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One Physician’s Account at a Time
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Cover photo (by J.P. Bell, MD): Dr. Taggart interviews Joe Stallings, MD, as part of a project that will tell the story of medicine in Arkansas. More on page 270.

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Winner of the ASAE Excellence in Communications Award

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The Story of Medicine in Arkansas

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Join us to stay updated on health care news in Arkansas.

Health care disparity and the pursuit of happiness

As we walk away from the re-approval of the Arkansas Medicaid expansion under its new name “Arkansas Works,” the mission does not feel accomplished and much still needs to be done. In just a few months, we will start this journey all over again. Many questions remain with the most pressing among them being “is health care a right or a privilege?” Depending on the answer to this question, we may consider the expansion either a fair step for our citizens who need it the most or another concession to a bunch of “lazy free-riders,” like Nancy (?).

Nancy was a lively 52-year-old nurse who worked day and night to raise her two children by herself after a bitter divorce. When I saw her for the first time, one of her children had just finished college and the other was finishing high school. The diagnosis of triple negative breast cancer was not on the map, but she told me with determination that she would beat it. As the cancer was locally advanced we started with chemotherapy first. The response was at best modest. We changed to another chemotherapy and were able to achieve some local control and send her to surgery. The rocky course of chemotherapy and surgery forced her to use all her free-riders,” like Nancy (?).

The ACA is a step forward, albeit imperfect, toward the right to universal health care. Do we really need to present all the arguments that support the idea that health care is a right? How can we conceive the right to “life, liberty and the pursuit of happiness” without the right to affordable health care? Can anybody really be happy or free without being healthy? Once we have agreed that health care is a right and a necessity, balancing the budget becomes a matter of finding items that are not vital and eliminating them.

It is estimated that the ACA will cost up to 1.2 trillion dollars by 2025. However, it is also estimated that Medicare and Medicaid, and the health system as a whole, will lose more than 2.7 trillion dollars for the same period in fraud. If even half of this money is recovered, we would more than break even. Cheaters are individuals and institutions that abuse the system in this fee-for-service environment. Rewarding quality not quantity and encouraging preventative care in parallel with the implementation of the ACA should help generate additional savings. If Congress were willing to tackle the price of prescription and generic drugs, significant savings would result from stabilizing generic drug prices and allowing public entities to negotiate drug prices with manufacturers. Political courage and good policies are needed, not further restriction of access to health care.

Health care is a right; it is a matter of dignity and decency too. Millions of our citizens would rather die than beg for help, out of dignity. But it is our obligation to reach out to the weakest and poorest in our society, out of decency. If we allow this disparity to continue we would be betraying the very principles on which our society is founded and sowing the seeds of its destruction.

Would Nancy be alive now to celebrate the graduation of her son that had the declaration of independence said “the preservation of healthy life, liberty and the pursuit of happiness?”


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The Story of Medicine in Arkansas
One Physician’s Account at a Time

Sam Taggart, MD, retired family practice physician, wants to discover and share the “story of medicine here in this place we call Arkansas.” He is doing just that – one physician interview at a time.

He phrases it that way, “here in this place we call Arkansas,” because he knows the state’s history. A historian, particularly of health and disease, Dr. Taggart knows that “this place” that we live in pre-dates its name by centuries. “The first documentation of humans living and being buried here on this land is 8500 BCE,” shared Dr. Taggart, giving credit for this and other interesting facts to the Arkansas Geological Survey.

An archeologist in 1974, Dan Morris, got a call from what came to be the Sloane Site. They had uncovered the first documented cemetery in the western hemisphere, here on the banks of the Cache River in Northeast Arkansas, just west of Jonesboro.”

Like the story of this land, the practice of Arkansas medicine goes back centuries too, and is full of history, human interest, and stories worth telling. For a start, Taggart’s own story includes growing up on a small farm in the Delta, working through college at Jonesboro, and attending medical school at UAMS. After that, he served in the U.S. Army before embarking on 37 years of medical practice – mostly in Benton, where he founded Family Practice Associates, retiring as senior partner in 2013.

From just this small summation, we pick up hints of a much bigger story, rife with human connections and habits, history, geography, and more. Digging deeper, we find out that Dr. Taggart is a published author of fiction and nonfiction, has a love for photography that captures the past, and was among the first physicians in Benton to practice without a suit and tie – a sure sign of the times. Still other facets of his “bigger” picture include lessons learned from his father, a sharecropper whose family worked the rice lands for the Conner Company east of Augusta, near the White River. In the middle of a teenage crisis, young Taggart was told by his father, “Son, the deepest, darkest secret you have is the one you share in common with the most people.”

“There has never been a day in my life since that I have not thought of that moment. It has formed my practice of medicine and my writing,” recalled Dr. Taggart, adding to his story the value of his mother’s example in his life, too. “My mother’s lot in life seemed to be caring for the elderly. She passed the time reading, and one of the highlights of my youth was getting my first library card. She passed her love of reading to her children and the caregiving element was probably why I choose medicine.”

Such moments as these have given weight and value to others, Dr. Taggart knows. Indeed, he has shared many detailed human-interest stories as a regular contributor to local publications including the UAMS Historical Research Center’s newsletter and the Arkansas Academy of Family Practice Journal. For The Public’s Health (2012), Taggart was commissioned by the Arkansas Times and the Arkansas State Health Department to write a nonfiction account of the evolution of public health in Arkansas.

“In doing research for The Public’s Health [and other projects], I realized that there were few good pieces on health and disease in Arkansas,” said Dr. Taggart, “and that already I had a good start toward such a work.”

After all, he had conducted recorded interviews of physicians (particularly in Saline County) and gathered other research – all unpublished to date – from the Arkansas Chapter, American Academy of Family Physicians; the Arkansas Archeological Survey; and other sources.

To tell more of the (human) story of medicine in Arkansas, Dr. Taggart has embarked on a new writing project, this time about the evolution of rural health and health care over the last two centuries. “Right now, our working title is Country Doctors: the Ever-Changing Face of Rural Health in Arkansas,” said Dr. Taggart, who is working on the project with his colleague and friend since medical school, J.P. Bell, MD.

A retired ER physician residing in Fayetteville, Dr. Bell ran across Dr. Taggart’s book and after reading it, called him up. “We renewed our friendship and discovered we both had the same love of telling stories about people through words and pictures,” explained Dr. Bell, who had been contributing photography and writing for books and magazine articles for more than three decades. “Sam invited me to do the photography and videography for a project documenting the face of rural medicine in Arkansas. I jumped at the chance to photograph mentors and colleagues in medicine, old and young, across the state.

