<table>
<thead>
<tr>
<th>Page</th>
<th>Title</th>
<th>Authors</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>Editors</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Case Report: An atypical case of pediatric dysautonomia: neurocardiogenic syncope in an 8-year-old male</td>
<td>King et al.</td>
</tr>
<tr>
<td>7</td>
<td>Drug-Induced Eosinophilic Pneumonia in the Setting of Rheumatoid Arthritis</td>
<td>Chernis et al.</td>
</tr>
<tr>
<td>11</td>
<td>Ocular Findings in Wernicke’s encephalopathy</td>
<td>Khatker et al.</td>
</tr>
<tr>
<td>15</td>
<td>The Tip of the Iceberg: A rare case of Massive Substernal Goiter discovered through Trans-radial Cardiac Angiogram</td>
<td>Jacob et al.</td>
</tr>
<tr>
<td>17</td>
<td>Spontaneous pneumopericardium in the intensive care unit</td>
<td>Abelhad et al.</td>
</tr>
<tr>
<td>18</td>
<td>Predictors of Severe Coronary Artery Lesions among Patients with End Stage Renal Disease</td>
<td>Pabba et al.</td>
</tr>
<tr>
<td>24</td>
<td>Hypokalemia in Patients treated with Nafcillin: A Retrospective Cohort Study</td>
<td>Gonzalo et al.</td>
</tr>
</tbody>
</table>
Faculty Mentors
Dr. Gabriel Aisenberg and Dr. Jennifer Swails

Editor-in-Chief
Katyayini Aribindi, MD

Associate Editors:
Ritesh Patel, MD
Astrid Grouls, MD
Nadia Abelhad, MD
Abin Puravath, MD
Case Report: An atypical case of pediatric dysautonomia: neurocardiogenic syncope in an 8-year-old male

King, Nicholas E
Arnold, Kristen
Stang, Kristina
Khetan, Nikita MD

Abstract:
Dysautonomia is a clinical spectrum ranging from reflex syncope to autonomic failure. Neurocardiogenic “reflex” syncope (NCS) is the most common cause of syncope, typically presenting between the ages of 15 and 19, and is due to an imbalance of sympathetic and parasympathetic tone leading to under-perfusion of the brain. It is often preceded by dizziness and tinnitus, and further diagnostic tests for cardiac or neurologic etiologies will be negative. Treatment consists of volume expansion and avoidance of potential triggers. Here, we present a unique case of NCS dysautonomia in an 8-year-old male. The patient presented with syncopal episodes and bradycardia. His inpatient work-up including formal electrocardiograms, echocardiograms, and head imaging were unremarkable for serious conditions. A tilt table test confirmed the diagnosis of moderate NCS dysautonomia.

Introduction:
Syncope is a common emergency room presentation in the pediatric population and has etiologies ranging from benign to life-threatening. It is most often due to an underlying dysregulation of the autonomic nervous system that leads to a transient under-perfusion of the brain. This dysregulation ranges from “reflex” syncope to total autonomic failure, collectively known as dysautonomia. Neurocardiogenic “reflex” syncope (NCS) is the most common subtype of reflex syncope, typically presenting in the late teenage years. Here, we present an atypically early case of NCS and offer a brief review of the literature surrounding this common presentation.

Case Description:
A healthy 8-year-old male with no significant past medical history presented with the acute onset of multiple syncopal episodes. As he was walking, he lost consciousness, collapsed, and began to shake. The episode lasted 30 seconds, after which the patient awoke and returned to a fully alert and orient state after approximately one minute. No bowel or bladder incontinence, tongue biting, or other stigmata of a seizure were noted. Over the following 24 hours, he experienced three more similar episodes with no clear trigger that were preceded by variable dizziness, tinnitus, and chest pain, and followed by a quick return to baseline with no evidence of a post-ictal state.

Vital signs throughout his admission were significant for a bradycardic heart rate averaging 50 beats per minute (BPM) (Figure 1). The heart rate would respond to activity with accelerations to over 100 BPM. The remaining aspects of his exam were intact, including orthostatic blood pressures. Electrocardiogram (EKG) (Figure 1), echocardiogram, and cardiac enzyme evaluation were unremarkable. Suspecting dysautonomia, the patient underwent a tilt-table test that demonstrated moderate NCS dysautonomia.

The patient was discharged on fludrocortisone, and counseled on adequate fluid and salt intake, and the need to refrain from high-intensity physical activity.
Discussion:
Approximately 1 in 7 children will experience a syncopal event, most often between the age of 15 and 19 years.\(^1\sim3\) Of these events, a dysautonomic syndrome is the most common cause.\(^1\sim3\) Dysautonomia describes a spectrum of autonomic dysregulation, encompassing "reflex" syncope, neurogenic syncope, and other syndromes of total autonomic failure (Table 1).\(^5\)

"Reflex" syncope, more appropriately termed neurocardiogenic (NCS) or vasovagal syncope, is the most common subtype of dysautonomia, accounting for 66\%-90\% of syncopal episodes.\(^2\sim3,5\) It is characterized by a physiologic imbalance between sympathetic and parasympathetic tone that leads to bradycardia and hypotension. As a result, the brain is transiently under-perfused and the patient experiences syncope. The traditional explanation is based on the Bezold-Jarisch reflex,\(^5\) in which hypotension is sensed by the carotid sinus, triggering an increase in cardiac contractility and rate afferent nerve fibers. The resultant increase in cardiac pressure is sensed by baroreceptor C-fiber afferent nerves which then trigger a paradoxical increase in vagal tone resulting in bradycardia, decreased contractility, and relative sudden hypotension leading to syncope. This explanation, however, does not account for all causes of reflex syncope. Higher neural centers clearly mediate vasovagal syncope such as at the sight of blood, while inappropriate autonomic regulation mediates unprovoked NCS.\(^6\)

**Table 1:** Dysautonomic etiologies of syncope
The typical presentation of NCS is syncope preceded by a prodrome, which can include dizziness, loss of hearing, vision changes, diaphoresis, pallor, or flushing. The syncopal episode typically lasts a few minutes or less with a rapid return to baseline and no post-ictal state. Objective signs such as bradycardia or hypotension may be present with the episode.

NCS can be divided into classical and non-classical forms, with the classical form having a clear trigger, such as an increase in vagal tone with intense emotion or micturition, and the non-classical form lacking a clear trigger.

