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Cardiac POCUS: Another Tool in the Armory
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University of Kansas School of Medicine–Wichita, Wichita, KS
1Internal Medicine/Pediatrics Residency Program
2Department of Internal Medicine
3University of Kansas Medical Center, Kansas City, KS
Department of Cardiovascular Medicine
Received March 15, 2023; Accepted for publication June 5, 2023; Published online July 25, 2023
https://doi.org/10.17161/kjm.vol16.19802

ABSTRACT
Introduction. This study assessed the educational impact of hybrid cardiac Point of Care Ultrasonography (POCUS) training in a community-based academic setting.

Methods. Internal Medicine and Medicine/Pediatrics residents across all post-graduate years (PGY) at a midwestern medical school undertook a structured hybrid (online and hands-on teaching) model of POCUS training. Anonymous surveys with Likert-type scale responses were administered before and after the curriculum. Questions were categorized into domains to assess the residents’ interest in learning POCUS, their understanding of fundamental cardiac ultrasound (US) concepts, and their confidence in its application. The authors used Fisher’s Exact and t-test, and estimated odds ratios to gauge the impact of the training to achieve net scores above 0 on each domain.

Results. A total of 27 and 26 residents completed the pre- and post-training surveys, respectively. Experience with previous cardiac US use showed a positive skew. The training resulted in a significant increase in both, the understanding of the principles, and the residents’ confidence in its application. These findings were most significant amongst PGY 2 and 3 residents. Post-training mean scores were similar across all domains for subgroups of PGY level and previous ultrasound experience.

Conclusions. Residents displayed greater understanding of the fundamental cardiac ultrasound concepts with improved confidence levels after implementing a structured hybrid teaching model for POCUS. Future studies with objective assessment tools are needed to gauge the clinical impact of POCUS and its adoption rate in clinical practice to guide a recommendation for its incorporation into the residency curriculum.

INTRODUCTION
Point-of-care ultrasound (POCUS) rapidly is gaining importance as a tool in the internists’ arsenal for bedside evaluation. Studies have shown that the addition of POCUS to standard diagnostic pathways yielded greater evidence in making the timely and correct diagnosis, along with improved and prompt administration of treatments in the emergency setting. The role of POCUS in cardiology presents a unique opportunity in improving patient care. In-patient focused cardiac ultrasound shows high diagnostic sensitivity, comparable to a cardiologist-performed ultrasound, in identifying cases such as pleural effusion, signs of right ventricular enlargement, left ventricular systolic dysfunction, ultimately helping guide diagnosis, for example, cardiogenic vs obstructive shock.

With growing evidence supporting the use of POCUS, the American College of Physicians formally announced a statement in 2018 acknowledging the importance of POCUS in the practice of medicine. Other professional groups also have presented guidelines for its use by physicians. In 2014, international evidence-based recommendations for focused cardiac ultrasound use were released to standardize its adoption in clinical practice, as POCUS continued to be incorporated rapidly into the medical school and residency curriculums.

The Department of Internal Medicine at the University of Kansas School of Medicine–Wichita, a community-based academic program, introduced a hybrid POCUS training model of the cardiovascular system for its residents across all post-graduate years in the year 2020. The training was incorporated alongside a quality project to explore the benefits residents perceived from this training on their interest in, understanding, and confidence with cardiac POCUS.

METHODS
Training Model. Internal Medicine and Medicine/Pediatrics residents across all post-graduate years undertook a structured hybrid (online and hands-on teaching) model of POCUS training. Residents were required to complete an online training module (SonoSim®) dedicated to cardiac POCUS the week prior to their in-person training and present their completion certificates on the day of their hands-on training. The online module was comprised of sections dedicated to understanding the fundamentals of cardiac ultrasound, the anatomy of the heart, and the clinical application of cardiac POCUS. For the hands-on training, residents were split into groups of six and seven to practice the application of cardiac POCUS, and asked to save their image findings onto the ultrasound software being used. Hand-held portable ultrasound probes (Butterfly Network, Inc.) were used for hands-on training.

Assessment. Anonymous surveys were conducted with Likert-type scale responses before and after the hands-on training and categorized questions into domains to assess the residents’ interest in learning POCUS, their understanding of fundamental cardiac ultrasound (US) concepts, and their confidence in its application. Additional questions on the surveys queried the curriculum structure, which would guide future alterations to the curriculum. Responses were scaled to each question on a range of -2 to +2, representing responses from “strongly disagree” to “strongly agree”, respectively. A total of one question, two questions, and eleven questions assessed the residents’ “Interest” (range of score (ROS) -2 to +2), “Understanding” (ROS -4 to +4), and “Confidence” (ROS -22 to +22) domains, respectively.

Statistical Analysis. A descriptive analysis was conducted for responses on both the pre-and post-surveys and direct comparisons were made of mean responses using Fisher’s Exact and t-test as appropriate. To gauge the impact of the training, the percentage of responses was assessed on each domain greater than zero post-training.
compared to responses pre-training. Odds ratios (OR) also estimated the impact of training on scores greater than zero with its respective 95% confidence interval (CI). Additional sub-group analysis evaluated the differences in scores post-training between PGY level and previous US experience. All analyses were conducted using R software 4.0.5.

RESULTS

A total of 27 and 26 residents completed the pre- and post-training surveys, respectively. The pre-training survey analysis revealed that 11 residents had some previous US experience, and the number of previous US experiences demonstrated a positive skew. Over 44.4% of residents who participated in the survey were in their second year of training, and 55.6% of respondents were male (Table 1). Table 1 shows demographics of survey participants using data on pre-test surveys. Residents who participated in the survey were in their second year of training, and 55.6% of respondents were male (Table 1). Table 1 shows demographics of survey participants using data on pre-test surveys.

Table 1. Characteristics of participants who completed the pre-training cardiac POCUS survey.

<table>
<thead>
<tr>
<th>Number of Residents, Total n</th>
<th>27</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex, n (%)</td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>11 (40.7)</td>
</tr>
<tr>
<td>Male</td>
<td>15 (55.6)</td>
</tr>
<tr>
<td>No response</td>
<td>1 (3.7)</td>
</tr>
<tr>
<td>PGY, n (%)</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>8 (29.6)</td>
</tr>
<tr>
<td>2</td>
<td>12 (44.4)</td>
</tr>
<tr>
<td>3</td>
<td>5 (18.5)</td>
</tr>
<tr>
<td>No response</td>
<td>2 (7.4)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Previous US Experience</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean (SD)</td>
<td>1.14 (1.46)</td>
</tr>
<tr>
<td>Median</td>
<td>1</td>
</tr>
<tr>
<td>Range</td>
<td>0 - 5</td>
</tr>
<tr>
<td>No previous experience, n (%)</td>
<td>10 (37.0)</td>
</tr>
<tr>
<td>Any previous experience, n (%)</td>
<td>11 (40.7)</td>
</tr>
<tr>
<td>No response for previous experience, n (%)</td>
<td>6 (22.2)</td>
</tr>
</tbody>
</table>

Abbreviations: POCUS = point-of-care ultrasound, n = number of participants, SD = standard deviation.