“I realized some 20 years ago that there were physicians walking the corridors in my hospital who had practiced medicine during the era when penicillin was the new wonder drug,” said Dr. Bell. “Practicing medicine alongside these aging physicians were young doctors just out of training who had never known the days when a stat CT was not available for a head trauma or other problems. Medicine has come far in these few short years. I hope that this project illuminates the particular challenges facing the physicians in rural Arkansas who chose to serve the communities and towns outside of the major population centers of our state.”

Carla Coleman, executive vice president of the Arkansas Chapter, American Academy of Family Physicians (ARAFP), expressed excitement to see the project taking shape. “Doctors Taggart and Bell are a perfect team,” says Coleman. “They are enthusiastic about the project, and the enthusiasm is contagious! To pursue such an endeavor is more than just a curiosity about the past. It is [their] mission to [learn] as much as possible about those who provided health care across our state.”

The project will rely on multiple sources for its information, including AARFP; however, its main source will be the personal stories of the doctors who have lived the practice of medicine in Arkansas day-to-day. To learn more about the project, The Journal interviewed the project’s main interviewer, Dr. Sam Taggart.

What can participants expect from the overall scope of your project now and long term?

We decided last fall to reach out to older physi-
cians who had retired, to record some of the first-hand knowledge that we’re going to lose if we don’t capture it now. At first, we tried to focus on retired docs in those little towns, but as we’ve gone along, we’ve realized we also need to interview doctors that are not retired, some younger, etc.

Within the next year, we plan to interview a minimum of 25 physicians (likely more) from small towns around the state – with video – with the idea of getting as accurate a picture as we can about what it has been like to practice medicine in Arkansas. With AMS’ help, we started with a list of 500 doctors, and narrowed that to 240 physicians who practiced in small towns all over the state.

You have begun interviews, is that right? How is it going and what do you plan to do once you have concluded the interviews?

We have begun interviews, and I cannot tell you how much fun this is. We hope to complete them in early 2017. After interviews are complete and edited, we will give each interviewee a DVD and a hard copy of the interview.

We will also give copies to ARAFP, the Historical Research Center at UAMS, the Pryor Center for Oral History in Fayetteville, and The Country Doctor Museum in Prairie Grove. We hope our interviews will be of help to a number of local historical societies, museums, and libraries from around the state.

Phase two of this project will be to publish a photo-essay book using the interviews and the photography as the backbone.

Beyond that, we hope to create an hour-long video detailing the evolution of rural health over the last two hundred years. Right now, we are working from our own dollars, but we have begun to explore options for funding.

What are some of the questions participating doctors can expect to answer?

I have about a 75-100 question interview that I send in advance. It includes:

Where were you born (town, county)? What hospital? Do you know the name of the physician who delivered you? Where were you raised? What type work did your mother, father, and extended family do?

The questions build from there and delve into all kinds of things that our interviewees are willing to dis-

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cuss. We’ve been very touched, as have they, by what they have shared. Final questions include things like Have you enjoyed your life? What you have chosen to do? and Would you do it again?"

What is your take away from the interviews you have conducted so far? Have you – and your interviewees – felt rewarded for the effort?

In one of our earlier interviews, we talked to a physician about his experiences with a patient — an older man who was dying. Near the end of that story, I looked up and the four of us (JP, the doctor, his wife, and me) were all crying. It is very touching.

The very last question I ask in these interviews is basically, Pretend the camera is not here, but your great, great, great grandchildren are sitting out here. What do you want to say to them? How do you want to finish this interview today? In all four cases, so far, they’ve hesitated, but they’ve answered. Essentially, their answers amount to, “I did my best.”

What are the larger issues in health care that will be touched on, even if inadvertently, through these interviews?

As you likely know, 40 counties in Arkansas are losing at least 10% of their population each year.

Through these interviews, physicians will touch on such things as this — and other things affecting the state now or that have affected their practices. For example, we’ll be interviewing Steve Collier from Augusta, who went back to practice in the late 1970s, looked around and realized needs were not being met. He created the White River Rural Health Cooperative, now called ARcare. From one small clinic in the town of Augusta, it has grown to include facilities in 26 counties in Arkansas (and 9 or 10 in Kentucky). Now in his 60s, Collier still runs the cooperative, which is using more telemedicine, sending radiologists and nurse practitioners out in the community to serve residents, and more. In that one interview alone, we’ll touch on rural medicine, how population change affects the future of medicine, and the corporatization of medicine.

We’ll interview Joe Stallings, MD, who has had a major impact on rural health in the Jonesboro area. Frank Thibault, MD, is another subject, a retired OB/GYN and a third-generation physician, so we’ll learn about how medicine came to be passed down in this family.

Through the project, we hope to look at the history of minority physicians in Arkansas and the difficulties they may have faced in serving patients; the evolution of hospital care (from little tiny doctors’ hospitals in small towns to large, regional centers); and more.

In what areas of the state do you still need to hear from physicians?

Gaps in our coverage include south central east of the I-30 corridor; the I-40 corridor east of Little Rock (Brinkley, Forrest City, Wynn); and the I-40 corridor west of Little Rock (Morrilton, Clarksville, Ozark). We would like to hear from physicians in these areas — particularly good storytellers.

As for this project’s future influence, (both on participants and those of us who will simply enjoy the finished product), ARAFP’s Coleman summed it up well. “Dr. Taggart’s work on this project will provide a background into where we have been, where we are, and in a lot of cases, where we are going,” she said. “To have a written history of rural medicine in Arkansas for the future is priceless to future health professionals.”

If you’d like to be part of such a history lesson for generations to come, or if you have someone in mind, please call Dr. Taggart (501-773-7830) or Dr. Bell (479-650-2328) or email samtaggart@att.net.