Here, we present a unique case of early onset NCS and dysautonomia with an atypical presentation. A young patient with acute onset of unprovoked syncopal episodes caused an increased suspicion that the etiology was a significant cardiac or neurologic condition, triggering extensive evaluation. However, the reported prodrome of tinnitus and dizziness, combined with significant bradycardia and the absence of other findings guided the diagnosis to NCS dysautonomic syncope. With the exclusion of more serious diagnoses by cardiac evaluation and the positive findings of the tilt-table test, we were able to diagnosis this patient with NCS dysautonomia.

Reaching the diagnosis of NCS focuses on ruling out more serious underlying cardiac or central nervous system diseases. The diagnostic test of choice for NCS remains the head-up tilt table test, which has a sensitivity of 75% and a specificity of 90%. The tilt challenges the autonomic system to compensate for the change in head position and maintain cerebral perfusion, thus unmasking any degree of autonomic dysfunction. If the patient becomes symptomatic, the blood pressure drops, or vital signs do not respond to the change in position, then the test is considered positive. More recent work has identified p-wave dispersion as a potential marker of autonomic dysfunction; however, its diagnostic significance is undetermined.

Therapy is directed at maintaining intravascular volume through adequate salt and fluid intake. Medications such as beta blockers, midodrine, and fludrocortisone have shown mixed results in trials, and can be considered as adjuvants to lifestyle modifications. However, no medication is FDA-approved for the treatment of NCS.

Dysautonomic syndromes continue to pose challenges to clinicians and patients. The lack of reliable diagnostic tests for NCS despite its frequency results in many patients like ours undergoing extensive and potentially invasive evaluations.

Table 1: A review of the dysautonomic causes of syncope. Neurocardiogenic syncope (NCS), postural orthostatic tachycardia syndrome (POTS), multiple system atrophy (MSA).

<table>
<thead>
<tr>
<th>Type</th>
<th>Examples</th>
<th>Pathophysiology</th>
<th>Manifestations</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>“Reflex” syncope</td>
<td>Vasovagal NCS</td>
<td>Sympathetic/parasympathetic tone imbalance</td>
<td>Syncope with or without trigger</td>
<td>Volume expansion, avoid triggers (if known)</td>
</tr>
<tr>
<td>Neurogenic syncope</td>
<td>Orthostatic hypotension POTS</td>
<td>Delayed autonomic response leading to decreased cardiac filling</td>
<td>Dizziness, syncope upon standing</td>
<td>Volume expansion, standing slowly, compression garments</td>
</tr>
<tr>
<td>Autonomic failure</td>
<td>Pure autonomic failure MSA</td>
<td>Total loss of autonomic function</td>
<td>Chronic fatigue, syncope, urinary/bowel and sexual dysfunction</td>
<td>Volume expansion, compression garments, vasopressors if needed</td>
</tr>
</tbody>
</table>

The typical presentation of NCS is syncope preceded by a prodrome, which can include dizziness, loss of hearing, vision changes, diaphoresis, pallor, or flushing. The syncopal episode typically lasts a few minutes or less with a rapid return to baseline and no post-ictal state. Objective signs such as bradycardia or hypotension may be present with the episode. NCS can be divided into classical and non-classical forms, with the classical form having a clear trigger, such as an increase in vagal tone with intense emotion or micturition, and the non-classical form lacking a clear trigger.
studies. Further work is needed to identify stronger diagnostic tests and elucidate the pathophysiology of NCS to better guide therapy. Clinicians need to be aware of the frequency of NCS as they manage syncopal patients to better guide their clinical reasoning.

References
Drug-Induced Eosinophilic Pneumonia in the Setting of Rheumatoid Arthritis

Julia Chernis*
Elliot Ghorayeb*
Mehmet Camkurt, MD
Sujith V Cherian, MD
Bobak Akhavan, MD

Abstract:
Eosinophilic pneumonia (EP) is a rapidly progressive respiratory condition characterized by cough, dyspnea, respiratory failure, and pleuritic chest pain. Numerous reports have classified EP as a rare side effect in a wide range of medications, including immunomodulating agents such as sulfasalazine. Due to nonspecific symptoms, the clinical presentation may lead to a delayed diagnosis. Here, we present a case of a 60-year-old woman with rheumatoid arthritis who developed EP secondary to sulfasalazine.

Introduction:
Eosinophilic pneumonia (EP) is a condition characterized by a nonproductive cough, dyspnea that may lead to respiratory failure, and pleuritic chest pain that develops either acutely in less than seven days or chronically over the course of more than two weeks. It has a diverse range of etiologies, including parasitic infection, environmental triggers, malignancy, pharmacologic agents, and idiopathic causes. Among these drug induced causes is sulfasalazine, a disease modifying antirheumatic drug (DMARD) used in the treatment of rheumatoid arthritis.

Case Description:
A 60-year-old female presented to the Emergency Department with a nonproductive cough and dyspnea for nine weeks. She had been prescribed antibiotics and a short course of steroids a month prior to presentation with improvement in her symptoms, but returned due to symptom recurrence. The patient was a 25-pack-year smoker whose past medical history included rheumatoid arthritis managed with hydroxychloroquine and sulfasalazine initiated 11 months prior to admission.

On physical examination, she was in respiratory distress with accessory-muscle use. She was afebrile and tachycardic to 109 beats per minute with a normal blood pressure. Oxygen saturations were 95% on room air, but dropped and eventually required 3 L of supplemental oxygen to maintain saturations > 88%. Auscultation of the chest revealed coarse wheezes bilaterally. Complete blood count revealed leukocytosis with absolute eosinophil count of 0.71 K/uL (8.1%) and an IgE level of 496 IU/mL. Infectious disease workup was negative, including testing for bacterial, parasitic, and fungal organisms. Sputum stain and culture demonstrated only normal flora and 4+ WBCs. Workup for autoimmune etiology showed a negative ANA, anti-histone antibody, and RA factor. The chest radiograph showed bilateral mild upper lobe interstitial opacities (Figure 1). Chest computed tomography showed bilateral, scattered, patchy ground glass opacities, predominantly in the upper lobes (Figure 2). A bronchoscopy with bronchoalveolar lavage was performed, which revealed a markedly elevated (77%) eosinophil counts with a WBC count of 1,578 WBC/uL. Transbronchial biopsies showed eosinophilic infiltration into alveolar and interstitial spaces consistent with EP (Figures 3, 4). Given her history of sulfasalazine use, a rare but known causative agent of drug-induced EP, a diagnosis of EP secondary to sulfasalazine use was made.

