender identity disorder: A prospective follow-up study. J Sex Med 2011; 8(8):2276-2283. PMID: 21044177.
18. Safer JD, Pearce EN. A simple curriculum content change increased medical student comfort with transgender medicine. Endocr Pract 2013; 19(4):633-637. PMID: 23425566.
19. Goldberg JM, Matte N, MacMillan M. Community survey: Transition/ crossing services in BC—Final report. Vancouver Coastal Health and Transgender Support & Education Society, http://www.spectrumwyny.org/info/bsurvey.pdf. Accessed May 23, 2017.
20. US Department of Health and Human Services, Gay and Lesbian Medical Association, Healthy People 2010: Companion document for lesbian, gay, bisexual, and transgender (LGBT) health. Ann Arbor, Michigan: Gay and Lesbian Medical Association, 2001.
21. Sperber J, Landers S, Lawrence S. Access to health care for transgendered persons: Results of a needs assessment in Boston. Int J Transgend 2005; 8(2-3):75-91.
22. Kelley L, Chou CL, Dibble SL, Robertson PA. A critical intervention in lesbian, gay, bisexual, and transgender health: Knowledge and attitudes outcomes among second-year medical students. Teach Learn Med 2008; 20(3):248-253. PMID: 18615302.
23. World Professional Association for Transgender Health. Standards of care for the health of transgender patients and gender nonconforming people. http://www.wpath.org/site_home.cfm. Accessed May 24, 2017.
24. Bockting WO, Miner MH, Swinburne Romine RE, Hamilton A, Coleman E. Stigma, mental health, and resilience in an online sample of US transgender population. Am J Public Health 2013; 103(5):943-951. PMID: 23488522.
25. Gorin-Lazard A, Baumstarck K, Boyer L, et al. Is hormonal therapy associated with better quality of life in transsexuals? A cross-sectional study. J Sex Med 2012; 9(2):531-541. PMID: 22154968.

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The Increased Vulnerability of Refugee Population to Mental Health Disorders

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INTRODUCTION

Around the world, the number of refugees displaced by war or violence reaches over 19 million. Rates of mental health disorders, such as anxiety disorders, post-traumatic stress disorder (PTSD) and depression were higher among refugee populations in comparison to the general population. This increased vulnerability has been linked to experiences prior to migration, such as war exposure and trauma. Additionally, anxiety and other mental health disorders can manifest due to stressors post-migration, such as separation anxiety and the added load of resettlement in a new country. In general, increased rates of these disorders remain prevalent in refugee populations long after resettlement; however, some studies have shown otherwise.1

In the Karen refugees along the Burmese-Thai border, depression and anxiety rates (41% and 42%, respectively) were higher than the average rates of depression and anxiety among the general US population (7% and 10%, respectively).2 These rates have been linked to traumatic events like violence, harassment, and a lack of basic needs. Moreover, the mental health of refugees is thought to be distinct from the experiences of other traumatized populations, such as veterans and sexual assault victims, due to their unique traumatic experiences as well as acculturative stress that follows the resettlement process, which features entirely new settings, practices, and a lack of familiar support systems.3 Furthermore, this population showed a correlation of depression and anxiety disorders with post-resettlement hardships in regards to finding employment and adapting to a new environment culturally and linguistically. In another population, 82.6% of Cambodian refugees residing in a refugee camp on the Thailand-Cambodia border self-reported depression. Fifty-five percent were confirmed by the Hopkins Symptoms Checklist to have experienced symptoms of major depression.2

Symptoms of depression include changes in weight, sleep pattern, exhibiting a depressed mood for much of a day, a loss of interest in activities, lack of energy, feelings of worthlessness and guilt almost daily, lack of focus, and recurrent thoughts of death and suicide, which can include attempting or creating plans for suicide. Symptoms of PTSD include intrusion, avoidance, and hyperarousal. PTSD typically is associated with traumatic experiences. These traumatic events can include experiencing war, being held prisoner/hostage, torture and physical violence, death of a loved one, serious accidents/explosions, sexual harassment, and serious illness. Symptoms of generalized anxiety disorder include restlessness, irritability, fatigue, excessive worrying, having trouble relaxing, sleeping, and focusing.1

The current refugee demographic is a highly heterogeneous group, however, there has been an increase of refugees from Arabic speaking countries in recent years. Europe, in particular, has seen a large increase of asylum applicants from Arabic speaking countries, the most frequent being Syria (35.9% of applications) and Iraq (6.9% of applications). Despite the growth of Arabic speaking refugees, few studies have investigated the mental health of these populations in recent years. The large variations in results show that the refugee population is a diverse group. Complications in studies that inhibit direct comparison between refugee populations include the use of different psychometric instruments to measure mental health.5