*Taggart’s acclaimed fictional novel We All Hear Voices (date published), garnered 3rd place for popular fiction by Independent Book Publishers (2010).
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SOFT-TISSUE SARCOMAS (STS) ARE A GROUP OF RARE MALIGNANT TUMORS THAT ARE BELIEVED TO ARISE FROM MESENCHYMAL TISSUE (fat, muscle, connective tissues, nerve, vessels and others of uncertain etiologies) with the majority of these tumors developing in the extremities.1 Overall incidence for these tumors is less than 10,000 cases annually in the United States making them one of the most uncommon causes of cancer. The annual incidence in Arkansas is only 20 – 25 cases per year. Benign soft tissue masses are far more prevalent (estimated by at least 100 fold). Perhaps because a STS often presents as a painless and slowly growing mass, it can be difficult for the physician to distinguish clinically between these two very different lesions.1 Although soft tissue sarcomas vary in size and symptomatology, red flags at the time of patient presentation include large size of the mass (>5cm in diameter), a location deep to the fascia (or inside the muscle), firm and immobile character, and rapid growth. Pain is often a late finding and may be caused by local soft tissue damage, nerve compression, or tumor necrosis. Proper identification and a high degree of suspicion are needed in order to correctly identify these rare malignant lesions so that appropriate therapy can be initiated, and potentially avoidable adverse situations can be avoided.

In order to avoid misdiagnosis of a malignant lesion or uninformative resection of a malignant process, an appropriate evaluation is required in the management of all soft-tissue masses. Additional testing is indicated and simple excision should not be the first treatment of choice when faced with the need to diagnose a rapidly enlarging tumor, masses greater than 5cm in size, or masses deep to the fascia (often fixed or immobile). Over the past 20 years, MRI scanning (with contrast) has become the gold standard for the evaluation of soft-tissue masses. The MRI characteristics of a soft-tissue sarcoma include a heterogeneous mass typically deep to the fascia and >5 cm in size.

After appropriate imaging has been obtained, suspicious masses with worrisome or heterogeneous signal characteristics should be biopsied prior to complete removal. An “unplanned excision” of a STS can lead to the incomplete resection of a malignant mass often because it was initially felt to be benign without appropriate attention to preoperative imaging, antecedent biopsy, or operative margins.2 The literature has shown that patients referred to centers with experience and a comprehensive team approach for STS have better oncological results and fewer complications than those patients biopsied prior to referral.2 One reason for this difference in outcome is that many asymptomatic patients who presented to their local physician were thought to have a benign lesion (such as a lipoma) and many underwent simple excision of the mass or had an incorrectly placed excision biopsy site. The technique for performing an appropriate biopsy is also important. Improperly placed and performed biopsies can increase future surgically-related morbidity or potentially compromise limb-salvage options. In many cases, unplanned excisions and improperly placed biopsies increase the need for complex soft-tissue reconstruction procedures (free or rotational flaps) and skin-grafting.

In general, soft tissue sarcomas, although they arise from many different tissue types can be roughly divided into low and high grade lesions. In the end “Tissue is the issue.” Being aware of the correct histological diagnosis of a mass prior to surgical removal allows the surgeon to determine the proper extent of surgical excision, timing for additional adjuvants such as radiation, and needed additional staging studies to help direct future care. Complete surgical excision is the only treatment necessary in the management of most low-grade soft tissue sarcomas. With such procedures, known as wide surgical excision, there is an attempt to remove the tumor with a surrounding cuff of normal tissue to decrease the chance of local recurrence. Even in low grade lesions, residual tumor significantly increases the chance of local recurrence which may not respond to radiotherapy. High grade lesions, which are often larger, may require more radical resections, but the concept is similar—that of wide resection and complete removal of the entire tumor with an adequate margin of normal surrounding tissue. Additional treatment with radiation therapy has been shown to reduce the chance for local recurrence of the high-grade lesions and is generally recommended. Chemotherapy is not often a frontline treatment, but can be used selectively or in cases of metastasis.1 In STS, overall prognosis is best predicted in relation to the grade of the tumor with little correlation to local recurrence. Low grade sarcomas have a generally reported 5 years survival of 90% while high grade tumors 5 year survival statistics range from 50-70% with pulmonary metastasis being both the most common site for disease spread and cause of patient mortality.1 Local recurrence can significantly affect the patient’s long term function and is a cause for significant morbidity.

Figure 1. MRI example of an anterior thigh soft-tissue sarcoma. The mass is >5cm in size with heterogeneous signal characteristics. Masses with these findings are highly suggestive of a soft-tissue sarcoma.
Although malignancies of mesenchymal tissue are rare, treatment of patients with STS can be complex and comprehensive evaluation and care is needed to assure that each of the patients has the best possible chance for a good outcome. In general, many of the benign and malignant lesions present as non-painful masses. In contrast to rare STS, the majority of soft tissue masses are benign and their clinical presentation is typified by a superficial, small, soft and non-fixed appearance. When there are concerns that a soft tissue mass does not fit the usual pattern, an MRI is the appropriate next step. Clearly not every superficial, soft and mobile soft tissue mass needs an MRI for evaluation and many wrist ganglia and extremity lipomas can be safely identified and treated without such expensive imaging. However, suspicion of an atypical presentation and a knowledge of how these malignant soft tissue sarcomas behave should help prevent misdiagnosis and incomplete treatment.

REFERENCES
Beneficiary Surveys Track Medicaid Improvements

The Arkansas Medicaid program constantly strives to improve the quality of services to Medicaid beneficiaries. Surveys show that Medicaid beneficiaries continue to see improvement in several key indicators of health care quality and cost effectiveness.

To evaluate how well Medicaid works for its beneficiaries, what services are used, and how beneficiaries evaluate the program in general, the Arkansas Department of Human Services, Division of Medical Services, contracts with AFMC to conduct surveys to learn more about beneficiaries’ satisfaction with their doctors and recent care received, and experiences with the Medicaid program and its customer service. AFMC uses the Consumer Assessment of Healthcare Providers and Systems (CAHPS®) 5.0H surveys as a baseline to measure beneficiaries’ satisfaction with Medicaid and assess how beneficiaries perceive their own health.

CAHPS® surveys are publicly available and are widely used by quality monitoring organizations, regulators, community collaboratives, provider organizations and health plans to improve health service quality and develop public policies. The CAHPS® 5.0H surveys ask consumers about their experiences with health care available in four versions that can be administered to adults or a child’s caregiver (Medicaid and commercially insured). Questionnaires, fielding protocols, analysis and reporting are all standardized to ensure comparability. They may be administered across a range of heterogeneous populations including adults and children with chronic conditions.

AFMC’s analytics department manages all aspects of survey design, population design, sampling, data collection, analysis and reporting. In addition to the ratings questions, composites, health scales and question frequencies from the CAHPS® questions, program-specific supplemental questions are often incorporated to measure satisfaction. AFMC also submits data to national data repositories such as the National CAHPS® Benchmarking Database. This enables regional and national comparisons and highlights any significant differences.