The patient’s sulfasalazine regimen was discontinued and 40 mg prednisone therapy resulted in rapid clinical improvement in her pulmonary function. She was discharged with a tapering regimen of oral prednisone with trimethoprim-sulfamethoxazole prophylaxis and reported resolution of her symptoms 2 days after discharge. Patient followed up in the pulmonary clinic two months after discharge and was doing clinically well with no respiratory insufficiency after being tapered off of prednisone.
Figure 1. Chest Radiograph. Chest radiograph on admission showing bilateral upper lobe interstitial prominences and scattered subsegmental atelectasis.

Figure 2. Chest Computed Tomography scan of upper lung lobes showing bilateral, scattered, patchy ground glass opacities.

Figure 3. Pulmonary parenchyma demonstrating fibrinous exudate and numerous inflammatory cells. 10X magnification.
Discussion:
Acute eosinophilic pneumonia (AEP) is characterized by a nonproductive cough, fever, pleuritic chest pain, and dyspnea that may lead to respiratory failure requiring intubation that develops acutely in less than seven days. The clinical picture of chronic eosinophilic pneumonia (CEP) differs in that patients are typically afebrile at presentation with milder respiratory symptoms that last over two weeks before the onset of respiratory failure. Although AEP is often characterized by neutrophilic leukocytosis, peripheral eosinophilia and elevated IgE are a hallmark of the CEP. Both are characterized by bilateral basal inspiratory crackles on physical exam and a chest radiograph showing bilateral, diffuse, mixed reticulonodular opacities. Small pleural effusions are also commonly seen. Bronchoalveolar lavage demonstrating an increased eosinophil fraction of >25% compared to a normal fraction of <1% confirms diagnosis. A lung biopsy is rarely needed when the other criteria are present.

This patient’s symptom duration was greater than 7 days, she was afebrile on presentation, and only required 3L of O2, which more resembles CEP. Smoking, specifically initiation or increased frequency, is more commonly associated with AEP, and was thus ruled out as a cause for her condition. AEP is treated with high-dose steroids with a gradual taper of up to four weeks, which generally leads to rapid and progressive improvement without relapse. Because of CEP’s longer time course and tendency to relapse, it is treated with a 6 months of corticosteroid therapy.

In the context of lung disease, EP is a rare syndrome, and sulfasalazine-induced EP is even rarer. A review of 196 case reports has shown sulfasalazine to account for only 6% of all published cases of drug-induced EP between 1990 and 2017. Ultimately, the diagnosis was reached on the basis of a combination of symptom timing, imaging, diagnostic testing showing eosinophilic predominance peripherally and in the BAL, and marked improvement with steroids after ruling out infectious, environmental, and autoimmune etiologies. Sulfasalazine-induced EP has previously been reported to occur after a time lag, which could explain why this patient’s symptoms occurred 11 months after starting the medication. Given its nonspecific presentation, the diagnosis is often delayed. However, the recognition of drug-induced EP is especially important because, in addition to symptomatic treatment with steroids, withdrawal of the offending agent is crucial for its resolution and prevention of its recurrence.

Conclusion:
It is important to include eosinophilic pneumonia in a differential when working up acute
respiratory failure within the appropriate clinical context. Bronchoscopy is key in diagnosis. After reviewing more common etiologies, a consideration should be made for drug-induced eosinophilic pneumonia secondary to sulfasalazine, which carries an excellent prognosis once the offending agent is removed.

Acknowledgements:

Histology images provided by Michaelangelo Friscia, DO, Pathology PGY-1 (Michaelangelo.Friscia@uth.tmc.edu).

*Authors above are two co-first authors

References

Ocular Findings in Wernicke’s encephalopathy

Meesha Khatker
Subhan Tabba
Ashwini Kini, MD
Bayan Al Othman, MD
Andrew G. Lee, MD

Abstract:
Wernicke’s encephalopathy can present as a variety of symptoms including as isolated ocular manifestations. There are many risk factors to developing Wernicke’s encephalopathy, but it is important to determine the correct diagnosis and treat the condition early, as there can be severe complications associated with delayed or incorrect treatment.

Introduction:
Wernicke’s Encephalopathy (WE) is a syndrome of central and peripheral nervous system lesions due to thiamine (Vitamin B1) deficiency. Although classically described in the setting of alcohol abuse, WE can be due to any cause of decreased vitamin intake, decreased absorption or increased metabolism. WE is typically associated with a triad of ophthalmoplegia, confusion, and ataxia, however up to 90% of patients may present with an incomplete triad or with other associated symptoms. The ocular findings of this disease can vary but most commonly ophthalmoplegia (e.g. incomplete horizontal gaze palsy with abduction deficits) and/or nystagmus.

The diagnosis of WE is made clinically (e.g., Caine Criteria) and a full work-up for alternative etiologies should be carried out to exclude other causes of nervous system disease. Characteristic brain MRI findings are also helpful in making a diagnosis.

Treatment involves early recognition followed by rapid and aggressive replenishment of thiamine parenterally. It is important that thiamine supplementation begin immediately before any additional glucose load (e.g. 5% dextrose in water) is given to the patient. While WE, if treated early, is often reversible, delayed treatment or severe cases may have permanent neurologic deficits (i.e., Korsakoff syndrome).

Case Description:
A 28-year-old, previously healthy, woman at 21 weeks gestation presented to the ophthalmologist with subacute painless, binocular diplopia worse on lateral gaze associated with 10 weeks of nausea and vomiting (hyperemesis gravidia) not responsive to ondansetron, aromatherapy, or ginger. Her husband states that in the preceding week she has also become confused and is having trouble with her memory.

She has no relevant past medical history. She ate a good diet and was only taking pre-natal vitamins.