Another factor that could promote the symptoms of PTSD, depression, and anxiety is acculturative stress. While trauma related to war negatively impacts mental health, the effects of acculturative stress on mental health among refugees resettled in Australia and Austria demonstrated that stress which accompanies the migration process can have similar effects. One cause of these stressors is acculturation, the process of integrating into a new culture while also maintaining one’s origin culture and identity. This process is dependent on the attitudes of both the migrant and host groups. There are inconsistencies present in existing studies investigating the effects of acculturation on mental health; however, acculturative stress in migration has been identified as a mental health risk factor.6

The purpose of this review was to investigate the relationship between refugee populations and their increased vulnerability to post-traumatic stress disorder, depression, and anxiety disorders. This study also examined the factors before, during, and after the migration process associated with increased vulnerability of refugees to mental health disorders.

Refugee Health

In a particular population of Yazidi refugees, Nasiroglu et al.7 determined the frequency of post-traumatic stress disorder and depression among children and adolescents and examined the possible differences in experience and diagnosis between males and females. Big differences existed in the resulting diagnosis between children and adolescents. Children generally had fewer problems with mental illnesses than adolescents, who may have increased stress related to having more siblings. Adolescents had more siblings, on average, than children. Other risk factors for depression, in particular, included having older parents, being female, and witnessing someone undergoing a violent or fatal situation. In terms of gender, females of both the children and adolescent groups were significantly more likely to have an established diagnosis, as compared to males, who in general, did not have one.7
To examine the mental health of Yazidi children and adolescents further, Ceri et al.\textsuperscript{8} investigated the presence of psychiatric disorders immediately following forced migration. Various disorders, not only PTSD, had manifested in the refugee population within the early days of resettlement. Children who experienced forced migration exhibited more behavioral and emotional problems than children who had not experienced such trauma. Following forced migration, children were observed to be very shy after their arrival to the camp and avoided contact with other children. Additionally, they communicated fears of being captured and generally did not feel safe in their new environment. Most children also had difficulty sleeping. Over one-third of the children were diagnosed with depressive disorder.\textsuperscript{9}

Factors that could be associated with psychiatric symptoms and disorders were torture and other traumatic events. Civilians in warzones typically experienced at least one traumatic event due to war, and war refugees often were subjected to torture. Among Syrian Kurdish refugees, there were positive correlations between PTSD symptoms and traumatic events such as being forced to flee one’s country, witnessing violence, and confinement due to violence. Moreover, while males were more likely to experience trauma, females were more likely to have symptoms of PTSD. However, Syrian Kurdish refugees in the Kurdistan region of Iraq displayed no significant difference in the prevalence of PTSD among males and females, which may be a result of cultural differences.\textsuperscript{9}

Refugee populations who have experienced traumatic events often are vulnerable to increased symptoms if they experience another stressful event. Thus, it has been investigated whether new traumatic or stressful events affect mental health of an already PTSD diagnosed individual. Schock et al.\textsuperscript{10} studied refugees from Iran, the Balkan region, and Turkey. All participants were diagnosed with PTSD. Groups that experienced a new significant life event displayed increased avoidance behavior. Such behavior may be a mechanism for these individuals to avoid re-experiencing their past trauma. Additionally, stressful life events affected symptoms more than traumatic life events. Overall, new significant life events resulted in a significant increase in PTSD symptoms, especially avoidance.\textsuperscript{10}

Furthermore, refugees diagnosed with PTSD often were diagnosed with secondary psychotic features as well. Nygaard et al.\textsuperscript{3} found 74 of 181 refugees (41%) diagnosed with PTSD were identified to have secondary psychotic features. These secondary psychotic features included hallucinations and delusions, and the impact of these features can make PTSD with Secondary Psychotic features (PTSD-SP) a burdening disorder. Refugees are uniquely vulnerable to developing secondary psychotic features with PTSD, as these features are assumed to manifest because refugees usually are subjected to more long-term trauma than other PTSD patients. Moreover, refugee populations often lack familiar support systems as they seek asylum abroad to escape their threatening situations, exacerbating the problem.\textsuperscript{3}

**Prior to Migration**

Prior to the migration process, there are environmental factors that can be associated with the development of mental disorders. In Ethiopian immigrants and refugees, rates of depression were significantly higher among individuals who experienced pre-migration trauma as well as internment in a refugee camp.\textsuperscript{11}

Other factors, like witnessing death in a family and lacking resources such as water, shelter, and food, were associated with depression. Individuals who experienced more traumatic events were more vulnerable to depression, as trauma can lead to hopelessness and a loss of interest in activities. In North Korea, war and organized violence are not the primary reason for individuals to seek asylum, rather they often are trying to escape political oppression.\textsuperscript{12} Nevertheless, the traumatic experiences, such as torture, violence, imprisonment, and witnessing death, are shared.