MEDICAID PROGRAMS IN ARKANSAS

ConnectCare manages Medicaid care and helps patients find providers. ARKids First A is Medicaid’s program for children. Together, these programs provided health care services to more than 390,000 Arkansas adults and children in state fiscal year (SFY) 2015. Additional benefits are available to Medicaid beneficiaries through ARKids First B, one of the most successful and innovative children’s health care programs in the United States. In SFY 2015, ARKids First B provided for the health care needs of approximately 48,000 Arkansas children who were not eligible for ARKids First A.

Another source of help for Arkansas children includes the Tax Equity and Fiscal Responsibility Act of 1982 (TEFRA). This program allows Arkansas to open its Medicaid program to children with physical or mental disabilities who would not ordinarily be eligible because of their parents’ income or resources. It also permits disabled children to be cared for at home rather than in an institution. As of 2010, Arkansas is one of only 18 states offering TEFRA benefits. More than 3,000 Arkansas families are currently enrolled.
More information about these programs is at: www.medicaid.state.ar.us/General/General.aspx

MEDICAID BENEFICIARIES’ SATISFACTION SURVEYS

The CAHPS® 5.0H Medicaid adult and child surveys include five composite measures, four rating questions, two question summary rates and five effectiveness-of-care measures. AFMC has conducted the Medicaid beneficiary satisfaction surveys since 2007, making it possible to trend available data. Figure 1 shows the trend of three CAHPS® measures for adults and children. The measures represent the percentage of beneficiaries who responded favorably to:

- **Getting needed care**: Measures beneficiaries’ ease of seeing a specialist and getting any care, tests or treatment
- **Getting care quickly**: Measures beneficiaries’ access to urgent and non-urgent care in a timely manner
- **Health promotion and education**: Measures how often beneficiaries and doctors talk about specific preventive measures to improve health
- The ratings for personal doctor, specialists, health care and health plans show the percentage of beneficiaries who responded with an 8, 9 or 10 on questions rated from 0 (worst possible) to 10 (best possible).

The adult survey also captures effectiveness-of-care measures by inquiring about:

- **Aspirin use**: Did beneficiaries discuss with a doctor or other provider the risks and benefits of using aspirin
- **Smoking cessation**: Beneficiaries age 18 and older were asked if they are current smokers or tobacco users and, if yes, whether they received advice to quit
- **Smoking cessation medications/strategies**: Beneficiaries age 18 and older who are current smokers or tobacco users were asked if they had discussed or had been recommended cessation medications or discussed cessation strategies

Complete summaries of previous years’ results are available at mmcs.afmc.org/HealthCareProfessionals/SurveysReporting.aspx

AFMC is NCQA Certified

The National Committee for Quality Assurance (NCQA) is a private non-profit organization dedicated to improving health care quality. The NCQA’s Survey Vendor Certification team trains, certifies and provides quality oversight to survey vendors.

Since 2007, AFMC has been one of only 16 NCQA-certified HEDIS survey vendors in the nation and the only one based in Arkansas.3 To become a NCQA-certified CAHPS 5.0H survey vendor, an organization must demonstrate that it has the capabilities, experience and expert personnel to accurately collect and report survey results.

**REFERENCES**


Ms. Pullman is manager of survey research and Ms. Joshi is a statistician, both in the AFMC’s analytics department.
CASE REPORT

A 30-year-old woman with past history of type 1 diabetes, hypertension and chronic kidney disease (baseline creatinine of 2.0 – 2.5 mg/dL) presented to the emergency department (ED) with complaints of nausea, vomiting and abdominal pain of one day duration. She was treated symptomatically with antiemetics and was discharged home after she tolerated emetics and was discharged home after she tolerated oral challenge. The following day, the patient returned to the ED with persistent nausea and vomiting. She had a recent hospitalization for osteomyelitis of her right foot and was status post partial amputation of her right 4th and 5th toe. Also, she was on intravenous (IV) Vancomycin and Metronidazole for her osteomyelitis which was to be continued for a total of six weeks. Her family history was non-significant. She did not use any tobacco, alcohol or illicit drugs. Her medications included insulin 70/30, amiodipine, sertraline, metoprolol, and promethazine as needed for nausea and hydrocodone as needed for pain, along with her IV antibiotics. At the time of her second presentation, she was febrile to 102°F; pulse was 71 beats/min, respiratory rate was 17 breaths/min, and blood pressure of 131/70 mm Hg. She was hypoxic with a pulse oximetry reading fluctuating between 50% and 60%. Her initial blood gases were consistent with hypoxia (pH 7.38, pCO2 33 mm Hg, pO2 52 mm Hg, HCO3 20 mEq/L on 4 Liter oxygen) and she was placed on oxygen via non-breather. Bibasilar crackles were heard on chest examination and chest X-ray showed possible interstitial infiltrates, but no obvious lobar pneumonia was seen. She had a wound vac on her right foot, and the rest of her exam was unremarkable. In the emergency department, she received piperacillin-tazobactam and one dose of therapeutic enoxaparin for possible pulmonary embolism.

The patient was admitted to the Intensive Care Unit (ICU) for hypoxic respiratory failure. A non-contrast computed tomography (CT) scan of the chest showed bilateral scattered opacities consistent with pneumonia and also trace bilateral pleural effusions. Her nasopharyngeal swab was positive for influenza A. The patient was started on oseltamivir, piperacillin-tazobactam (for hospital acquired pneumonia) and also doxycycline for atypical coverage. Her vancomycin was continued, but the metronidazole was stopped as piperacillin-tazobactam provided coverage for anaerobic organisms. The patient required intubation and was placed on mechanical ventilation for 4 days. Her blood, urine, respiratory, and broncho-alveolar lavage (BAL) cultures all returned negative, including legionella antigen. A ventilation-perfusion scan showed low probability for pulmonary embolism; hence therapeutic heparin was changed to prophylactic dose for deep venous thrombosis (DVT) prevention. The patient also developed septic shock but did not require any vasopressor support. She improved clinically on day five of hospitalization, was successfully extubated to 2 liters oxygen via nasal cannula and was transferred back to the floor.

After two days of observation on the floor, the patient developed multi-organ dysfunction including worsening renal failure, liver failure and leukocytosis. On day seven of hospitalization, piperacillin-tazobactam was discontinued based on the infectious disease team’s recommendations. The patient continued to receive renally dosed vancomycin and metronidazole for her osteomyelitis.
On day eight of hospitalization, she was transferred to ICU for new-onset multi-organ failure. After two days, she was hemodynamically stable and transferred back to the floor.