The overall mean ‘Interest’, ‘Understanding’, and ‘Confidence’ scores increased significantly after the training (Table 2). The percentage of residents with a net score above 0 for ‘Interest’ increased from 66.7% to 84.6% after the training (p = 0.11). The percentage of residents with a net score above 0 increased from 59.3% to 92.3% (p = 0.01) for the ‘Understanding’ domain, and from 63.0% to 88.5% (p = 0.006) for the ‘Confidence’ domain. The odds ratios for these findings are shown in Table 2.

On subgroup analysis, no statistical difference was observed in the mean score for all three domains amongst subgroups of PGY level and previous US experience on the post-training survey (Table 3). After the training, a significant increase in mean interest scores was noted amongst PGY 1. PGY 2 and PGY 3 residents demonstrated a significant increase in mean understanding and confidence scores post-training, while these increases were not significant amongst PGY 1 residents. Residents with and without previous US experience showed a similar increase in scores on all domains after the training. These changes in mean scores post-training by various subgroups is shown in Table 4.

Table 2. Comparing mean scores and odds ratios for net scores above 0 before and after the cardiac POCUS training across the ‘Interest’, ‘Understanding’, and ‘Confidence’ domains.

<table>
<thead>
<tr>
<th>Domain</th>
<th>Pre-Training Mean Score (SD)</th>
<th>Post-Training Mean Score (SD)</th>
<th>p Value</th>
<th>Odds Ratio (95% CI) for net score &gt; 0</th>
</tr>
</thead>
<tbody>
<tr>
<td>Interest*</td>
<td>0.74 (0.71)</td>
<td>1.36 (0.70)</td>
<td>0.003</td>
<td>3.66 (0.86 - 15.39)</td>
</tr>
<tr>
<td>Understanding†</td>
<td>0.56 (1.42)</td>
<td>2.62 (1.20)</td>
<td>&lt; 0.001</td>
<td>8.25 (1.61 - 42.28)</td>
</tr>
<tr>
<td>Confidence‡</td>
<td>0.48 (4.91)</td>
<td>10.50 (6.50)</td>
<td>&lt; 0.001</td>
<td>13.53 (1.58 - 116.04)</td>
</tr>
</tbody>
</table>

*Interest range of score = -2 to +2
†Understanding range of score = -4 to +4
‡Confidence range of score = -22 to +22
Abbreviations: POCUS = point-of-care ultrasound, N = number of participants, SD = standard deviation, CI = confidence interval.

DISCUSSION

These results demonstrated that a hybrid training model for Cardiac POCUS significantly increased the understanding of its concepts and confidence in its use amongst residents in a community-academic setting. These findings were most significant amongst PGY levels 2 and 3. The training model also resulted in post-training scores that were comparable across all sub-groups on all three domains of ‘Interest’, ‘Understanding’, and ‘Confidence’.

With the increasing adoption and awareness of POCUS, interest levels in learning these techniques are anticipated to increase. Among our residents, there was a good interest in learning cardiac POCUS before the training. The training appeared to impact interest scores significantly among residents in the PGY 1 level of training. This may reflect more familiarity with POCUS and related concepts amongst residents already in training (i.e., PGY levels 2 and 3). In addition, the overall confidence and the understanding scores were noted to increase amongst our residents after the training. This was consistent with results in other contexts upon implementing an ultrasound training curriculum. However, the increase in mean confidence and understanding scores amongst PGY 1 level residents was not significant in our
Our program adopted a hybrid teaching model considering busy resident schedules and to encourage self-directed learning. With these results, the hybrid model generally was well-received and proved to be effective. This was consistent with other hybrid training styles adopted amongst first-year residents. Of note, having any previous ultrasound experience or a resident's level of training had no impact on the mean scores post-training. These results demonstrated the model's effectiveness in training residents across various sub-groups.

Our program adopted a hybrid teaching model considering busy resident schedules and to encourage self-directed learning. With these results, the hybrid model generally was well-received and proved to be effective. This was consistent with other hybrid training styles adopted by other institutions. However, there are challenges to the effective implementation of such a model. The feasibility of acquiring readily available POCUS probes should be considered and appropriate funding available. Additionally, faculty physicians may require additional training before implementing such a program for residents, especially since the limited availability of general medicine faculty trained in POCUS is likely to contribute to the slow adoption of POCUS by residency programs. Other challenges may include the availability of documentation templates, electronic storage to archive imaging, and discussing billing and quality assurance policies. Some experts discussed that reimbursement may cover costs associated with POCUS education and maintenance of the equipment. Such implementation is justified since POCUS training may contribute to the evaluation and management of patients by affecting the complexity of decision-making. Future reimbursement methods, known as "bundling" (which overlooks an "episode of care"), may be beneficial for documentation, billing, and image archiving purposes.

There were some limitations to this study. First, not all residents participated in the study, which may have resulted in a potential bias towards those interested in undertaking POCUS training. In addition, this study was limited to a single academic community hospital with internal medicine residents. Further research should be conducted in larger community hospitals compared with training in university hospitals. Moreover, these results indicated resident perspective in the short term, since responses were recorded immediately after the training.

Studies found that the fundamental understanding of POCUS principles declined several months after training, however, long-term retention was improved with a longitudinal ultrasound curriculum. In response, our program plans to make POCUS probes readily available for all in-patient rotations and create a longitudinal curriculum with at least annual training available for all residents. This process should foster a culture of incorporating POCUS in patient care by encouraging POCUS-based presentations by already trained residents during hospital rounds. Future studies with a comprehensive teaching curriculum and more objective assessment tools are needed to gauge its clinical impact and adoption rate in clinical practice. These ultimately will help to formulate formal recommendations for integration of POCUS as a required curriculum during residency training.

CONCLUSIONS

A hybrid-teaching model for cardiac POCUS was an effective curriculum for residents in a community-academic setting. Residents demonstrated an increase in understanding and confidence with cardiac POCUS skills after the training. Post-training scores were similar across subgroups of PGY level and having previous US experience. Thoughtful integration of a cardiac POCUS program among residents provides an opportunity to create a stimulating and clinically impactful environment.

ACKNOWLEDGEMENTS

This study was presented as a poster at the virtual AHA scientific sessions QCOR on November 15, 2021, an abstract for which was published as a supplement: Circulation 2021; 144:A12830.