Patient had a blood pressure of 118/78, breathing rate of 16 breaths per minute, pulse of 80 beats per minute and temperature of 98.5°F. On exam she was alert, but oriented only to person and not time or place. On ophthalmic exam, she exhibited spontaneous upbeat nystagmus and bilateral abduction deficits.
Speech was clear and fluent with good repetition, but poor comprehension and naming. She recalled 1/3 objects at 5 minutes. Facial sensation was intact to pinprick in all three divisions bilaterally. Corneal responses were intact. Face was symmetric with normal eye closure and smile. Hearing was normal to rubbing fingers. Palate elevated symmetrically. Phonation was normal. Head turning and shoulder shrugging were intact. Tongue was midline. There was no pronator drift of out-stretched arms. Muscle bulk and tone were normal. Strengths was full bilaterally. Reflexes were 2+ and symmetric at the biceps, triceps, knees and ankles. Sensation on light touch, pinprick, position sense and vibration sense were intact in fingers and toes. Rapid alternating movements and fine finger movements were intact. There was no dysmetria on finger to nose and heel-knee-shin. However, there were some extraneous movements during the maneuver. Romberg was absent. Posture was normal. Gait was ataxic with wide steps, and notable arm swing and turning. Patient was unable to complete heel and toe walking maneuvers. The rest of her exam was normal. Her best corrected visual acuity was 20/20 bilaterally. Her pupils were 4mm in the dark and 3mm in the light. No relative afferent pupillary defect (RAPD) was observed. External examination and slit lamp biomicroscopy was unremarkable. Her intraocular pressure was normal at 14 mmHg bilaterally. Fundoscopic exam was normal with no evidence of abnormal disc or retina. The Humphrey visual field analyzer was used to measure the patients visual fields, which were found to be normal bilaterally. Macular and optic nerve optical coherence tomography (OCT) tests are noninvasive diagnostic tests that look at a cross section of the macula and retina. These tests were conducted and the macula, optic nerve and retina were normal bilaterally.

Her complete blood count and comprehensive metabolic panel were unremarkable except for mild anemia. A magnetic resonance imaging (MRI) of the brain showed T2 hyperintensities of the tectal plate and the periaqueductal white matter.

The patient was diagnosed with Wernicke’s encephalopathy secondary to hyperemesis gravidarum and given 500 mg of thiamine intravenously, 3 times a day for 2 days before switching to 250 mg intramuscularly, daily.

On follow-up 2 weeks later, her ophthalmoplegia and nystagmus had completely resolved, and she was alert and oriented to person, place, and time. She remained on intramuscular thiamine throughout her second trimester, until her hyperemesis gravidarum resolved.

Discussion: The triad of signs classically described in Wernicke’s encephalopathy includes ophthalmoplegia, altered mental status, and ataxia resulting from cerebellar dysfunction. However, WE may present with initially isolated ocular findings with or without disturbance of consciousness. Other associated symptoms include hypothermia, hypotension, and peripheral sensory neuropathy of the lower extremities. Our patient did not have ataxia. Absence of the full triad likely leads to underdiagnosis of WE. In one necropsy study 80% of 131 patients were not
diagnosed during their life and 34% of these patients presented with ocular findings alone. Only around 10% of patients present with the classic triad. Ocular presentations of WE can vary and most often consist of an incomplete horizontal gaze palsy with abduction deficits and horizontal nystagmus. The nystagmus however may be vertical or horizontal and may be spontaneous and/or gaze-evoked on exam. Bilateral internuclear ophthalmoplegia has also been described in WE.

The underlying cause of WE is thiamine (Vitamin B1) deficiency. Thiamine Pyrophosphate or TPP a cofactor required for activity of the glycolytic enzyme pyruvate dehydrogenase, the citric acid cycle enzyme alpha-ketoglutarate dehydrogenase and branched-keto-acid dehydrogenase, and the pentose phosphate pathway enzyme transketolase. The cause of the ocular findings is damage and secondary reversible cytotoxic edema resulting from decreased glucose metabolism involving the cranial nerve nuclei. This is secondary to the shunting toward less efficient anaerobic and acid producing pathways of metabolism without the thiamine cofactor. The thiamine deficient membranes cannot maintain osmotic gradients leading to edema of intracellular and extra cellular spaces. The ataxia results from cerebellar damage for the same mechanism.

This syndrome is classically described in alcohol abuse. However, thiamine deficiency can result from decrease in vitamin consumption (anorexia), decreased absorption, or excessive rapid loss (e.g., hyperemesis gravidarum, bariatric surgery). Our patient suffered from hyperemesis gravidarum, a severe form of nausea and vomiting of pregnancy which is associated with nutritional deficiency, fluid, electrolyte, acid-base imbalance, and weight loss. Other causes of decreased vitamin absorption include malnutrition for any reason including alcoholism, prolonged parenteral nutrition without vitamin supplementation, bariatric surgery, liver disease, and severe anorexia nervosa. Thiamine deficiency can also be secondary to increased metabolism such as in the setting of hyperthyroidism, sepsis, or malignancy. The symptoms of WE can be exacerbated by an increase in carbohydrate intake, such as by administration of intravenous glucose or dextrose.

The differential diagnosis for a patient with altered mental status and ataxia with suspected Wernicke’s includes cerebrovascular accident, hepatic encephalopathy, alcohol withdrawal syndrome/delirium tremens, chronic hypoxia, and normal pressure hydrocephalus. In patients with ocular findings such as nystagmus or ophthalmoplegia, especially when they are isolated, the differential includes brainstem stroke or structural lesion, ocular myasthenia gravis, Miller Fisher variant of Guillain-Barre syndrome thyroid eye disease, and internuclear ophthalmoplegia due to MS.

The diagnosis of WE is clinical, based on history and presentation, and is supported by the finding of two or more Caine criteria on exam (nutritional deficiency, altered mental status or memory impairment, oculomotor abnormalities, cerebellar dysfunction). Sensitivity with two or more criteria in the setting of alcoholism without hepatic encephalopathy is almost 100 percent. In cases suspected of having WE, thiamine should be administered immediately even while the diagnostic work up is still pending. A full work-up should be done to exclude other causes of neurological disease, including a complete blood count (CBC) and comprehensive metabolic panel (CMP). A CBC is necessary to rule out anemia or infectious causes for the patients acute neurological symptoms while a CMP would indicate any metabolic reasons for the patients presentation including electrolyte abnormalities, acid/base abnormalities, changes in blood glucose and proteins, and indicators of kidney and liver health. The activity of transketolase, an erythrocyte enzyme which uses thiamine as a cofactor, is typically low in patients with untreated thiamine deficiency, though this is not a diagnostic requirement.