Her creatinine gradually worsened to 5.4 mg/dL (Figure 1), and she was anuric for over 24 hours requiring hemodialysis. Her white blood cell count increased to 39.04 x 10^3 cells/mm^3 (day 14) gradually from 8.23 x 10^3 cells/mm^3 on admission (Figure 2), and she continued to develop fever almost daily up to 104°F associated with a rash on her arm. Liver function tests increased to bilirubin 2.3 mg/dL, AST 268 IU/L, ALT 180 IU/L, GGT 128 IU/L, alkaline phosphatase of 708 IU/L, lactate dehydrogenase 1356 IU/L (day 14), which were 0.8 mg/dl, 41 IU/L, 26 IU/L, 27 IU/L, 102 IU/L and 313 IU/L respectively on admission. INR increased steadily to 2.5 on day 15 from 1.0 on admission (Figure 3). Her blood cultures remained negative and all other work up for sepsis remained negative. She had elevated absolute eosinophil count, and peripheral smear showed leukocytosis with eosinophilia, along with plasma cell proliferation that were about 10% of total nucleated cells consistent with a leukemoid reaction. She remained anuric requiring scheduled dialysis. Renal biopsy demonstrated acute interstitial nephritis in a background of chronic interstitial nephritis. Since the development of rash and eosinophilia was suggestive of an allergic reaction, infectious disease specialists diagnosed her to have a piperacillin-tazobactam induced leukemoid reaction. The time course of events also suggested piperacillin-tazobactam to be the cause of the acute interstitial nephritis. On day 17 of her hospitalization, she was started on methylprednisolone intravenous at 125 mg daily for initial three days and then switched to oral prednisone 60 mg daily. Following this, her fever, rash and leukocytosis improved. Her liver function recovered and her kidney function started to normalize gradually. She started to produce urine and after a week of observation, the renal team deferred any further dialysis.
Acute Interstitial Nephritis (AIN):

AIN is a common cause of acute kidney dysfunction due to infiltration of the renal interstitium with inflammatory cells. Drugs account for about two-thirds of AIN cases. Other causes can be infection, renal disease associated with other systemic syndromes, idiopathic AIN, beta lactam antibiotics, non-steroidal anti-inflammatory drugs (NSAIDs), antiretrovirals, anticonvulsants, proton pump inhibitors and a few other agents. In drug-induced AIN, the onset of renal dysfunction may vary anywhere from a day to several months after the initial insult, with a mean of around 10 days varying with the offending agent. There can be other allergic symptoms associated with AIN such as fever, rash and arthralgias. Although rise in blood urea nitrogen (BUN) and creatinine (which may or may not be associated with oliguria) is seen commonly, other laboratory signs of leukocytosis with predominant eosinophilia, acid-base disturbances, elevation of inflammatory markers, abnormal liver function tests and elevated serum IgE levels may all occur as well.

Eosinophilia may not always be present and hence should not be used to exclude the diagnosis of AIN. Renal biopsy is the only definitive test for diagnosis of AIN, but non-availability of biopsy results should not delay the management. The mainstay of treatment is to stop the offending agent. The role of steroids in the treatment of AIN is controversial, but some studies suggest that early use of steroids within a couple of weeks may lead to complete recovery of baseline renal function. A recent case report described four cases of piperacillin-tazobactam induced AIN in children as well.

**Drug induced hepatitis:**

Drug-induced liver injury is a common cause of liver toxicity and can be either immune-mediated hypersensitivity or an idiosyncratic reaction. This can occur anywhere from a few hours up to a few weeks after initiation of the drug. Most cases of liver injury have been benign and reversible on discontinuation of the drug. Reported hepatic reactions with penicillins have been mainly cholestatic, although a mixed cholestatic and hepatocellular pattern may also occur. Symptoms of fatigue, jaundice, choloruria, and pruritis are common and are sometimes associated with rash and fever. Tender hepatomegaly may be found on examination. The mainstay of treatment is withdrawal of the drug, along with supportive measures to correct volume depletion, electrolyte imbalance and the use of antiemetics in case of vomiting. There have been reports of use of corticosteroids for treatment for amoxycillin/clavulanic acid induced liver injury.

In our case, early diagnosis, concurrent stopping of the offending agent (piperacillin-tazobactam), and treatment with steroids together led to complete recovery of our patient.

**REFERENCES**


**CONCLUSION**

Clinicians must be cognizant of piperacillin-tazobactam as a drug capable of causing hepatitis and interstitial nephritis with other systemic manifestations of hypersensitivity reactions. Since complete recovery is possible after discontinuing the drug, the process of making a definitive diagnosis should not delay the cessation of the drug. Even though the use of steroids is controversial, earlier consideration of initiating systemic steroids or even liver transplantation referral in hopes of avoiding progressive systemic response might be worthwhile especially in severe cases. Although piperacillin-tazobactam is considered to be relatively safe and serious adverse effects are rare, these reactions should always be kept in mind in a patient who presents with organ failure and hypersensitivity reaction.

**DISCUSSION**

The timeline of events in our patient including acute renal failure, hepatic failure along with fever, eosinophilia, and rash were suggestive of a drug-induced reaction. Out of all her medications, piperacillin-tazobactam was thought to be the most likely contributor, and her rapid improvement after discontinuation of the drug proved the causal relationship. Our diagnosis of AIN was based on renal biopsy, which is definitive and excluded other etiologies for her renal failure. According to our review of literature, AIN is rarely reported with piperacillin/tazobactam and its occurrence with liver injury and serum sickness together is even rarer.
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Acute Eosinophilic Pneumonia: Pyrethroid Exposure & Change In Smoking Habit!

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ABSTRACT

We report a case of Acute Eosinophilic Pneumonia (AEP) in a 29-year-old white woman with recent use of a ‘flea bomb’ (containing pyrethroids) at home while remaining indoors, about 48 hours prior to presentation, and recent change in smoking habit (restarted 2 weeks prior after quitting for 10 years). She presented with two days of worsening fever, shortness of breath, productive cough, chest tightness, sweating, and weakness. She has a past medical history of well-controlled Asthma (rare use of albuterol PRN). She had a history of smoking one pack of cigarettes per week which she quit about 10 years ago, and restarted a few weeks prior to presentation. She also used a ‘flea bomb’ (contains pyrethroids) at home, and remained indoors, 48 hours prior to presentation. Vitals on admission were T: 98, HR: 130, BP: 134/100, RR: 20-30, O2 sat: 91% on 50% Venturi Mask. On examination, she was found to be using accessory muscles of respiration, unable to complete sentences, with bilateral diffuse crackles on auscultation. She was intubated for hypoxic respiratory failure and ARDS. She required a PEEP of 20 and 100% FiO2 to maintain oxygenation. Bronchoalveolar lavage showed 36% Eosinophils. She was given IV steroids with dramatic clinical and radiological improvement. To the best of our knowledge, this is the second report associating AEP with pyrethroid exposure.