REFERENCES


<table>
<thead>
<tr>
<th>Domain</th>
<th>Pre-Training</th>
<th>Post Training</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Interest*</td>
<td>PGY 1</td>
<td>0.38 (0.74)</td>
<td>1.38 (0.74)</td>
</tr>
<tr>
<td>PGY 2</td>
<td>0.83 (0.72)</td>
<td>1.11 (0.78)</td>
<td>0.409</td>
</tr>
<tr>
<td>PGY 3</td>
<td>1.00 (0.71)</td>
<td>1.67 (0.52)</td>
<td>0.104</td>
</tr>
<tr>
<td>No previous experience</td>
<td>0.90 (0.74)</td>
<td>1.12 (0.64)</td>
<td>0.506</td>
</tr>
<tr>
<td>Any previous experience</td>
<td>0.50 (0.58)</td>
<td>1.40 (0.89)</td>
<td>0.127</td>
</tr>
<tr>
<td>Understanding†</td>
<td>PGY 1</td>
<td>1.25 (0.89)</td>
<td>2.25 (1.28)</td>
</tr>
<tr>
<td>PGY 2</td>
<td>0.17 (1.11)</td>
<td>2.56 (0.88)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>PGY 3</td>
<td>-0.20 (2.28)</td>
<td>2.86 (1.57)</td>
<td>0.02</td>
</tr>
<tr>
<td>No previous experience</td>
<td>0.10 (1.66)</td>
<td>2.22 (1.20)</td>
<td>0.006</td>
</tr>
<tr>
<td>Any previous experience</td>
<td>-0.25 (1.50)</td>
<td>1.80 (1.10)</td>
<td>0.049</td>
</tr>
<tr>
<td>Confidence‡</td>
<td>PGY 1</td>
<td>-3.38 (4.66)</td>
<td>10.57 (8.30)</td>
</tr>
<tr>
<td>PGY 2</td>
<td>-0.25 (3.89)</td>
<td>8.78 (6.40)</td>
<td>0.001</td>
</tr>
<tr>
<td>PGY 3</td>
<td>-3.00 (6.48)</td>
<td>12.17 (5.78)</td>
<td>0.003</td>
</tr>
<tr>
<td>No previous experience</td>
<td>-1.00 (6.32)</td>
<td>9.88 (8.53)</td>
<td>0.007</td>
</tr>
<tr>
<td>Any previous experience</td>
<td>0.00 (5.16)</td>
<td>9.50 (4.20)</td>
<td>0.029</td>
</tr>
</tbody>
</table>

*Interest range of score = -2 to +2
†Understanding range of score = -4 to +4
‡Confidence range of score = -22 to +22

Abbreviations: POCUS - point-of-care ultrasound, PGY - post-graduate year, SD - standard deviation

Table 4. Comparing mean scores on pre-training and post-training surveys for cardiac POCUS by various subgroups across all domains.


Keywords: cardiovascular system, ultrasonography, point of care system, online learning, internship and residency
A Case of Low Ejection Fraction Unrelated to Anthracycline Therapy: Chemo Tells a Fib

De'ya Alkhatib, M.D.1, Kimberly DeCarr2, Issa Pour- Ghaz, M.D.1, Omar Al-Tawel, M.D.3, Buthainah Alhwarat, M.D.4, Addison Bond2, Tracy Wineinger, MPH5, John Alexander, D.O.1, Sharif Kayali, D.O.1, Neeraja Yellapati, M.D.1, Isaac Rhea, M.D.1

1Division of Cardiovascular Disease
2College of Medicine
3University of Nevada—Las Vegas, Kirk Kerkorian School of Medicine, Las Vegas, NV
4Hashemite University, Faculty of Medicine, Zarqa, Jordan
5Kansas College of Osteopathic Medicine, Wichita, KS

INTRODUCTION

Anthracyclines are effective and widely used chemotherapeutic agents, but their inherent properties confer cardiotoxic risk.1 Echocardiographic changes in left ventricular ejection fraction (LVEF) and global longitudinal strain (GLS) have served as primary surveillance for monitoring impaired cardiac function following anthracycline use.2,3 However, echocardiographic imaging is not always capable of elucidating the underlying mechanism of heart failure (HF) and may affect patient management and outcome in instances where multiple HF risk factors co-exist.

Atrial fibrillation (AF) is another well-known exacerbator of left ventricular (LV) dysfunction, primarily due to tachycardia-induced LV strain and perturbations in diastolic filling. However, the relationship between AF and HF is complex with each contributing to the pathophysiology of the other.4 As such, it can be easy to dismiss AF as a potential cause of HF in patients with more obvious explanations, particularly in patients with rate-controlled AF.

Cardiac magnetic resonance (CMR) imaging techniques such as T1 mapping and late gadolinium enhancement (LGE) have emerged as strategies for detecting substrate changes more specific to anthracycline therapy, including extracellular volume expansion and LV fibrosis.5 In patients with multiple possible causes of HF, CMR may play an essential role in elucidating the underlying mechanism and properly guiding HF management.

This case describes a patient with presumed anthracycline-induced cardiomyopathy with subsequent LVEF recovery following AF and atrial flutter (AFL) ablation and discusses the value of CMR in differentiating anthracycline-induced cardiomyopathy from other non-ischemic causes of HF.

CASE REPORT

A 77-year-old man with a past medical history of hypertension, coronary artery disease (CAD), and diffuse large B-cell lymphoma treated with six cycles of rituximab-cyclophosphamide-hydroxydaunorubicin-oncovin-prednisone (R-CHOP) was referred to cardio-oncology for HF exacerbation, presumed to be secondary to anthracycline therapy. Depressed LVEF (45%) was first detected five months after completion of the R-CHOP regimen during an unrelated hospital stay; LVEF previously was normal at the start of chemotherapy (Figure 1). At the time of diagnosis, the patient was started on guideline-directed medical therapy (GDMT), including carvedilol, candesartan, spironolactone, and torsemide, and repeat coronary angiography revealed non-obstructive CAD. During his inpatient stay, he was diagnosed with new-onset AF and started on amiodarone to restore normal sinus rhythm (NSR).
bpm, approximately 30 points lower than pre-ablation ECGs (Figure 3A).

Figure 2. CMR images demonstrated an absence of LV fibrosis. CMR = cardiac magnetic resonance, LV = left ventricular.

Figure 3. Changes in heart rate (A) and indirect markers of cardiac function (B-D) following catheter ablation for AF/AFL. (A) Pre-ablation ECGs demonstrated AF with controlled ventricular rates; post-ablation ECGs revealed NSR with an average heart rate of 61 bpm. (B) PA diastolic pressure was elevated prior to ablation and subsequently stabilized to an average of 21 mmHg one-month post-ablation. (C) Serum creatinine was elevated pre-ablation and subsequently decreased to normal range within two months of ablation. (D) NT-proBNP was elevated pre-ablation and subsequently decreased post-ablation.