In a group of North Korean refugees, insomnia, often associated with depressive and post-traumatic stress symptoms, was higher in those individuals who had experienced traumatic events prior to migration.\textsuperscript{12} These findings suggested that development of refugee insomnia could be associated with these traumatic experiences. A study of Syrian refugees in Turkey found that other factors could contribute to the development of PTSD, like being diagnosed with a psychiatric disorder in the past or having a family history of psychiatric disorder, along with experiencing trauma. Refugees face major obstacles to meet health care needs, along with trauma and prior diagnoses, while in war zones or areas affected by natural disasters.\textsuperscript{13}

**During Migration**

During migration, there are other stressors that can be associated with depression and anxiety. Stress can be from an uncertainty in the future, as is typical of asylum seekers. In two Danish asylum centers, the mental health of rejected Iraqi asylum seekers was evaluated. In this group, the prevalence of anxiety symptoms was 94% and depression symptoms had a prevalence of 100%. The lengths of stay in the asylum centers, as well as the number of traumatic events, were thought to be risk factors associated with psychological distress.\textsuperscript{14}

Among those in refugee camps, daily stressors can exacerbate mental problems, such as lacking basic necessities, restricted movement, and continued concern for safety, as refugee camps are only short-term solutions.\textsuperscript{15} Consistency in the life of refugees can ease mental distress. For example, the prevalence of PTSD was lower than expected in a group of Syrian child refugees, perhaps because these children travelled with at least one parent, transferring a crucial part of the child’s psychosocial environment. Therefore, having a parent accompany children during travel in the migration process could be a protective factor that can reduce post-traumatic stress rates among some children. Additionally, a successful flight during migration was associated with creating feelings of hope for the future.\textsuperscript{16} However, the anxiety of the parent accompanying the child can also influence the child’s own anxiety, therefore, the presence of a parent may not always be favorable, especially if parents have mental distress.\textsuperscript{17}
Post-Migration

Often, depression among refugees has long-term effects. A study of Guatemalan refugees in Mexico found a 38.8% lifetime prevalence of depression.\(^1\) Karenina refugees settled on the Thai-Burma border had a lifetime depression prevalence of 41.8%. Post-migration stress can be related to feelings of insecurity. A group of North Korean refugees settled in South Korea felt unsafe due to a fear of being arrested and deported back to North Korea.\(^2\) Post-migration mental distress has also been associated with acculturative stress. Refugees were about ten times more likely to have PTSD than the host country’s general population, illustrating that the mental distress in refugee populations does not disappear after resettlement.\(^1\) These PTSD rates were among 7,000 refugees resettled in western countries. The comparison of refugees with the general population may not be reflective of a whole picture, with need to consider refugee rates with other populations including veterans and/or domestic violence victims in future studies.\(^18\)

Acculturation is the process of integrating oneself into a new culture while maintaining one’s origin culture and identity. This process can create a considerable amount of stress for new refugees trying to restart lives in new countries, often resulting in anxiety and depression, as well as the exacerbation of post-traumatic stress. Acculturative stress is based on the demands of immigration experience. It is related to experiences that cause stress among immigrants and refugees. These include unfamiliarity with daily tasks, difficulties in finding employment, learning the host country’s language, discrimination, and a feeling of not belonging in one’s new environment. As an example of overcoming language barriers and its effect on mental health, Bosnian refugees living in Australia reported significantly more stress in terms of accommodating to the host language than Bosnian refugees living in Austria. Acculturative stress affects mental health based on the social atmosphere a refugee experiences in a host country, indicated by immigration policies and the general attitude of the host society towards refugees and different cultures.

Discussion and Conclusion

Refugee populations have increased vulnerability to post-traumatic stress disorder, depression, and anxiety due to their exposure to traumatic experiences prior to migration.\(^12\)\(^,\)\(^13\) Prior to the migration process, refugees often experience trauma from organized violence and political oppression, which can include the death of a loved one, torture, imprisonment, witnessing public executions, and lacking basic necessities.\(^12\)\(^,\)\(^13\) Other risk factors prior to migration include previous diagnoses of psychiatric disorders in oneself and/or family members.\(^14\) The development of such disorders can happen regardless of age; however, some age groups may experience more intense symptoms than others. Children in particular can develop behavioral and emotional problems as a result of certain traumatic experiences they may face, including forced migration.\(^8\) However, adolescents were more likely to have PTSD, which could be related to risk factors such as having more siblings or older parents, among others.\(^7\)

Studies varied in regards to showing differences in the manifestation of mental distress between males and females. Nasiroglu et al.\(^7\), however, showed being female as a risk factor for depression. Females were more likely than males to have an established mental health diagnosis. In relation to PTSD, women were more likely to exhibit PTSD symptoms; however, this has not been consistent when the prevalence of PTSD was investigated among some Syrian Kurdish refugees, possibly due to culture differences.\(^9\) Nevertheless, being female generally was associated with increased prevalence of mental distress.