KEYWORDS:
Eosinophilic pneumonia, eosinophilic pneumonitis, smoking, pyrethroid, eosinophils, and steroids

INTRODUCTION

AEP was first described in 1989 in a patient presenting with acute respiratory failure, as a separate entity from CEP (Chronic Eosinophilic Pneumonia). Following this, there has been an increase in the number of cases reported worldwide. The underlying etiology/pathophysiology is currently unknown but has been most commonly associated with recent new onset, increased frequency, or resumption of smoking after a period of quitting. Other possible associations are cocaine, heroin, and environmental factors. Usual age of occurrence is 20-40 years and it occurs twice as often in men. AEP presents as an acute febrile illness of less than three weeks duration, with nonproductive cough, dyspnea, and pleuritic chest pain. Patients with AEP can present with acute hypoxemic respiratory failure and ARDS. Labs are significant for neutrophilic leukocytosis, and BAL fluid showing eosinophilia. Possible underlying mechanisms may involve IL-5 in BAL fluid that is produced by T helper cells, which is a potent chemotaxin for eosinophils. Steroids are the mainstay of treatment.

CASE REPORT

We describe a case of a 29-year-old white woman who presented with two days of worsening fever (102°-104°F), shortness of breath, productive cough, chest tightness, sweating, and weakness. She has a past medical history of well-controlled Asthma (rare use of albuterol PRN). She had a history of smoking one pack of cigarettes per week which she quit about 10 years ago, and restarted a few weeks prior to presentation. She also used a ‘flea bomb’ (contains pyrethroids) at home, and remained indoors, 48 hours prior to presentation. Vitals on admission were T: 98, HR: 130, BP: 134/100, RR: 20-30, O2 sat: 91% on 50% Venturi Mask. On examination, she was found to be using accessory muscles of respiration, unable to complete sentences, with bilateral diffuse crackles on auscultation. She was intubated for hypoxic respiratory failure and ARDS. She required a PEEP of 20 and 100% FiO2 to maintain oxygenation. Labs showed a WBC count of 25.61k, Neutrophils 91.5%. Rapid flu was negative. A bronchoscopy with Bronchoalveolar lavage was done which showed 36% eosinophils. Following BAL results, the patient was started on IV Methylprednisone 1g IV daily. This resulted in dramatic improvement, with both oxygen requirement, and radiographic improvement. The dose was decreased to 80mg IV Q6H after two days. She was extubated on day six, and transferred out of the Intensive Care Unit. She was discharged the following day. Treatment with oral prednisone was continued for four weeks, and then tapered (along with Bactrim for PCP prophylaxis).

DISCUSSION

AEP is a well-known entity but has unknown etiology and underlying pathophysiology. It has been associated with a recent change in smoking habits (Recent new onset, increased frequency, or resumption after a period of quitting). Other possible associations, which have been described, include cocaine, heroin, environmental factors, and medications (Amiodarone, Bleomycin, Captopril, gold salts, iodine, radiographic contrast media, L-tryptophan, methotrexate, nitrofurantoin, phenytoin).

Hypothesized underlying mechanisms involve the role of GM-CSF and IL-5 produced by Th-cells (a potent chemotaxin for eosinophils). Others include (BAL fluid): IL-1ra, soluble type II IL-1 receptor, VEGF, GM-CSF, IL-6.

Diagnostic criteria have been proposed that include an acute febrile illness of short duration, hypoxemic respiratory failure, diffuse pulmonary opacities on chest radiograph and BAL eosinophilia >25% or lung biopsy evidence of eosinophilic infiltrates. Chest X-ray findings have good specificity, and usually shows diffuse pulmonary opacities with small pleural effusions. Challenge tests are possible but not the standard of care. Motivation to abstain from the potential etiologies is recommended. As the patient in our case quit smoking and remains motivated to...
never restart, and also not to stay indoor when using flea bomb. Lung biopsy findings are usually non-specific with acute and organizing diffuse alveolar damage, marked numbers of interstitial and lesser numbers of alveolar eosinophils, type II pneumocyte hyperplasia, interstitial lymphocytes, organizing intra-alveolar fibrous exudate, perivascular and intra-mural inflammation without necrosis. Treatment is based on early initiation of steroids, but no trials have identified an ideal dose. The ideal dose is likely to depend on the severity of symptoms. AEP often shows rapid, dramatic response to steroids (within 12 to 48 hours).11,23

Figure 2: Day 1 of admission. Chest radiograph showing diffuse bilateral extensive airspace disease with patchy character.

Our patient used a ‘flea bomb’ about 48 hours prior to presentation, while remaining indoors. ‘Flea bombs’ contains pyrethroids. In Brazil in 2001, there was concern regarding the use of cypermethrin (pyrethroid) and diesel, possibly causing Eosinophilic Pneumonitis. We could find just one study that has been done to explore this possibility. There have been multiple case reports that suggest an association between AEP and a recent change in smoking habits.3,11 Symptoms have been reproduced in some of the patients in whom AEP was attributed to smoking or other environmental factors by provocation tests.5,26 To the best of our knowledge, there has been only one published article testing possible association of pyrethroid exposure and AEP. An experimental study in mice in Brazil in 2001 by Garcia et al concluded that diesel and cypermethrin induced lung hyper-responsiveness was associated with increased eosinophils in blood (p = 0.03) and lung tissue (p = 0.005). This study was pursued following cases of AEP in a population, following aerosolization with diesel and cypermethrin for mosquito control.