**DISCUSSION**

Heart failure following anthracycline-based chemotherapy is not uncommon, with incidence ranging from 3% to 26% depending on the cumulative dose and duration of follow-up.2 The risk of clinical HF is elevated in patients more than 65 years old with pre-existing cardiovascular conditions, and typically presents within one year of chemotherapy completion.

Advanced cardiac imaging, such as CMR, has emerged as a powerful tool capable of elucidating the underlying mechanism of anthracycline-induced cardiomyopathy (i.e., intra-cardiomyocyte edema and extracellular fibrosis).7 In patients with multiple risk factors for HF, CMR has the potential to differentiate anthracycline-induced cardiomyopathy from other non-ischemic causes of HF, such as AF, and may play an essential role in guiding HF management and improving patient outcomes.8,9

Recent studies have explored the prognostic value of CMR in predicting LVEF improvement following AF ablation in patients with non-ischemic heart failure with reduced ejection fraction (HFrEF).3,10 The absence of LV fibrosis, as detected by LGE and T1 extracellular volume fraction mapping, has been shown to correlate with greater improvement in LV systolic function following AF ablation. As such, CMR may play an additional role in predicting which patients are likely to have a favorable response to catheter ablation, thereby risk-stratifying patients based on the likelihood of clinical improvement and allowing for improved shared decision-making.

Pharmaceutical-based rhythm control has not been shown to improve clinical outcomes in patients with AF and HFrEF.11 However, several recent studies have explored the superiority of ablation-based rhythm restoration. A meta-analysis comparing several randomized-controlled trials revealed significant improvements in LVEF, decreased number of hospitalizations, and reduced mortality in patients receiving catheter ablation for AF compared to those managed with medical therapy alone.12 In patients with HFrEF and rate-controlled AF, an impaired systolic function may be more heavily dependent on diminished ventricular preload and irregular filling times; therefore, restoration of sinus rhythm may be even more critical to improvement in LV systolic function.

**CONCLUSIONS**

Rate-controlled atrial fibrillation is an important and easily overlooked cause of HFrEF in patients with a history of anthracycline use. Ablation-based rhythm restoration may be helpful in AF patients with controlled ventricular rates. Cardiac magnetic resonance imaging can be used to differentiate anthracycline-induced cardiomyopathy from other non-ischemic causes of HF and further can predict LVEF recovery in certain patient populations.

**REFERENCES**


Keywords: heart failure reduced ejection fraction, anthracyclines, left ventricular ejection fraction, atrial fibrillation, cardiac imaging techniques
A Case of Catastrophic Aspergillus Endocarditis

Roshan Bisarya, M.D.1, Kenneth Villareal, D.O.2, Bhanu Gupta, M.D.3, Rachel Weihe, M.D.2
University of Kansas Medical Center, Kansas City, KS
1Department of Internal Medicine
2Department of Infectious Diseases
3Department of Cardiovascular Medicine

Received Feb. 22, 2023; Accepted for publication May 22, 2023; Published online July 25, 2023
https://doi.org/10.17161/kjm.vol16.19546

INTRODUCTION

Aspergillus endocarditis accounts for less than 5% of infective endocarditis cases, but it has a high mortality and diagnosis is often elusive.1,2 Previous case reviews have suggested the mortality rate of Aspergillus endocarditis to be between 62-68%, with the most common species identified as Aspergillus fumigatus.2-4 Those populations often affected are patients who are immunosuppressed, on long-term antibiotic therapy, or have prosthetic heart valves.5,6 Previous valvular surgery has been reported in 35-50% of patients with Aspergillus endocarditis.2,7 Additionally, blood cultures are rarely positive for Aspergillus species, making the diagnosis difficult.2,8 Complications of Aspergillus endocarditis include peripheral emboli, mycotic abscesses, endophthalmitis, and aneurysms.2,7

A case of Aspergillus prosthetic valve endocarditis with a timely diagnosis is presented, but the patient was not a surgical candidate and had complications with progression of his infection despite a prolonged course of antifungal therapy.

CASE REPORT

A 69-year-old male with a past medical history of hyperlipidemia, hypertension, and bioprosthetic mitral valve replacement (one year prior to presentation) presented as a transfer to our institution with a left parietal hemorrhage after having been admitted two weeks prior for a left middle cerebral artery stroke treated with thrombectomy. He was sedated, intubated, and underwent placement of an external ventricular drain. Per the family, the patient was doing well since the first stroke. He had reported mild fatigue and a headache the night prior to presentation. He was on warfarin, aspirin, and rosuvastatin at home. He worked as an attorney in a rural town in Kansas, raised horses, and had no recent history of travel.

Initial physical exam was significant for a III/IV holosystolic murmur at the apex, but he did not have any other peripheral stigmata of endocarditis. Transesophageal echocardiogram (TEE) during stroke workup demonstrated three mobile echodensities on the posterior mitral valve leaflet: the largest measuring 6 mm x 18 mm (Figure 1). These mobile echodensities were not present on the transthoracic echocardiogram (TTE) completed during the previous hospitalization. He originally was started empirically on cefepime (2 g IV every eight hours), gentamicin (80 mg IV every eight hours), and vancomycin (1250 mg IV twice daily). Lab work revealed an Aspergillus galactomannan antigen of 0.899 (normal < 0.500) and 1,3-β-D-glucan greater than 500 pg/mL (normal < 80 pg/mL). The Karius® cell-free DNA plasma quantitative test returned positive for Aspergillus fumigatus. Liposomal amphotericin B (3 mg/kg IV every 24 hours) and voriconazole (360 mg per oral twice daily) were added to his antimicrobial regimen. On day six of hospitalization, his fungal blood cultures grew Aspergillus fumigatus, susceptible to amphotericin, micafungin, isavuconazole, and voriconazole. He developed right eye fungal endophthalmitis, which was treated with intravitreal voriconazole (100 mcg). After discussion with the cardiothoracic surgery service, valve repair surgery was deferred at the time due to his recent hemorrhagic stroke preventing use of anticoagulation. The plan was to complete a six to eight week course of combined antifungal therapy and re-evaluate.

On day six of hospitalization, his fungal blood cultures grew Aspergillus fumigatus, susceptible to amphotericin, micafungin, isavuconazole, and voriconazole. He developed right eye fungal endophthalmitis, which was treated with intravitreal voriconazole (100 mcg). After discussion with the cardiothoracic surgery service, valve repair surgery was deferred at the time due to his recent hemorrhagic stroke preventing use of anticoagulation. The plan was to complete a six to eight week course of combined antifungal therapy and re-evaluate.