Brain MRI is also used as a diagnostic tool. The characteristic radiologic findings include reversible cytotoxic edema shown as hyperintensity on T2 MRI, most commonly in periventricular and periaqueductal midline regions. Symmetric involvement of the medial thalami, mammillary bodies and tectal plate can also be seen. Other findings classically seen in alcoholics such as atrophy of the mamillary bodies and cerebellar vermis are seen in patients with WE due to alcoholism.
Treatment of WE is early, rapid parenteral administration of thiamine. Patients who are already at risk of WE due to malabsorption or alcoholism may have unreliable gastrointestinal absorption of thiamine, so in addition to being too slow, oral treatment is less bioavailable. Hence, intravenous administration helps restore serum concentrations of the vitamin quickly. The recommended starting dose is 500mg, three times a day for 2 days, then 250mg intramuscularly once a day for an additional 5 days. Glucose or dextrose administration without co-administration of thiamine in the setting of dehydrated patient could further worsen the condition due to consumption of remaining cofactors. To avoid the consequences of irreversible neurologic deficits and Korsakoff syndrome it is recommended that at-risk patients with confusion, ophthalmoplegia, or ataxia receive parenteral thiamine before laboratory confirmation is available. Concomitant administration of magnesium, an essential cofactor to convert thiamine into its active diphosphate and triphosphate forms, is also recommended.

Ocular findings in WE typically rapidly resolve with treatment, often within hours to days. The ocular signs are the least likely to remain as residual symptoms compared to ataxia or confusion. If ophthalmoplegia or nystagmus does not resolve with thiamine, other causes should be evaluated. Ataxia and confusion should resolve within weeks.

**Conclusion:**
Wernicke’s Encephalopathy classically presents as a triad of ocular abnormalities (e.g., ophthalmoplegia, nystagmus), altered mental status and ataxia, though this complete triad is rarely seen in practice. WE should be suspected in any patient with these findings alone or in combination and especially if there is a risk factor for thiamine deficiency (e.g., poor intake, malabsorption, emesis, or increased metabolism). Rapid correction of the deficiency with high dose parenteral thiamine is essential to reversing the symptoms and signs and should be administered empirically in patient with suspected thiamine deficiency, especially before any glucose infusion. Oculomotor disturbances typically fully resolve, though there may be residual deficits in patients with confusion or ataxia.

**Affiliations:**
1. Baylor College of Medicine, 1 Baylor Plaza, Houston, TX 77030
2. McGovern Medical School, 6431 Fannin Street Houston, TX 77030
3. Department of Ophthalmology, Blanton Eye Institute, Houston Methodist Hospital, 6550 Fannin St, Houston, TX 77030
4. MCPHS University School of Optometry, 10 Lincoln Sq., Worcester, MA 01608

**References:**
The Tip of the Iceberg: A rare case of Massive Substernal Goiter discovered through Trans-radial Cardiac Angiogram

Robin Jacob, MD
Katia Bravo-Jaimes, MD
Christopher Do, MD
Konstantinos Charitakis, MD

Introduction:
Goiters may sometimes only be revealed through imaging, such as computed tomography scans. Knowledge of goiters allow for routine monitoring in case condition changes. Here we present an 83-year old male who arrived to the emergency room with dyspnea on admission. Labs and imaging warranted coronary angiography. When angiogram was attempted, contrast injection revealed a highly vascularize structure which was later identified as a massive substernal goiter. In conclusion, angiography may reveal incidental findings that alter or change a patient’s prognosis.

Case Description:
An 83-year-old male with hypertension and chronic kidney disease presented with dyspnea. Vital signs were stable, and physical exam revealed findings of volume overload with lower extremity pitting edema. Of note, thyroid exam was normal with no enlargement, tenderness, or surrounding lymphadenopathy. Bloodwork revealed minor troponin elevation. Chest x-ray showed bilateral pleural effusions (Figure 1). Transthoracic echocardiogram revealed ejection fraction of 40-45%. Coronary angiography was attempted via right trans-radial approach due to a concern for non ST elevated myocardial infarction. There was difficulty engaging the JL 4 catheter but after cannulation and contrast injection a highly vascularized structure was noted (Figure 2) anterior to the cardiac silhouette. Trans-radial approach was aborted and right femoral access was obtained. Cardiac angiogram showed severe stenosis in the mid left circumflex, first obtuse marginal and mid right coronary artery. The procedure was stopped with plan for staged percutaneous coronary intervention (PCI) given the elevated creatinine. A chest computerized tomography (CT) was obtained, which showed bilobar enlargement of the thyroid gland with the left lobe extending into the mediastinum (Figure 3). Given normal thyroid function tests, negative Pemberton’s test, and minor progression on imaging (we were able to locate a prior CT chest from 6 years ago), Endocrinology consultants recommended observation. Patient later underwent successful PCI to a right coronary artery lesion via left trans-radial approach.

Figure 1: Chest roentgenography on admission showing bilateral pleural effusion, widened mediastinum, and deviated trachea.
Discussion:
Subternal goiter is a rare condition, defined as 50% of the thyroid being below the thoracic inlet, accounting for 5 to 20% of thyroidectomies.¹ To our knowledge, this is the first time that a massive substernal goiter was diagnosed as an incidental finding during cardiac angiography via right transradial approach. Potential risks of performing coronary angiography in patients with substernal goiter not only include those related to the usual vascular complications but also provoking iodine-induced thyrotoxicosis.²

References:
Spontaneous pneumopericardium in the intensive care unit

Nadia Isabel Abelhad, BS, MD
Katia M. Bravo-Jaimes, MD
Francisco Fuentes, MD

Introduction:
A 64-year old man with remote kidney transplant and coronary artery bypass grafts presented with increasing respiratory distress and altered mental status due to respiratory syncytial virus pneumonia and herpes simplex virus encephalitis. His course was complicated by development of acute respiratory distress syndrome, which required prolonged intubation and eventual tracheostomy. One week after tracheostomy, he underwent brain magnetic resonance imaging using a portable ventilator. Immediately after, he was noted to have subcutaneous emphysema, pneumopericardium and pneumomediastinum on chest radiography (Figure 1A). His heart rate was 108 beats per minute and blood pressure was 120/72 mmHg. His physical exam was notable for distant heart sounds. Bruit de Moulin and Hamman’s signs were absent. The electrocardiogram showed sinus tachycardia. He was treated conservatively with 100% oxygen and two days later his pneumopericardium improved, (Figure 1B) and ultimately resolved.

Discussion:
Pneumopericardium most commonly results from blunt or sharp chest trauma. However, it has been reported after pericardiocentesis, catheter ablation, thoracic surgery, endotracheal intubation and positive pressure mechanical ventilation. In the absence of tracheobronchial or gastroesophageal-pericardial fistulae, it is produced due to increased pressure gradients between alveoli and interstitial space leading to alveolar rupture with air tracking from the pulmonary perivascular sheaths to the hilum and subsequent pericardium (Macklin effect). In this patient, the use of a portable ventilator for the MRI caused barotrauma with resultant pneumopericardium. Though this patient improved with conservative management alone, surgical correction would be indicated next if the patient did not respond or hemodynamically instability occurred.