CONCLUSIONS
This case involved a young woman who was admitted with acute hypoxic respiratory failure, and treated with antibiotics for suspected pneumonia before key historical elements revealed the true nature of her illness. Acute eosinophilic pneumonia has been recognized for many years, but the pathophysiology is only recently being understood. In addition, there are a growing number of insults that are thought to contribute to the development of eosinophilic pneumonia. The most likely cause of AEP in our case is the re-initiation of smoking that was associated with exposure to the pesticide. Smoking or resumption of smoking has been associated with two-thirds of all cases of AEP published in the literature with only one case was associated with the pesticide. It is also important to note that the damaged airway epithelium is more likely to allow toxins to get into the lung and induce this type of reaction. Pyrethroids may need to be added to a growing number of insults that are thought to contribute to the development of eosinophilic pneumonia. It is also important to note that damaged airway epithelium is more likely to allow toxins to get into the lung and induce this type of reaction. Pyrethroids may need to be added to a growing list as a stimulus for this disease. Awareness of this is critical, not only for enhanced understanding of causal factors, but also to expedite critical diagnostic studies such as BAL that, as in this case, can be lifesaving. As noted above, her response to steroid therapy was rapid and dramatic. Further studies, such as provocation-type experiments in animal models, will help refine our understanding of pyrethroids and its role in AEP. In the meantime, it is our hope that case reports such as these may elevate the index of suspicion for AEP among physicians.

REFERENCES

Contact AMS for a complete list of references. AMS
Sick Day Management in Children and Adolescents with Type 1 Diabetes
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Department of Pediatrics, Arkansas Children's Hospital, Little Rock

Abstract:
Diabetic Ketoacidosis (DKA) has high morbidity and mortality and can be prevented. It is extremely important to give clear guidance to patients and families on how to manage diabetes during intercurrent illnesses to avoid complications of ketoacidosis, dehydration, uncontrolled or symptomatic hyperglycemia and hypoglycemia.

This descriptive review of clinical cases and management guidelines for sick days in children and adolescents with diabetes is provided as a resource for physicians who may take calls from parents of sick children with diabetes or manage these children in a clinic, emergency room or hospital setting.

Background:
Children and adolescents whose diabetes is under good metabolic control should not experience more illness or infections than children without diabetes. However, those with poor metabolic control may have altered immune function, increased susceptibility to and delayed recovery from infections. There is some evidence for impaired leukocyte function and low immunoglobulin G concentration in poorly controlled individuals with diabetes. Adults with diabetes in a Dutch study had a higher risk of urinary tract infections, bacterial skin, lower respiratory tract and mucous membrane infections.

Illnesses may increase blood glucose as a result of higher levels of cortisol leading to gluconeogenesis and insulin resistance. Illnesses with vomiting and diarrhea may lower blood glucose due to decreased food intake, poor absorption and slower gastric emptying. In an individual with type 1 diabetes (T1D), any illness can cause dehydration, ketone production and an increased insulin body utilization due to low insulin levels. Hyperglycemia and acidosis also cause osmotic diuresis, dehydration and obligate renal loss of electrolytes.

Pathophysiology:
DKA is caused by a decrease in effective circulating insulin associated with elevations in the counter regulatory hormones glucagon, cortisol, growth hormone, and catecholamines. This leads to increased hepatic glucose production and impaired peripheral glucose utilization, with resultant hyperglycemia and hyperosmolality. Ketones are produced by the liver from free fatty acids that are mobilized as an alternative energy source when there is insufficient glucose for intracellular metabolism. Ketones accumulate and metabolic acidosis develops because of increased lipolysis, increased ketogenesis and decreased ketone body utilization due to low insulin levels. Hyperglycemia and acidosis also cause osmotic diuresis, dehydration and obligate renal loss of electrolytes.

The biochemical criteria for DKA include hyperglycemia (blood glucose > 200 mg/dl) and metabolic acidosis (venous PH < 7.3 and/or bicarbonate ≤ 15 mmol/L). This is associated with glycosuria, ketonuria and ketonemia. DKA is categorized by the severity of the acidosis: mild (venous PH: 7.25 to 7.3, bicarbonate concentration: 15 to 18), moderate (venous PH: < 7.1 to 7.24, bicarbonate concentration: 10 to 14), and severe (venous PH: <7.1, bicarbonate concentration: <10).

Clinical cases and management guidelines:
Case 1:
A 4-year-old girl, with known type 1 diabetes (T1D) has a fever to 102°F. Her finger stick blood glucose (FSBG) is 450 mg/dl. She has moderate ketones in her urine. She is able to tolerate fluids by mouth and is not vomiting. Her mother calls the after-hours phone line for advice.

Home management guidelines:
1. Do not skip insulin even if not eating or vomiting. Even in fasting state, insulin is required for basal metabolic needs, which may increase during an acute illness situation.
2. Frequent monitoring of FSBG every three hours.
3. Check urinary ketones if FSBG > 240 or if fever or vomiting.
4. Hydration (see Table 1: Hydration guidelines) and Insulin are the keys to clear ketones and prevent progression to DKA.

5. Treat the underlying illness.

6. If sick looking and/or continued vomiting, please go to the nearest emergency room.

- If ketones are small:
  - Drink sugar free fluids (“age in years” ounces/hour) until ketones clear. For example if the patient is 10-years-old, they need to drink 10 oz/hour of appropriate fluids.

- If ketones are moderate to large:
  - Drink fluids (“age in years” ounces/hour) until ketones clear.
  - Check FSBG every three hours and correct with subcutaneous rapid acting insulin (Insulin Aspart/Insulin Lispro) per patients home correction insulin doses which is typically one unit for every 50 above 150 for children over five years of age and one unit for 100 above 150 for children less than five years of age.
  - If ketones large, in addition, give extra Insulin Aspart/Insulin Lispro: For children <10 years give two extra units and for children ≥10 year, give four extra units.

**Table 1: Hydration guidelines**

<table>
<thead>
<tr>
<th>Blood glucose (mg/dl)</th>
<th>Type of fluids (oral)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt; 180</td>
<td>Sugar free fluids (water, diet soda)</td>
</tr>
<tr>
<td>100 – 180</td>
<td>Drink ½ water and ½ sugary fluids</td>
</tr>
<tr>
<td>≤ 100</td>
<td>Sugary fluids like juice, sodas, gatorade</td>
</tr>
</tbody>
</table>

Ketone checks: Ketone strips are dipped in urine and checked at home as follows. (Figure 2)

**Figure 2: Ketones in urine**

4. Hydration (see Table 1: Hydration guidelines) and Insulin are the keys to clear ketones and prevent progression to DKA.

5. Treat the underlying illness.

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- If ketones large, in addition, give extra Insulin Aspart/Insulin Lispro: For children <10 years give two extra units and for children ≥10 year, give four extra units.

**Case 2:**

six-year-old girl with known T1D presents with fever and vomiting. Her FSBG is 500 mg/dl. She has moderate urine ketones. She is brought to the local emergency room.