After 21 days of treatment with liposomal amphotericin B and voriconazole, the patient was switched from voriconazole to isavuconazole (372 mg daily) due to a prolonged QTc (0.55 sec). Isavuconazole levels were confirmed to be therapeutic. On day 33 of admission, he was discharged to inpatient rehabilitation. He then transitioned to a skilled nursing facility. After 50 days of treatment, amphotericin B was switched to micafungin (150 mg IV daily) due to the patient developing renal insufficiency (creatinine increased by 0.34 mg/dL). The plan was to continue micafungin and isavuconazole until an appointment with the cardiothoracic surgery service three weeks later to discuss valve replacement.

Two weeks after the switch to micafungin, the patient returned to the emergency department with gradually worsening generalized weakness and failure to thrive. He also had a white blood cell count elevation of 14.3 K/UL. Computed tomography (CT) scan demonstrated splenic infarcts with associated splenic abscess and wedge-shaped renal infarcts likely from septic emboli (Figure 2).

Figure 1. 2D Transesophageal echocardiogram showed a large mobile echodensity (red arrow) attached to the posterior leaflet of the bioprosthetic mitral valve (green arrow).

Figure 2. D Transesophageal echocardiogram showed a large mobile echodensity (red arrow) attached to the posterior leaflet of the bioprosthetic mitral valve (green arrow).
The patient was continued on micafungin and isavuconazole. Vancomycin (1250 mg IV) and cefepime (2 g IV every eight hours) were added. The abscesses were not amenable to drainage per interventional radiology. The next day he became hypotensive requiring vasopressor initiation and was transferred to the Intensive Care Unit. A repeat TTE demonstrated a persistent mobile echodensity attached to the mitral valve leaflets. Serum 1,3-β-D-glucan was greater than 500 pg/mL and galactomannan antigen was 5.863. The cardiothoracic surgeon met with the family, and they decided to not pursue surgical intervention. His hospital course was complicated by acute embolic occlusions in the lower extremities treated with thrombectomy, worsening hypoxemia requiring intubation and mechanical ventilation, and increased vasopressor requirements. On day seven of that admission, he was transitioned to comfort measures and died shortly after that.

**DISCUSSION**

This patient represented a case of severe *Aspergillus* endocarditis that ultimately led to his death despite completing a 10-week course of appropriate combined antifungal therapy. The Infectious Disease Society of America (IDSA) guidelines for *Aspergillus* endocarditis recommend early surgical intervention with antifungal therapy (voriconazole or liposomal amphotericin B) followed by lifelong suppressive antifungal therapy. However, there is only low-moderate quality of evidence for this recommendation, and many patients with this disease are not appropriate candidates for high-risk surgery.1

One of the unique aspects of this case was the use of Karius® cell-free DNA technology to identify the organism in addition to a fungal culture growing *Aspergillus fumigatus*. The Karius® test detects microbial cell-free DNA (mcfDNA) circulating in the bloodstream from 1,250 clinically relevant organisms. This microbial cell-free DNA technology may be a novel way to diagnose fungemia given the high frequency of negative blood cultures. Reviews have found blood cultures resulting as negative in at least 96% of *Aspergillus* endocarditis cases.3 A previously published analytical and clinical validation study for the Karius® test showed a 93.7% agreement with blood culture in a cohort of 350 patients.4 In other examples, authors of two case reports used the test to diagnose central nervous system aspergillosis and pulmonary aspergillosis, but neither had infective endocarditis.5,6 As shown in our patient, the Karius® test was a useful tool for diagnosis, but the clinical utility of the test was somewhat disputed. A recent retrospective study explored the diagnostic benefit of the test in clinical settings and demonstrated overall limited real-world clinical impact.7 For now, the gold standard for diagnosis of *Aspergillus* endocarditis remains histological with tissue culture confirmation. Fortunately, our patient also had a positive blood culture for *Aspergillus fumigatus*, which helped to guide therapy with our ability to obtain antifungal susceptibilities.

As far as treatment for *Aspergillus* endocarditis, early surgical intervention is considered crucial particularly in cases with prosthetic valve endocarditis.8 In our review of current literature, only four patients with *Aspergillus* endocarditis have survived without surgical intervention, and three of them were infants or children.9,10 Higher mortality rates exist for immunosuppressed patients and for patients with mitral valve involvement.2 However, it was important to remember that like our patient, many patients initially are not surgical candidates and medical treatment in the form of antifungal therapy is the only option. Most recent literature suggested the use of combined antifungal therapy, but even with this mortality remained high.3 Additionally, long-term use of antifungal agents is challenging due to the various toxicities.

Unfortunately, it is not always possible to complete early surgical intervention for patients with *Aspergillus* endocarditis as the guidelines suggest.3 Like our patient, most patients also develop complications, such as emboli, later in the course of the disease, further complicating the ability to perform surgery.2 This case illustrated that prolonged combined antifungal therapy alone for *Aspergillus* endocarditis was ineffective, and it was important to complete valve replacement surgery at some point during the treatment process to optimize the chance of survival. Newer diagnostic modalities, such as the use of cell-free DNA technology, can assist in making the diagnosis. Further research, ideally with multi-center involvement, is needed to optimize our approach to antifungal therapy regimens and duration of treatment in *Aspergillus* endocarditis, particularly in cases where surgical debridement is not feasible or able to be performed in a timely manner.

**REFERENCES**


Keywords: endocarditis, Aspergillus fumigatus, cell free DNA, case report
Management of Labile Blood Pressure due to COVID-19 Infection and Radiation Induced Baroreceptor Dysfunction

Timothy Nguyen, D.O.,1 Stephen S. Wanjala, M.D.,1, Mona B. Brake, M.D., FACN1,2
1University of Kansas School of Medicine-Wichita, Wichita, KS
2Robert J. Dole Veterans Affairs Medical Center, Wichita, KS

INTRODUCTION

The baroreceptor reflex is responsible for beat-to-beat blood pressure (BP) regulation and its failure results in different clinical syndromes that manifest with blood pressure dysregulation.1 Baroreceptor dysfunction can be caused by neck irradiation, neck surgery, repeated neck trauma, and more recently COVID-19.2 In patients with hypertension, baroreceptor dysfunction can lead to extremely labile blood pressures that are difficult to manage.3

Head and neck cancers account for more than 900,000 yearly cases of cancer.4 From this population, 4% develop lower cranial nerve deficits from radiotherapy that results in baroreceptor failure. More recently, autonomic dysfunction, including that of the baroreceptor reflex, has been identified as part of the post-COVID-19 syndrome.5,6 Cases of both new autonomic dysfunction and worsening of existing dysfunction have been described.2,6

A case of labile blood pressure due to damage to their baroreceptors by radiation is presented, which became significantly worsened after COVID-19 infection. Within this case, the challenges and approach to manage such uncontrolled blood pressures were discussed.