During the migration period, there were several factors that contributed to mental distress, such as lingering feelings of unsafety and uncertainty in the future. Prevalence rates of depression and anxiety among refugee populations who were denied asylum were high.\(^14\)\(^,\)\(^15\) A protective factor that helps when migrating with children is maintaining some aspects of a refugee’s previous environment, such as ensuring the child travels with at least one parent.\(^16\)

Post-migration can include many difficulties that can cause mental distress to be worsened and/or have a long-term presence of mental health symptoms. A common factor associated with mental distress post-migration is acculturative stress, often experienced by refugees and immigrants.\(^6\) Experiences that result in acculturative stress include unfamiliarity with daily tasks, overcoming language barriers, and facing discrimination, among others. Acculturative stress often is unique to one’s environment because of the attitudes of the host country and whether certain changes in environment, such as language, are great. Not only are refugee populations vulnerable to PTSD, but they also face secondary features with their PTSD, increasing the burden of the mental disorder. These features can include hallucinations and delusions. Refugees are uniquely vulnerable to these secondary features because of their more long-term trauma. They are thrust into unfamiliar environments and lack familiar support systems. Consequently, refugees with PTSD are likely to experience secondary psychotic symptoms as well.\(^19\)

This review article highlighted the higher prevalence rates of mental health disorders among refugees, especially depression, anxiety, and PTSD, who have experienced trauma and forced migration from their regions/countries. It underscored the importance of managing mental health scars and disorders with great empathy and higher level of care. Possible scenarios to help include, but are not limited to, involving family members in their care, language interpreters, being patient with them, and establishing an inclusive environment that accounts for the psycho-socio-cultural aspects of refugee lives.\(^8\)\(^,\)\(^18\) Comparing refugees with the general population may not be reflective of whole picture. Therefore, further need exists to compare refugee mental health rates with other populations including veterans, domestic violence victims, and/or other violence victims in future studies.

There are few studies available on mental health issues in the refugee population, possibly due to a lack of funding in this clinical arena. Moreover, few studies have mentioned potential errors in reporting data due to inability of the refugees to report their
symptoms accurately under moderate to severe mental distress. More studies are needed to examine the increased vulnerability of refugee populations to mental health disorder and management guidelines to integrate them better and more fully into a new host society.

REFERENCES
1 Bogic M, Njoku A, Priebe S. Long-term mental health of war-refugees: A systematic literature review. BMC Int Health Hum Rights 2015; 15:29. PMID: 26510473.
2 Vonnahme LA, Lankau EW, Ao T, Shetty S, Cardozo BL. Factors associated with symptoms of depression among Bhutanese Refugees in the United States. J Immigr Minor Health 2015; 17(6):1705-1714. PMID: 25348425.
3 Nygaard M, Sonne C, Carlsson J. Secondary psychotic features in refugees diagnosed with post-traumatic stress disorder: A retrospective cohort study. BMC Psychiatry 2017; 17(1):5. PMID: 28056884.
4 American Psychiatric Association, Diagnostic and Statistical Manual of Mental Disorders: DSM-IV-TR. 4th ed. Washington, DC: American Psychiatric Association, 2000.
5 Georgiadou E, Morawa E, Erim Y. High manifestations of mental distress in Arabic asylum seekers accommodated in collective centers for refugees in Germany. Int J Environ Res Public Health 2017; 14(6):1-6. PMID: 28590438.
6 Kartal D, Kiropoulos L. Effects of acculturative stress on PTSD, depressive, and anxiety symptoms among refugees resettled in Australia and Austria. Eur J Psychotraumatol 2016; 7:28711. PMID: 26886488.
7 Nasiroglu S, Ceri V. Posttraumatic stress and depression in Yazidi refugees. Neuropsychiatr Dis Treat 2016; 12:2941-2948. PMID: 27881919.
8 Ceri V, Ozlu-Erdik Z, Ozer U, Yalcin M, Popow C, Akkaya-Kalayci T. Psychiatric symptoms and disorders among Yazidi children and adolescents immediately after forced migration following ISIS attacks. Neuropsychiatr 2016; 30(3):145-150. PMID: 27628299.
9 Ibrahim H, Hassan CQ. Post-traumatic stress disorder symptoms resulting from torture and other traumatic events among Syrian Kurdish refugees in Kurdistan Region, Iraq. Front Psychol 2017; 8:241. PMID: 28265252.
10 Schock K, Bottche M, Wenk-Ansohn M, Knaevelsrud C. Impact of new traumatic or stressful life events on pre-existing PTSD in traumatized refugees: Results of a longitudinal study. Eur J Psychotraumatol 2016; 7:32106. PMID: 27834172.

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