**Emergency Room management guidelines:**

1. Hydration
   a. Fluids Bolus with normal saline at 20 ml/kg.
   b. Maintenance IV fluids: Dextrose free ½ Normal saline with 20 meq/L of potassium supplements at 1.5 to 2 times maintenance rate.
2. Ondansetron by mouth if needed [4 to 11 years: 4 mg three times a day as needed for nausea ; > 11 years : 8 mg three times a day as needed for nausea].
3. Recommended laboratory testing includes Basic Metabolic Panel (BMP), Venous blood gas (VBG), urine ketones.
4. FSBG every 2-3 hours and correct using home insulin regimen.
5. If urine ketones are “moderate to large”, give extra rapid acting insulin (as suggested in case 1 above).
6. Administer home Glargine or insulin Detemir dose at the usual time or dose if the child has not received it in the past 24 hours.
7. Treat the underlying illness

**Case 3:**

10-year-old girl with known T1D presented with fever to 104°F to the emergency room. The initial diabetes work up showed a FSBG: 250 mg/dl, urine ketones small and BMP showed no acidosis. She was admitted to the pediatric floor for observation. She received her long-acting basal insulin analogue dose at 9 pm. Her FSBG since admission have been elevated to 350 and 450 mg/dl. Her urine ketones are now moderate at midnight.

**Floor management guidelines:**

1. Dextrose free ½ normal saline with 20 meq/L of potassium supplements at 1.5 to 2 times maintenance.
2. Dip each urine void for ketones.
3. Check FSBG every 3 hours and correct based on home insulin regimen till ketones are trace to negative.
4. Home long-acting basal insulin analogue (either insulin Glargine or insulin Detemir). Administer the usual home dose if the child has not received it in the last 24 hours.
5. If the child clinically worsens or the FSBG are trending up, check a stat BMP to exclude possible development of DKA.

**Case 4:**

16-year-old boy known T1D who has not been feeling well for the past 2 days has a FSBG of 480 mg/dl. He has large urine ketones. He is not vomiting. He wears an insulin pump.

**Management guidelines for insulin pump users:**

1. Be aggressive with management per the guidelines listed below since there is no long acting insulin in the pump.
2. Change insulin pump site using fresh supplies and insulin vial as soon as possible.
3. Blood glucose is entered in the pump and the pump calculates the correction dose of insulin required based on the child’s home regimen. This is given using the pump and blood glucose is rechecked in 2 hours.
4. If blood glucose has not come down by 100 mg/dl, give a correction dose using subcutaneous Insulin Aspart/Insulin Lispro pen.
5. Set a temporary basal rate at 120% for 12 hours on the pump. The parents should know how to set this.
6. Check FSBG every 2-3 hours and correct using the pump.
7. Implement Hydration guidelines as per Table 1.
8. If you suspect a pump malfunction, switch to insulin pens and ask the parents to call the 1-800 number at the back of the pump to obtain a replacement pump.

Case 5:
10-year-old boy, known T1D presents with vomiting, diarrhea and poor oral intake. His FSBG is 70 mg/dl. He has moderate urine ketones.

Management guidelines for a sick child with diabetes with hypoglycemia and ketones:
1. Maintain hydration: Sugar containing PO fluids/ Dextrose containing [D5 1/2 normal saline with 20 mEq/L of potassium supplements at 1.5 to 2 times maintenance rate.
2. Check and correct FSBG every three hours, check urine for ketones with every void.
3. Treat the underlying illness. Replace meals with easily digestible carbohydrate (See Table 3) containing food and sugar containing fluids to prevent further ketosis.
4. Glucose tablets, glucose gels and glucagon should always be available to treat low blood glucose.

Hypoglycemia treatment guidelines:
- For bedside glucose <70 mg/dl, begin hypoglycemia treatment guidelines.
- If patient is on an insulin pump, suspend the pump until bedside glucose > 70 mg/dl.

Severe hypoglycemia (Patient unconscious/ seizing)
Glucagon IM/Subcutaneous is given into the deltoid or anterior thigh
- Glucagon dosing (< 20 kg: 0.5 mg, > 20 kg: 1 mg).
Check glucose 15 minutes post intervention.

Mild/Moderate hypoglycemia treatment (Patient awake and alert) – Rule of 15
Bedside glucose <70 mg/dl
- Treat with 15 grams FAST ACTING CARBOHYDRATES (4oz juice, 4 oz regular soda, 4 glucose tablets, glucose gel containing 15 gm carbohydrates).
- Recheck bedside glucose 15 minutes after oral carbohydrate treatment.
- If bedside glucose <70 mg/dl, repeat treatment and retest every 15 minutes until bedside glucose is >70 mg/dl.
- Once bedside glucose >70 mg/dl, have child eat regularly scheduled meal or give a snack (15 grams carbohydrates plus a protein) if more than 30 minutes before next meal.
- Do not include fast acting carbohydrates that were used to treat hypoglycemia in the child's carbohydrate calculation for insulin.

Summary:
Sick days should be managed aggressively to prevent progression to DKA. When vomiting occurs in a child or adolescent with diabetes, it should always be considered a sign of insulin deficiency until proven otherwise. Elevated blood glucose with moderate or large urinary ketones is more serious and reflects actual or impending DKA. FSBG and urine ketones should be frequently monitored. Insulin should never be stopped. Extra Insulin and hydration are the only ways to clear the ketones and prevent worsening to DKA.

In a child or adolescent with an intercurrent illness, urgent specialist advice must be obtained if blood glucose continues to rise despite extra insulin, if the child is becoming exhausted, confused, hyperventilating or if there is a change in the neurological status, mental confusion, loss of consciousness or seizures. Any of these signs and/or symptoms may indicate impending or present cerebral edema.

Resources: There are resources available for sick day management at home on the Arkansas Children's Hospital website.

Table 3: Easily digestible food containing 15 gm carbohydrates

<table>
<thead>
<tr>
<th>Food</th>
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<tbody>
<tr>
<td>½ cup juice (4 ounces) fruit juice/ regular cola/ regular jello</td>
</tr>
<tr>
<td>½ cup (4 ounces) hot cereal, macaroni, noodles, rice, mashed potato</td>
</tr>
<tr>
<td>¼ cup (2 ounces) pudding</td>
</tr>
<tr>
<td>1 cup soup</td>
</tr>
<tr>
<td>1 popsicle (15 gm)</td>
</tr>
</tbody>
</table>

References:
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