CASE REPORT

A 76-year-old male veteran with a history of hypertension, basal cell carcinoma with radiation therapy to the neck in 2015, orthostatic hypotension (OH), congestive heart failure, coronary artery disease, type 2 diabetes, chronic obstructive pulmonary disease, gastroesophageal reflux disease, and recent COVID-19 infection presented with complaints of severe “charley horse-like” pain in his chest. He reported that his blood pressures over the last month have fluctuated rapidly from greater than 220 mmHg to below 100 mmHg systolic causing him to have significant orthostatic hypotension. On review of medical records, he had three emergency department (ED) visits during the prior month with similar complaints and findings. The only significant change in his medical history was a COVID-19 infection one month prior to this admission. The patient stated his blood pressure was manageable prior to his COVID-19 infection, averaging 100-130 mmHg systolic. Review of his home medications revealed a blood pressure control regimen of: lisinopril 20 mg BID, metoprolol tartrate 50 mg BID, and isosorbide mononitrate 30 mg daily.

In the ED, the patient had a sitting blood pressure of 226/109 mmHg, but his other vital signs were normal and physical examination was unrevealing. Complete metabolic panel and complete blood count were unremarkable. He had elevated troponins of 0.061 ng/ml (normal range of 0-0.033 ng/ml) and his electrocardiogram was unremarkable.

In response to his severely elevated blood pressure, the patient received IV hydralazine 10 mg which adequately lowered his blood pressures. He was admitted for hypertensive emergency given his chest pain, elevated troponins, and elevated blood pressure. The cardiology service was consulted for his elevated troponins and their final disposition attributed the troponins to a type II myocardial infarction secondary to his severely elevated blood pressures.

Upon admission, the patient’s home regimen of metoprolol tartrate 50 mg BID was continued, lisinopril was changed from 20 mg BID to 10 mg TID, hydralazine 10 mg was added as needed for standing systolic blood pressures greater than 160 two hours after lisinopril, and isosorbide mononitrate 30 mg was discontinued. During this hospital course, his fluctuating standing blood pressure measurements had no correlation to time of day or any other triggers. Within the one to two days of admission, the patient’s standing systolic blood pressures ranged from 97-214 mmHg (Figure 1). His blood pressures remained difficult to control and, in response, clonidine 0.1 mg was added. For one day only, his standing blood pressures were controlled tightly between 96-125 mmHg (Figure 1).

Figure 1. Blood pressure measurements and interventions were charted throughout the hospital course.

The patient experienced symptoms of lightheadedness and dizziness when his systolic blood pressure dropped below 100 mmHg. During this hospital stay, he had extensive work up for secondary causes of hypertension. His workup for cardiac dysfunction with an echocardiogram, adrenal insufficiency and Cushing Syndrome with 24-hour cortisol test, phaeochromocytoma with metanephrines and catecholamines, and renal artery stenosis with renal ultrasound were all unremarkable (Table 1). Nephrology/hypertension specialists were consulted, and they discontinued clonidine, started amlodipine 5 mg, recommended the patient to elevate the head of the bed at 30 degrees, started physical therapy for strengthening exercises, and gave the patient compression stockings. With these recommendations, the patient’s standing blood pressures over the course of two days, never peaked more than 160 mmHg systolic and never dropped lower than 107 mmHg systolic (Figure 1).
Table 1. Key patient characteristics, laboratory, and imaging findings during the hospital course.

<table>
<thead>
<tr>
<th>Description</th>
<th>Result</th>
<th>Normal Ranges</th>
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<tr>
<td><strong>Anthropometrics</strong></td>
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<tr>
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<tr>
<td>Height</td>
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<td>Body Mass Index</td>
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<td><strong>Relevant Laboratory Tests</strong></td>
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<tr>
<td>Creatinine</td>
<td>0.94 mg/dL</td>
<td>0.7-1.3 mg/dL</td>
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<td>Plasma free metanephrine</td>
<td>30 pg/mL</td>
<td>≤ 57 pg/mL</td>
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<td>Plasma Normetanephrine fraction</td>
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<td>Plasma metanephrine total</td>
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<td>≤ 205 pg/mL</td>
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<tr>
<td>Troponin peak</td>
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<td>(0-0.1ng/mL)</td>
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<tr>
<td>BNP</td>
<td>142 pg/mL</td>
<td>(HF likely, &gt; 400pg/mL)</td>
</tr>
<tr>
<td>TSH</td>
<td>3.74 IU/mL</td>
<td>(0.5-5.0 IU/mL)</td>
</tr>
<tr>
<td>AM Cortisol</td>
<td>3.9 ug/dL</td>
<td>(3.7-19.4 ug/dL)</td>
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<tr>
<td>Aldosterone (supine AM)</td>
<td>9 ng/dL</td>
<td>Supine: (3-16 ng/dL)</td>
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<td>Renal ultrasound</td>
<td>No sonographic evidence of significant renal artery stenosis</td>
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<tr>
<td>Echocardiogram</td>
<td>LVEF: 50%</td>
<td>Trace TR, MR, AI</td>
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</table>

Abbreviations: BNP-Brain Natriuretic Peptide, HF-Heart Failure, TSH-Thyroid Stimulating Hormone, AM-Morning, LVEF-Left Ventricular Ejection Fraction, TR-Tricuspid Regurgitation, MR-Mitral Regurgitation, AI-Aortic Insufficiency.

In a three-month follow-up with the cardiology service, they recorded his average blood pressure at 136/72 mmHg, with systolic blood pressure highs of 160’s and lows of 100’s. The patient closely adhered to the medication regimen, went to all his physical therapy appointments, and had little to no symptoms of OH.

**DISCUSSION**

Management of labile blood pressure due to baroreceptor failure remains difficult. It is challenging to establish a floor and ceiling for these patients as their blood pressure remains chronically volatile. Baroreflex nerve lesions can cause drastic changes in systolic blood pressures that can exceed 300 mmHg. This dangerous volatility was demonstrated in our patient with systolic blood pressures that ranged from the 90s to the 230s mmHg within a span of 20 minutes (Table 1). This established the difficulty of controlling a patient’s blood pressure in a range that was safe and allowed the patient to be asymptomatic. For this patient, a specific regimen of pharmacologic and nonpharmacologic treatments showed promising control of this labile blood pressure.

For pharmacological management, clonidine was discontinued as this patient began to have significant drops in his blood pressure. Some patients with baroreflex failure can be treated effectively with clonidine. Clonidine was compared against phenoxybenzamine and showed that clonidine was effective at reducing both the frequency and severity of hypertensive and tachycardic surges. For our patient, clonidine was effective at controlling his hypertensive episodes, but exacerbated his hypotensive episodes. Antihypertensives such as clonidine could cause precipitous drops in blood pressures and as a result, it was not an appropriate choice for this patient.