References

Figure 1. A: Subcutaneous emphysema marked by the asterisk, pneumopericardium marked by arrows, and pneumomediastinum marked by arrowheads. B: Resolving of previously seen subcutaneous emphysema, pneumopericardium and pneumomediastinum.
Predictors of Severe Coronary Artery Lesions among Patients with End Stage Renal Disease

Krishna Pabba, MD  
Fisayomi Shobayo, MD  
Michael Hust, MD  
Tariq Thannoun, MD  
Vincent Gacad, MD  
Gabriel M Aisenberg, MD

Abstract:
Background: Coronary atherosclerosis is highly prevalent among patients with end stage renal disease (ESRD) undergoing hemodialysis. Risk stratification tools have not been specifically created for these patients to determine when coronary angiography is necessary, therefore making the diagnostic evaluation of acute coronary syndrome a clinical challenge. Our study aims to determine predictors of severe coronary atherosclerosis in patients with ESRD.

Methods: We retrospectively identified patients 18 years of age or older with ESRD on dialysis admitted to our hospital between 2010 to 2017, who had undergone a coronary angiogram during the hospitalization. We excluded patients with ST-elevation myocardial infarction. The main outcome variable was the presence and severity of coronary atherosclerotic lesions. A multiple regression model was used to find predictors of severe coronary lesion, defined as those that would benefit from some form of revascularization.

Results: Among 238 patients, 130 (56%) patients had angiographic severe coronary lesions, 52 (22%) had less than severe lesions, and 51 (22%) had no coronary lesions. A multiple regression analysis found diabetes mellitus ($p = 0.0001$), new ST depressions ($p = 0.0092$), elevated serum troponin I levels ($P=0.0104$), and angina pectoris ($p = 0.0126$) being associated with severe coronary lesions.

Conclusions: Diabetes mellitus, new ST depressions on electrocardiogram, elevation of serum troponin I levels, and angina pectoris were associated with severe lesions on coronary angiography among patients with ESRD undergoing dialysis. Prospective trials will be needed to determine if these markers can better select candidates for coronary angiography in ESRD patients presenting with acute coronary syndrome.

Introduction:
Coronary atherosclerosis is highly prevalent among patients with ESRD. Cardiovascular disease accounts for 50% of mortality in hemodialysis patients with 52% of ESRD patients suffering from an acute myocardial infarction within 2 years of hemodialysis initiation.1,2

Although coronary angiography is the gold standard in detecting significant coronary atherosclerosis, the intervention is associated with complications and high costs in patients with ESRD. Risk stratification tools have not been specifically created for these patients to determine when angiography is necessary, therefore making the diagnostic evaluation of acute coronary syndrome a clinical challenge. This study aims to determine predictors of severe atherosclerosis in patients with ESRD.

Methods:
Subject Population

The study was undertaken at Lyndon B. Johnson Hospital - a tertiary-care, county-based center in Houston, Texas, after obtaining Institutional Review Board approval (HSC-MS-18-0047); patient consent requirements were waived. We collected information from electronic medical records from November 1st 2010 to December 31st 2017.
Inclusion criteria: Patients 18 years of age or older who have end-stage renal disease (ESRD) on dialysis, were admitted to the hospital, and had undergone a coronary angiogram during the hospitalization.

Exclusion criteria: Patients younger than 18 years of age and those with incomplete or inaccurate medical records were excluded. We also excluded patients with ST segment elevation myocardial infarction, for this would have been an indication for angiogram regardless of any other predictor. Patients with ESRD referred to have a coronary angiogram from the clinics were also excluded.

Data Collection
From the examined medical records we retrieved patients’ age, gender, and ethnicity; whether the patient reported chest pain on presentation, and if that pain was interpreted as being consistent with angina pectoris. We evaluated if there was history of diabetes mellitus, coronary bypass graft or percutaneous coronary intervention, and the type of dialysis (scheduled three times weekly or emergent only, modality adopted among patients with no funding as described elsewhere). We recorded, when available, the results of the highest troponin level for the event (normal = 0 - 0.045 ng/mL), the serum hemoglobin A1c, the description of the electrocardiogram (emphasizing the presence of ST-segment depression, non-specific wave abnormalities, T wave inversions, new Q waves, new right or left bundle branch blocks, and signs of left ventricular hypertrophy). We recorded echocardiographic evidence of a decreasing left ventricular ejection fraction, wall motion abnormalities, and signs of left ventricular hypertrophy. We documented, when available, evidence for reversible ischemia and wall motion abnormalities on nuclear stress test, as well as findings on coronary computerized angiogram.

The main outcome variable was the presence and severity of coronary artery atherosclerotic lesions. We considered as severe coronary artery disease the presence of any lesion causing a narrow flow greater than 70%. These patients were compared with those with milder or no lesions.

Statistical Analysis
MedCalc Statistical Software version 18 (MedCalc Software bvba, Ostend, Belgium; http://www.medcalc.org; 2018) was utilized for the statistical analysis. Categorical variables were analyzed using the Fisher exact test, and discrete variables were analyzed using the Student t-test for unpaired samples. A multiple regression model was used to assess the impact of the variables found to be significantly associated with severe lesions by univariate analysis. A two-sided p < 0.05 was considered indicative of statistical significance.

Results:
Two hundred and thirty-three patients met inclusion criteria. Their baseline characteristics are present in Table 1. Eighteen (8%) patients were included more than once (15 had 2 coronary angiograms, 1 had 3, and 2 had 4 each). All patients but two, who were treated with peritoneal dialysis, received hemodialysis for renal-replacement therapy. Among 168 diabetic patients, only 46 (28%) patients had serum hemoglobin A1c levels above 7%.
The indication to obtain a coronary angiogram was stated in 224 patients: evaluation of acute coronary syndrome was the most frequent (163 cases), followed by assessment of etiology of heart failure (38 cases), elevated serum troponin I levels (11 cases), abnormal stress test (8), and evaluation of aortic stenosis prior to valve replacement (4 cases).
In our cohort, 130 (56%) patients had angiographic severe coronary lesions, 52 (22%) had less than severe lesions, and 51 (22%) had no coronary lesions. A comparison of patients with severe coronary lesions and those with less than severe or no coronary lesions is presented in Table 2.