Our patient was started on amlodipine 5 mg QD with goals of titrating upwards. Amlodipine was the antihypertensive choice because the medication could control his elevated blood pressure and did not drop this patient’s blood pressure. Rivasi et al. showed that calcium channel blockers (CCBs) can have a protective effect against orthostatic blood pressure in older people. The study explained that a dihydropyridine CCB frequently can induce a compensatory increase in heart rate, which serves as a counteraction against a drop in blood pressure. Furthermore, CCBs have little to no association with causing orthostatic hypotension.

Lisinopril 10 mg TID was used to control his blood pressure in our patient. Angiotensin-converting-enzyme inhibitors (ACEi) and angiotensin II receptor (ARBs) blockers were not associated with OH and may have protective effects, which are attributed by the improvement of baroreceptor sensitivity and vascular compliance.

For nonpharmacological management, the patient should have his bed elevated at least 30 degrees. Elevating the head of the bed in treatment of labile blood pressure decreases nocturnal hypertension and nocturnal diuresis. The mechanism behind this demonstrated that fluctuations in blood pressure due to lying down may affect the body’s regulation of arginine vasopressin, an anti-diuretic hormone, which, in turn, can affect nocturnal blood pressure and diuresis. The increase in diuresis could affect further how the body regulates our blood pressure.

Physical and occupational therapy were recommended for strengthening exercises and can control this patient’s OH. Deconditioning from lack of exercise worsens OH. To combat deconditioning, physical therapy was a good choice to promote safe and supervised physical activity and mild physical exercise can improve orthostatic tolerance by decreasing venous pooling.

Our patient’s blood pressure variations during admissions prior to and after he suffered from COVID-19 revealed increased lability in his blood pressures after the infection. He received radiation to his neck for basal cell carcinoma five years prior to this presentation which may have resulted in baseline autonomic dysfunction. His baroreceptor dysfunction was worsened by COVID-19 based on the temporal associations. Regular and frequent follow-ups with his primary care physician and nephrologist for monitoring and adjusting of medications were recommended because of the lack of literature on the natural history of COVID-19 induced baroreceptor dysfunction. Improvements in baroreceptor function over time could necessitate treatment adjustments.
Recommendations for patients struggling with labile blood pressures due to autonomic dysfunction are to approach their management with an individualized, multifaceted strategy. For our patient, it proved important to individualize blood pressure control targets based on symptoms, response to therapy, and empiric ceilings to minimize end organ damage. Drugs were avoided that were associated more frequently with orthostasis like clonidine and antihypertensives were chosen that were associated less frequently with orthostasis such as CCB, ACEi, and ARBs. Non-pharmacologic interventions were included such as elevation of the head of the bed and physical therapy to decrease changes in orthostatic blood pressure and deconditioning.

It was difficult to control this patient’s blood pressure due to its unpredictable and fluctuating nature. There were significant limitations in antihypertensives that we could choose because his blood pressure would be consistently in the normal range. This regimen of pharmacologic and nonpharmacologic interventions allowed this patient to maintain his blood pressure with a ceiling that was safe and a foundation that prevented symptomatic OH.

**REFERENCES**


**Keywords:** hypertension, orthostatic hypotension, baroreceptor reflex, COVID-19
Unique Etiology of Trigeminal Neuralgia After Acute Ischemic Stroke

James L. Walker, M.D.1, Jared McLaughlin, D.O.1, John Dickerson, M.D.2, Sukruta S. Pradhan, M.D.1, Felicia A. Newton, Ph.D.1
1University of Kansas School of Medicine—Wichita, Wichita, KS
Department of Anesthesiology
2Kansas Spine and Specialty Hospital, Wichita, KS

INTRODUCTION

Trigeminal neuralgia (TGN) is a common neuropathic pain syndrome with several primary and secondary causes. Classical TGN (CTGN) and Symptomatic TGN (STGN) both have been described in the literature, but not as coexistent causes. CTGN occurs due to blood vessel compression of the trigeminal nerve, and magnetic resonance imaging (MRI) or surgical visualization of blood vessel compression and nerve atrophy are needed for confirmation.1 STGN follows the same diagnostic criteria, but has a radiographic cause other than blood vessel compression.2 Our case suggested contributions from both, including a unique etiology for the development of CTGN via arteriogenesis after acute ischemic stroke that may require surgical intervention. Written, informed consent was obtained from the patient for publication of this case report.

CASE REPORT

A 68-year-old male presented with complaints of dizziness and right upper extremity (RUE) weakness. Exam revealed RUE ataxia, nystagmus, and dysarthria. Aortic arch and 4-vessel cerebral angiogram revealed critical right vertebral artery (VA) and posterior inferior cerebellar artery (PICA) stenoses with right VA dissection and thrombus causing a suspected right lateral medullary infarct and Wallenberg syndrome, which typically consists of contralateral upper extremity hypoesthesia to pain and temperature, hoarseness, dysphagia, nystagmus, vertigo, and cerebellar symptoms. It can cause loss of ipsilateral facial pain and temperature sensation.3

In our patient, stroke symptoms evolved with development of aphonia, singultus, and dysphagia, and onset of ipsilateral facial pain with hypoesthesia to temperature four days after admission. MRI showed interval conspicuity of dorsolateral medullary infarct. All symptoms improved prior to discharge.

Two weeks post-stroke, the patient began to have right sided “tooth” pain that was treated over the next several months with antibiotics, dental work, and pregabalin without relief. One year after his stroke, he was evaluated by neurosurgery for unrelenting, excruciating right facial pain primarily in the trigeminal nerve (CN-V) distribution, specifically the CN-V1/V2 distribution. Suspecting CTGN, an MRI was obtained showing a vascular loop contacting the right trigeminal nerve. Adhesiolysis which resulted in near complete resolution of the pain for several months. Follow-up nearly three years after revealed recurrence of moderate trigeminal nerve pain in the CN-V2/V3 distribution, controlled with acupuncture and other noninvasive modalities.

DISCUSSION

Trigeminal neuralgia (classical or symptomatic) is a common neuropathic pain syndrome affecting 10,000-15,000 new patients every year in the U.S.4 CTGN occurs due to blood vessel compression. The most common cause of STGN is multiple sclerosis, but tumors, arteriovenous, and skull base malformations also may play a role.1 A rarely reported cause of STGN is brainstem infarction, specifically lateral medullary infarction, which results in damage to the spinothalamic tract, nucleus ambiguous, trigeminal tract, vestibular nucleus and/or the inferior cerebellar peduncle, causing Wallenberg syndrome.2 There are reports of TGN-like pain after dorsolateral medullary stroke that occurs in patients who initially had a loss of facial pain sensation.5,8

Ischemic stroke often leads to the development of collateral circulation via arteriogenesis induced by shear stress and growth factors released in an ischemic environment. It can take days to weeks for the collateral vessel to reach its final diameter, which often is associated with an increase in tortuosity and length.9 Arteriogenesis may be an etiology for CTGN after brainstem ischemic stroke if such vessel engorgement causes trigeminal nerve compression. TGN caused by neurovascular compression likely is due to pulsations causing microtrauma to the nerve, in turn leading to demyelination and remyelination that affects action potential transmission. The most vulnerable area of the nerve to this type of trauma is the nerve root entry zone.10 In our case, the surgeon specifically mentioned the vessel contacting the nerve root entry zone in the operative note.