<table>
<thead>
<tr>
<th></th>
<th>Severe lesions n=130</th>
<th>Less than severe or no lesions n=103</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender (male)</td>
<td>71 (30%)</td>
<td>56 (24%)</td>
<td>1.00</td>
</tr>
<tr>
<td>Age</td>
<td>60±10</td>
<td>52±12</td>
<td>0.0001</td>
</tr>
<tr>
<td>Chest pain</td>
<td>82 (35%)</td>
<td>58 (25%)</td>
<td>0.35</td>
</tr>
<tr>
<td>Angina</td>
<td>57 (24%)</td>
<td>30 (13%)</td>
<td>0.03</td>
</tr>
<tr>
<td>Previous PCI</td>
<td>34 (15%)</td>
<td>14 (6%)</td>
<td>0.02</td>
</tr>
<tr>
<td>Previous CABG</td>
<td>23 (10%)</td>
<td>2 (1%)</td>
<td>0.0001</td>
</tr>
<tr>
<td>Scheduled (versus emergent) dialysis</td>
<td>65 (28%)</td>
<td>64 (27%)</td>
<td>0.08</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>106 (45%)</td>
<td>62 (27%)</td>
<td></td>
</tr>
<tr>
<td>Hemoglobin A1c</td>
<td>6.6±1.7%</td>
<td>6.3±1.8%</td>
<td>0.28</td>
</tr>
<tr>
<td>Hemoglobin A1c&gt;7%</td>
<td>32 (14%)</td>
<td>14 (6%)</td>
<td>0.37</td>
</tr>
<tr>
<td>HDL cholesterol</td>
<td>42±16</td>
<td>43±16</td>
<td>0.58</td>
</tr>
<tr>
<td>LDL cholesterol</td>
<td>71±50</td>
<td>68±40</td>
<td>0.70</td>
</tr>
<tr>
<td>Highest serum troponin level</td>
<td>8±17</td>
<td>7±30</td>
<td>0.74</td>
</tr>
<tr>
<td>Elevated serum troponin I</td>
<td>109 (47%)</td>
<td>70 (30%)</td>
<td>0.005</td>
</tr>
<tr>
<td>Electrocardiographic abnormalities</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-specific T wave abnormalities</td>
<td>73 (31%)</td>
<td>58 (25%)</td>
<td>1.00</td>
</tr>
<tr>
<td>New T wave inversion</td>
<td>36 (15%)</td>
<td>28 (12%)</td>
<td>1.00</td>
</tr>
<tr>
<td>Left ventricular hypertrophy</td>
<td>33 (14%)</td>
<td>27 (12%)</td>
<td>0.56</td>
</tr>
<tr>
<td>ST segment depression</td>
<td>33 (14%)</td>
<td>8 (3%)</td>
<td>0.0004</td>
</tr>
<tr>
<td>New right bundle branch block</td>
<td>10 (4%)</td>
<td>4 (2%)</td>
<td>0.28</td>
</tr>
<tr>
<td>New Q wave</td>
<td>8 (3%)</td>
<td>6 (3%)</td>
<td>1.00</td>
</tr>
<tr>
<td>New left bundle branch block</td>
<td>3 (1%)</td>
<td>4 (2%)</td>
<td>0.70</td>
</tr>
<tr>
<td>Echocardiographic abnormalities *</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wall motion abnormalities</td>
<td>65 (38%)</td>
<td>37 (22%)</td>
<td>0.13</td>
</tr>
<tr>
<td>Left ventricular (LV) hypertrophy</td>
<td>50 (29%)</td>
<td>38 (22%)</td>
<td>0.65</td>
</tr>
<tr>
<td>Drop in LV Ejection fraction</td>
<td>31 (18%)</td>
<td>14 (8%)</td>
<td>0.12</td>
</tr>
<tr>
<td>LV Ejection fraction &lt; 30%</td>
<td>28 (16%)</td>
<td>21 (12%)</td>
<td>0.87</td>
</tr>
</tbody>
</table>

Table 2. Comparison of patients with severe coronary lesions and those with less than severe lesions or normal coronary angiograms.

Legend: (*) done on 170 patients

While the patients with severe coronary lesions were older, the difference of mean age was only of 5 years. Not surprisingly, history of percutaneous coronary intervention (PCI) or coronary-artery bypass graft (CABG) was more common among those with severe disease. Diabetes mellitus, quite prevalent in our cohort, was also more common among patients with severe coronary lesions; however, the hemoglobin A1c levels were similar in both groups. While the levels of serum troponin were more frequently elevated among patients with severe coronary lesions, the levels of serum troponin I were overall comparable. Depression of the ST segment was also more common among those with severe lesions.

The following variables were associated with severe coronary lesions in a multiple regression model: diabetes mellitus ($p = 0.0001$), new ST depression ($p = 0.0092$), elevated serum troponin I level ($p = 0.0104$), and angina.
pectoris ($p = 0.0126$). We excluded from this model: age, history of CABG and history of PCI (this history already defines severe coronary lesions).

Discussion:
In our cohort, diabetes mellitus, new ST depressions on electrocardiogram, elevation of serum troponin I and documented angina pectoris were associated with severe coronary lesions among patients with ESRD.

The prevalence of coronary atherosclerosis among patients with ESRD is higher than the general population, in part due to similar risk factors, such as hypertension and diabetes mellitus, and in part due to elevated levels of fibrinogen, homocysteine, and lipoprotein A.

Diabetes has been described as a coronary artery disease risk equivalent. Seventy-two percent of our cohort had diabetes mellitus. However, only 28% of the diabetics had a hemoglobin A1C of greater than 7%, with no difference between the patients with severe, or not-severe or absent coronary lesions. In patients with ESRD, normalization of hyperglycemia is commonly observed. Malnutrition, protein-energy wasting, diabetic gastroparesis, reduced clearance and degradation of exogenous insulin, and decline in the hepatic clearance of insulin all play a role in this normalization.