Our case was unique in that it supported potentially coexistent causes of TGN. Since this patient had no symptoms of TGN prior to his ischemic stroke, one simply could attribute this to STGN, as symptoms started two to three months post-stroke. Indeed, MRI confirmation of a dorsolateral medullary infarction and physical exam findings consistent with Wallenberg syndrome suggested a symptomatic etiology. However, because two to three months also would mirror the timeframe over which collateral circulation develops, CTGN due to post-ischemic arteriogenesis compressing the previously unaffected nerve also must be considered. Surgical confirmation of the classical etiology was evidenced by nerve atrophy and compression by the lateral petrosal vein and a branch or loop of the superior cerebellar artery that had to be freed from the nerve root entry zone.

A confounding aspect of this case was that TGN returned several weeks after the first surgery despite initial relief. Following the second surgery for adhesiolysis, the patient had significant relief from right-sided facial pain and reported only mild right periorbital hyperesthesia without other sensory loss. The persistent pain, which was unaffected by either surgery, was characteristic of STGN, likely secondary to lateral medullary infarction. CN-V has three divisions, each with its...
own sensory and motor distribution. STGN typically affects the area distributed by the first and second divisions of CN-V, whereas CTGN typically affects the area distributed by the second and third divisions of CN-V.1,2

The likelihood of facial pain is determined by the location of the infarct. Lesions to the dorsolateral medulla (as in our patient) lead to hypoesthesia and pain on the side of the lesion due to the involvement of the trigeminal descending tract and the trigeminal spinal nucleus.2 Our patient first developed hypoesthesia to the right face four days after admission. According to Fitzek et al.,3 50% of patients with lateral medullary strokes who initially had such hypoesthesia to temperature and pain of the ipsilateral face, developed TGN-like facial pain within 12 days to 24 months. For our patient, Wallenberg syndrome was the suspected culprit behind his persistent pain in the CN-V1 distribution. Pontine descending tractotomy might be an option for treatment of this residual pain component.10

This case report demonstrated a previously undescribed etiology of CTGN from arteriogenesis after VA dissection with resultant critical VA and PICA stenoses and dorsolateral medullary infarction. The proliferation and maturation of collateral circulation is well described after infarction, but collateral vessel engorgement leading to compression of the nerve root of the trigeminal nerve is an undescribed observation. Confounding this diagnosis was the more commonly referenced (though still rare) development of STGN after dorsolateral medullary infarction due to damage to several nerve tracts and nuclei in that region. Coexistent etiologies remain a distinct possibility in our case based on the intraoperative findings that clearly suggested CTGN. It is important to note this patient had no evidence of TGN prior to his stroke.

This case suggested dual causation for the development of TGN, with both classical and symptomatic components. The surgical appearance of the trigeminal nerve with atrophy secondary to vascular compression and symptomatic improvement post-surgery was evidence confirming CTGN. In addition, STGN was supported by previous case reports detailing TGN-like pain following lateral medullary stroke and by the persistent mild hyperesthesia in the CN-V1 distribution despite microvascular decompression. Furthermore, the suggested etiology of CTGN due to arteriogenesis after ischemic stroke in the vertebrobasilar circulation has not been described in the literature. We concluded that diagnosis of TGN occurring after lateral medullary infarction warrants workup to exclude a surgically correctable cause, namely the development of collateral circulation via arteriogenesis leading to CTGN.

REFERENCES

Keywords: trigeminal neuralgia, acute ischemic stroke, Wallenberg syndrome, case report
INTRODUCTION
Melanoma is a malignant cutaneous tumor that accounts for nearly 2% of cancer deaths worldwide. Acral melanoma is a rare melanoma subtype occurring on the palms, soles, and nail units, and nodular melanomas are characterized by prominent vertical invasion. Although the role that trauma plays in the pathogenesis of melanoma is controversial, studies have demonstrated an association between acral melanomas with mechanical or physical stress. We present a case of a patient who developed acral nodular melanoma (ANM) on his palm at a site where a history of local trauma directly preceded lesion formation. The situation was complicated by a lack of healthcare insurance, which was a significant barrier to receiving treatment.

CASE REPORT
A 54-year-old male presented to the dermatology clinic with a darkly pigmented nodular growth on his right palm measuring 1.2 x 0.8 x 0.4 cm (Figure 1). The patient built outdoor fencing for over 30 years and would use his right palm as a mallet to attach metal fence panels at their connective latches. Bruising was noticed in the area of trauma 10 years prior to presentation, which began to increase in size and cause significant pain over the course of three months. He was uninsured and presented to a federally qualified health center (FQHC), which provided healthcare services to medically underserved communities regardless of insurance status.

The patient had no family history of melanoma or other skin cancers. A shave biopsy was performed with gross findings displaying a crusted red-brown lesion measuring 1.2 x 0.8 x 0.4 cm. Histopathologic findings were consistent with malignant melanoma, acral, nodular type with areas of tissue necrosis and overlying ulceration. Neoplasms thickness was at least Clark’s level III, at least Breslow’s thickness 3.70 mm, and pTNM staging of at least pT3b. However, the melanoma involved all the biopsy margins, so an accurate Clark, Breslow, and pTNM staging could not be obtained. S-100, SOX10, and HMB-45 immunohistochemical stains performed were all positive, confirming the diagnosis.

Figure 1. A darkly pigmented nodular growth on the patient’s right palm.
evidence to recommend routine skin cancer screening in the adult general population by primary care physicians, the recent studies have highlighted that skin cancer screening may have the potential to reduce morbidity and mortality from melanoma. Furthermore, certain populations had high rates of advanced-stage melanoma, including white middle-aged and older men and individuals with low socioeconomic status, which was evident in our case. The USPSTF deemed further research on screening high-risk groups necessary to elucidate the benefit of skin cancer screening among these populations. Before implementing population-based skin cancer screening in high-risk groups, evidence from randomized control trials demonstrating that benefits outweigh harms is necessary.

In summary, the current recommendations regarding skin cancer screening as presented by the USPSTF and studies providing insight into the impact of skin cancer screening on mortality rates that have arisen in light of the current recommendations were highlighted. Taking into account that our patient would have been considered high-risk for advanced melanoma and that he was not aware of the seriousness of his lesion, thus presenting late for dermatologic consultation, the current case underscored the need for randomized control trials which demonstrate the impact of routine skin cancer screening among high-risk groups to determine if early detection reduces mortality in these populations.

REFERENCES


Keywords: melanoma, skin cancer