Serum troponin elevations have been described among patients with ESRD even in the absence of myocardial ischemia. In a study of 733 patients with asymptomatic ESRD treated with chronic intermittent dialysis, 82% had troponin T levels above the 99th percentile of reference vs. 6% for troponin I. Mounting data are now showing that chronic troponin elevations are strong predictors of all-cause mortality and future cardiovascular events (Troponin T more so than Troponin I). Whether or not patient with ESRD and acute elevations in troponins would benefit from early coronary intervention has not yet been established. However, studies have shown that elevated levels of Troponin T are associated with extensive coronary atherosclerotic disease (CAD). Coronary angiography of sixty-seven volunteer patients with ESRD found that multivessel disease and a CAD index greater than 48 were more prevalent as troponin T levels rose. In our study, any elevation of Troponin I above the reference value was statistically associated with severe coronary lesions.

In patients with large electrolyte shifts and left ventricular hypertrophy, frequent in our study population, the significance of ST depressions can be unclear. In one study, the incidence of ST depressions for at least one minute on Holter monitoring in 67 patients on hemodialysis was 23%. The 2-year mortality of patients with or without transient ST segment depression was the same. We found that ST depressions are associated with severe CAD in our patient population. This difference we observed is likely due to the clinical context in which the patients had a coronary angiogram done. Most of the patients were being evaluated for acute coronary syndrome so the index of suspicion for severe CAD was higher. Therefore, the ST depressions most likely represented subendocardial ischemia rather than transient electrolyte shifts.

The retrospective nature of our study represents its main limitation. The interpretation of chest pain as angina and the indication for coronary angiogram or other interventions were hard to establish. Our clinical question could be applied to patients with pre-dialysis levels of kidney function impairment. However, the use of potentially toxic contrast material, and the risk of promoting further kidney impairment lead most physicians away from this intervention.

In conclusion, among patients with ESRD undergoing dialysis, diabetes mellitus, new ST depressions on electrocardiogram, elevation of serum troponin I levels and angina pectoris were associated with severe coronary lesions on coronary angiography. These findings identify a potential role for these markers to be incorporated into future diagnostic and therapeutic strategies aimed at the earlier detection and management of high risk CAD in the hospital setting, especially when resources are limited.
Affiliations:
All authors are affiliated with the Department of Internal Medicine McGovern Medical School at The University of Texas Health Science Center at Houston, Texas.

References
Hypokalemia in Patients treated with Nafcillin: A Retrospective Cohort Study

Gonzalo Matzumura, MD
Jennifer D. Duke, MD
Adriana Rauseo, MD
John R Foringer, MD
Gabriel M Aisenberg, MD

Introduction:
Nafcillin is a beta-lactam commonly used in the United States for the treatment of infections caused by methicillin susceptible Staphylococcus aureus. Common reactions to nafcillin include hypersensitivity, gastrointestinal upset, and secondary yeast infections. Hypokalemia remains a relatively unrecognized complication. Our objective was to determine the incidence and duration of hypokalemia due to nafcillin use.

Methods:
The study was undertaken at Lyndon B. Johnson Hospital - a tertiary-care, county-based center in Houston, Texas. We collected information from electronic medical records of patients treated with at least one full day of intravenous nafcillin from March 1, 2013, to February 1, 2015. We selected patients with hypokalemia (serum potassium level less than 3.5 mmol/L). Serum potassium levels drawn starting one day after nafcillin therapy initiation until seven days after therapy completion were entered into our database. Patients exposed to diuretics or other drugs known to cause hypokalemia, diseases associated with hypokalemia (diarrhea) or with hypokalemia prior to nafcillin administration were excluded from the study.

From the examined medical records, we retrieved patients’ ages, gender, days of exposure to nafcillin and duration of hypokalemia. Additionally, the dose of nafcillin and patients’ urine studies (including urine electrolytes, urine pH, and urine osmolality) were recorded. Possible alternative causes of hypokalemia were investigated.

MedCalc Statistical Software version 18 (MedCalc Software bvba, Ostend, Belgium; http://www.medcalc.org; 2018) was utilized for the statistical analysis. Categorical variables were analyzed using the Fisher’s exact test, and discrete variables were analyzed using the Student t-test for unpaired samples. A two-sided p<0.05 was considered indicative of statistical significance. A correlation model was used to establish an association between nafcillin exposure and duration of hypokalemia.

Results:
One hundred and sixty-nine patients were treated with at least one full day of intravenous nafcillin on 177 occasions. Six patients were treated twice, and one was treated three times. One hundred and twenty-two (72%) patients were male. Median age was 47 years (range 0 to 89 years). Twenty-five patients were excluded for having hypokalemia prior to initiation or on the day nafcillin therapy initiation. Twelve patients were excluded due to alternative explanations other than nafcillin to justify their hypokalemia including concurrent diuretic use, diarrhea, diabetic ketoacidosis and lithium therapy.

We analyzed 30 patients (17%) with hypokalemia likely attributable to nafcillin. Eighteen (60%) were men, with a median age of 47 years (range 0 to 76 years). The median duration of nafcillin therapy was 4 days (range 1-31 days). Hypokalemia was noted 3.6 days after starting nafcillin therapy (range 1-11 days) and lasted a median of 2 days (range 1-17 days). Dosing regimens of nafcillin were variable, however, most of the patient’s in whom dosing intervals were recorded received a total of 6 grams of nafcillin per day. Seven of the 30 patients that developed hypokalemia had spot urinary potassium measured at the time of hypokalemia, and in each case, the result was a urinary potassium of greater than 20 mmol/L, suggesting renal loss. In one patient, the transtubular potassium gradient also confirmed potassium renal wasting. The correlation between days of exposure to nafcillin and duration of hypokalemia also suggested the antibiotic’s potential causative role (correlation coefficient r=0.4709, p=0.009) (Figure 1).
Discussion:
Hypokalemia in patients receiving nafcillin and other penicillins has been reported in previous cases dating back to the 1970s. The proposed mechanism is thought to be multifactorial but primarily due to increased urinary potassium loss. Theories explaining this effect include nafcillin as a non-re-absorbable anion in the distal tubule favoring potassium excretion, increased sodium delivery to the distal nephron, and antibiotic associated cellular re-distribution of potassium.

Viehman et al. noted similar findings of increased hypokalemia in patients receiving nafcillin with an incidence of 51%. The authors however, included a decrease in potassium level greater than 0.5 mmol/L as part of the definition of hypokalemia and did not consider alternative mechanisms of hypokalemia. Consequently, the incidence of hypokalemia in this series was significantly higher.

In our series, the duration of hypokalemia was relatively short and led to no major complications, explained by early recognition and prompt electrolyte replacement. For this reason, clinicians should closely monitor serum potassium levels in patients treated with nafcillin.

References: