

# Tennessee ACP awards

October, 2020

# The Laureate Award

- The Laureate award is the most prestigious award given at the local level. It honors those fellows or master of the College who have demonstrated, by their example and conduct, an abiding commitment to excellence in medical care, educations, and research, service to their community, their chapter and the ACP

- Dr. Ryan Mire



# Volunteerism and Community Service Award

- This award honors members who have distinguished themselves in voluntary service in the area of medicine. Volunteerism and community service is an established tradition for the College and for internists. The College considers volunteerism so important that it is a major criterion for advancing to Fellowship.

- Dr. Trey Harrell



# Joseph Frederick Ralston Jr. Medical Student Scholarship Award

- In memory of former TN ACP governor and national ACP president, who spent his career supporting and advocating for primary care internal medicine. He exemplified all that IM represents. This award will be presented to a 3<sup>rd</sup> or 4<sup>th</sup> year medical student from a Tennessee medical school, who plans a career in primary care IM.

Jim Ralston



Willis Ralston



# Chapter Service Award

- This award is to honor a member or fellow who has gone above and beyond in service to the chapter over many years. Their efforts and accomplishments have helped to sustain the chapter in all of its endeavors.
- Dr. Maria Tudor



# Resident Recognition Award for Leadership

- This award recognizes a resident with qualities that exemplify the College's mission to enhance the quality and effectiveness of health care by fostering excellence and professionalism in the practice of medicine, and who has made exemplary contributions to the College's mission on a local or national level.
- Dr. David Jones





# Woman Physician of the Year

- This award honors an outstanding woman physician with a distinguished career in areas of exceptional patient care, medical education and/or research.

- Dr. Mukta Panda



# Distinguished Teacher/Mentor Award

- This award is given to a member or fellow who has demonstrated outstanding mentorship of students, residents or colleagues as demonstrated by a sustained commitment to providing personal and professional guidance to other health care professionala.
- Dr. Paul McNabb





# Poster Awards

- Resident/Fellow Clinical Vignettes
- Resident/Fellow Research/QI
- Student First Place

# Resident/Fellow Clinical Vignettes

- Benjamin Emery, MD
- UT Chattanooga
- Getting a Handle on the Situation: An Uncommon Cause For A Common Presentation

## Getting a Handle on the Situation: An Uncommon Cause for a Common Presentation

Benjamin Emery MD MPH, Elizabeth Allyn Glover MS3, Juan Carlos Malpartida MD, Mukta Panda MD  
*Department of Internal Medicine, University of Tennessee College of Medicine – Chattanooga*

### Objectives:

1. Learn the presentation of “syndrome of Headache and transient Neurological Deficits with cerebrospinal fluid Lymphocytosis” (HaNDL)
2. Outline an approach to differentiating HaNDL from other similar diagnoses
3. Understand the importance of obtaining a detailed history in patients presenting with headache or neurologic deficits to avoid unnecessary treatment

### Case Presentation

Our patient was a 27-year-old female with PMH of frequent otitis media and possible Hashimoto's who presented with **1 week of fluctuating, progressive frontal headache, nausea, vomiting, an episode of peripheral vision loss, and increasing somnolence**. She reports a recent tick bite, enjoys rose gardening, is not sexually active and has no prior STIs.

**One year prior** was admitted with similar presentation.

- Severe frontal headache, photophobia, mild nuchal rigidity, and confusion after influenza virus upper respiratory illness
- LP showed WBC 953 with 99% lymphs, glucose 65, protein 99.
- Diagnosed with viral meningitis. Symptoms resolved in two weeks.

#### PHYSICAL EXAM

**Vitals:** hemodynamically stable

**General:** uncomfortable but non-toxic

**HEENT:** left-sided hearing deficit, no mastoid sinus tenderness

**CV:** systolic murmur

**Skin:** healing punctate lesions on lower extremities

**Neuro:** no nuchal rigidity, weakness, or other focal deficits

#### LAB & IMAGING

- LP showed normal opening pressure, WBC 625 with 95% lymphs, glucose 63, protein 44
- Meningitis and encephalitis panels, CSF cultures, VDRL, HSV, RPR, and cytology negative
- MRI and CTA brain revealed only a large left mastoid effusion

### Case Resolution

- Within 48 hours of presentation the patient's headache significantly improved. Given the combination of headache, transient visual defects, and lymphocytic pleocytosis, she was discharged with a diagnosis of HaNDL.
- At a subsequent clinic visit she reported continued headaches with associated nausea. Further questioning also revealed that prior to admission she had experienced expressive aphasia and limb motor deficits.

### Discussion

#### OVERVIEW

- HaNDL is a self-limited aseptic meningitis, featuring episodes of severe headache accompanied by neurologic deficits and lymphocytic pleocytosis, usually occurring multiple times within several months.
- Thought to represent a viral-triggered autoimmune response resulting in leptomeningeal vasculitis and cortical spreading depression as in migraine<sup>1</sup>
- It is a diagnosis of exclusion, often requiring an extensive workup to rule out life-threatening etiologies.
- Management involves only supportive treatment

#### DIAGNOSIS

##### Clinical:

- Transient neurologic deficits – hemiparesis, dysphagia, or hemiparesis – are part of the ICHD-3 diagnostic criteria, and may closely mimic stroke and the sensorimotor aura of hemiplegic migraine<sup>1</sup>.
- Also associated with aphasia (most common), visual field defects, encephalopathy, and other neurologic symptoms
- Approximately 50% of cases preceded by viral prodrome<sup>1,4</sup>

##### Imaging:

- MRI may show transient leptomeningeal enhancement and reduced venous signal representing focal hypoperfusion, in contrast to the enhanced venous signal of migraine. Changes often evolve in conjunction with pleocytosis and symptoms<sup>5</sup>

##### CSF findings<sup>1,2</sup>:

- Lymphocytic pleocytosis, with the majority of cases >100 WBCs with >90% lymphocytes
- Opening pressure elevated in 50-75% of cases
- Glucose usually normal
- Protein variable but often elevated

### Diagnostic Approach

	Predominant Symptoms	CSF Pleocytosis	Other Differentiators
HaNDL	Headache, transient neurologic deficits	Lymphocytic	
Hemiplegic Migraine	Headache, transient neurologic deficits	--	MRI - enhanced venous signal Family history
Mollaret's Meningitis	Meningeal	Lymphocytic	CSF HSV present in 50% of cases <sup>6</sup>
Drug-Induced Meningitis	Meningeal	Neutrophilic <sup>6</sup>	NSAID or other drug use
Bacterial meningitis	Meningeal	Neutrophilic	Low CSF glucose, CSF cultures
Viral Meningitis	Meningeal	Variable	--
Neoplastic Meningitis	Mixed	Lymphocytic	Cytology showing malignant cells <sup>7</sup>

### Key Takeaways

1. HaNDL is a self-limited, benign condition that presents with **headache, transient neurologic deficits, and lymphocytic pleocytosis**. This unique triad differentiates it from other life-threatening conditions such as stroke and hemiplegic migraine.
2. Imaging and lab testing alone are not definitive; these must be interpreted alongside the clinical presentation.
3. It is crucial to elicit a detailed history including symptoms resolved prior to presentation in all patients presenting with headache

### References

1. Armstrong-Jones and Krishnamoorthy. HaNDL Syndrome: Case Report and Literature Review. *Journal of Child Neurology* 2019, Vol. 34(3) 163-167
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3. Headache Classification Committee of the International Headache Society. The International Classification of Headache Disorders, 3rd Edition (beta). *Cephalalgia*. 2013;33(9):1-171
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Special thanks to the UT College of Medicine Chattanooga Department of Internal Medicine residents, faculty, and staff.

HaNDL-syndrome of headache and transient neurological deficits with CSF lymphocytosis

# Resident/Fellow Research/QI

- Ahmed Minas, MD
- ETSU
- Epidemiological Differences in Incidence and Survival of Ureteral Cancer in USA, 2000-2016,

## CONCLUSIONS

- Ureteral cancer is rare with a low age-adjusted incidence rate between 2000-2016.
- The 5-year relative survival was about 50%.
- Relevant patient factors that contribute to mortality rate include race and marital status.
  - Blacks have a higher mortality than whites.
  - Unmarried individuals have a higher mortality than married individuals.
- Relevant tumor characteristics that contribute to mortality rate include histology and grade.
  - Transitional cell carcinoma has better prognosis than adeno-, epithelial cell, and squamous cell carcinomas.
  - Grades III and IV have worse prognosis than Grade I does.
- These results identify disparities in survival outcomes among different patient populations with ureteral cancer.

# Student First Place-3 way tie

- Christine Joyce, UT Medical Center, Knoxville
  - Hyperbaric Oxygen Therapy-Induced Seizures in Patients on Tramadol: A Case Series
- Irtiqa Fazilli, MS4, UT Memphis
  - A case of COVID-induced SIADH
- Rohan Tummala, MS4, UT Memphis
  - Clinical and Socioeconomic Predictors of Palliative Care Use



# Hyperbaric Oxygen Therapy-Induced Seizures in Patients on Tramadol A Case Series

Christine Joyce, BS; Zach Poindexter, EMT; Michael Freeman, MD; Mitchell Goldman, MD; Daphne Norwood, MD  
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## Introduction

Tramadol is an analgesic used for pain management. At therapeutic levels there is a low incidence of seizures; however, at higher doses tramadol inhibits  $\gamma$ -aminobutyric acid (GABA) which can induce seizures.

Hyperbaric oxygen (HBO2) therapy works by increasing the atmospheric pressure while allowing the patient to breathe in 100% oxygen. This treatment is frequently utilized in wound healing centers for treatment of non-healing wounds but can also be used for a variety of other medical problems.

Seizures on HBO2 therapy are thought to be related to oxygen toxicity and lowering the seizure threshold. Occurrence of seizures at our institution when administering HBO2 therapy is less than 1%. In the last twelve months two out of three patients on tramadol had seizures while receiving HBO2 therapy in our wound care center. Therefore, we hypothesize that tramadol is a contributing factor to developing seizures while undergoing HBO2 therapy. This paper addresses various commonalities and differences between these three patients to determine the hazards of developing a seizure for patients receiving HBO2 therapy while on tramadol.

## Hyperbaric Oxygen Chamber



<https://www.xosofts.com/Software/Science/How-Doctors-Use-Treatments-Hyperbaric-Oxygen-Therapy/>

## Discussion

- HBOT has been shown to interact with numerous medications through pharmacodynamic or pharmacokinetic mechanisms.<sup>6</sup>
- Since these patients had not had seizures while on tramadol prior to HBOT, it can be assumed that they were not abusing or misusing the medication. Therefore, it must be concluded that the HBOT contributed to this decreased seizure threshold.
- Since patients A and C were on the same dosage of tramadol, we do not believe that dosage of tramadol is a contributing factor.
- Receiving over 30 treatments of HBOT may be a contributing factor to onset of seizure.
- Because patient A and C were on the same dosage for the same amount of time prior to HBOT this would suggest length of time on tramadol is not a contributing factor.

## Conclusions

- Patients should be carefully monitored if receiving > 30 treatments of HBOT while on Tramadol

## Cases

Patients	Age (years)	Race	Sex	Reason for HBOT	# of Sessions	Length of sessions (minutes)	Pressure (ATA)	# of 5-min Air Breaks	Tramadol Dosage	Length of Time on Tramadol Prior to Start of HBOT	Seizure?
Pt A	70	Caucasian	F	Soft Tissue Radionecrosis of lower GI & GU Tracts	54	90	2.5	2	50 mg BID prn	3 months	Yes
Pt B	73	Caucasian	M	Osteoradionecrosis of the mandible	32	90	2.5	2	50 mg TID prn	≥ 6 months	Yes
Pt C	49	African-American	F	Non-healing diabetic leg ulcer	30	90	2.0	0	50 mg BID prn	3 months	No

## Resources

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## A case of COVID-induced SIADH

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### Introduction

COVID-19 has a wide range of presentations, from asymptomatic cases to severe respiratory failure. Reports of atypical signs and symptoms are still emerging in the setting of the global pandemic. Here, we report a case of COVID-19 which presented with fever and hyponatremia caused by SIADH. Implications and possible mechanisms are discussed.

### Case Presentation

A 76-year-old African American female with a history of diabetes, hypertension, and hyperlipidemia presented to the ED with 4 days of sharp 10/10 abdominal pain localized to the left lower quadrant. The pain did not radiate and was not associated with food or defecation. She endorsed diarrhea and denied any nausea, vomiting, or constipation.

On presentation to the ED, the patient was febrile to 38.3, blood pressure 114/64, pulse of 101, and respirations of 22 breaths/min. Patient was initially noted to be saturating at 86% on room air, which increased to 93% on 4L nasal cannula. CT chest/abdomen/pelvis showed patchy sub-pleural ground-glass and interstitial opacities and enlargement of the pulmonary artery. Labs were notable for sodium of 124, ferritin of 684.4, procalcitonin 0.19, and creatinine of 2.64.

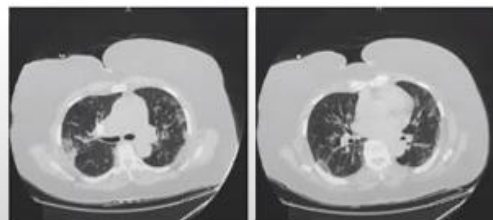


Figure 1. CT chest showing patchy subpleural ground-glass opacities compatible with atypical/viral pneumonia. The patient also had a positive COVID test.

### Hospital Course

On admission, the patient was admitted to the COVID unit and confirmed to be COVID positive. She continued to have increased work of breathing and respiratory distress on high flow nasal cannula with FIO2 50%, so she was escalated to Vapotherm and admitted to the ICU. There, she was treated with Remdesivir, convalescent plasma and steroids.

### Hospital Course (cont.)

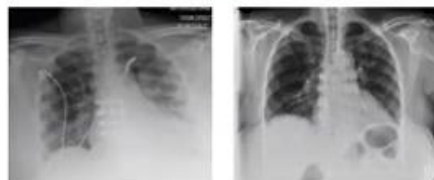


Figure 2. Chest radiograph on admission (left) showing patchy airspace disease and pleural effusion in the dependent left lung base, with patchy interstitial pneumonitis in the mid-right lung. Chest radiograph at transfer from ICU (right) showing interval improvement in pulmonary aeration with mild remaining reticulonodular infiltrates bilaterally.

When she was stabilized and transferred to the floor after two weeks, her persistent hyponatremia (Na 124-131) did not improve with normal saline and continued to worsen to 119. Of note, the patient had no prior diagnosis of kidney disease or hyponatremia and was not on any medications causing hyponatremia. On admission, the patient's urine osmolality was 372, which increased to 472 and then to 619 over the course of her admission, with urine sodium fluctuating between 41 and 75. The patient was fluid restricted and rapidly corrected with 3% sodium chloride. Her sodium improved to 130 and she was discharged on fluid restriction and salt tabs with close PCP follow-up.

### Case Discussion

SIADH causes euvolemic hyponatremia, and the following criteria should be fulfilled to make the diagnosis [1]:

- plasma sodium concentration <135 mmol/L
- plasma osmolality <280 mOsmol/kg
- urine osmolality >100 mOsmol/kg
- urinary sodium concentration >40mmol/L
- patient clinically euvolemic
- absence of clinical or biochemical features of adrenal and thyroid dysfunction
- no diuretic use

In our case of an elderly female who is recovering from COVID-19 infection, the patient has acute hyponatremia with highly concentrated urine and serum sodium about 40; thyroid and adrenal function are normal. She meets criteria for syndrome of inappropriate antidiuretic hormone. Common causes of SIADH include malignancy, pulmonary conditions, central nervous system disorders, and medications. These etiologies were excluded in our patient.

### Case Discussion (cont.)

More recently, as of June 2020, there have been reports of COVID-19 pneumonia-associated excess of vasopressin secretion. As in the case we present here, these cases have presented with fever, hyponatremia, and evidence of atypical COVID pneumonia on CT scan [2].

COVID-19 presentation varies widely, from asymptomatic to severe respiratory failure. Common clinical symptoms include fever (88.5% of cases), cough (68.6%), myalgia (35.8%), and dyspnea (21.9%). Less commonly, patients present with headache or dizziness (12.1%), diarrhea (4.8%), and nausea/vomiting (3.9%) [3]. In light of more recent cases, hyponatremia and electrolyte imbalances should be included in future meta-analyses to determine the rates at which COVID infections present with these signs. In the interim, the clinician should keep COVID-19 on his or her differential when a patient presents with fever and hyponatremia.

Viral, bacterial, and tubercular pneumonias can lead to SIADH, though the mechanism is unclear. Infrequently, a similar response can be seen with other pulmonary diseases like asthma, atelectasis, acute respiratory failure, and pneumothorax. We suggest that COVID-19-induced SIADH shares the same underlying pathophysiology as these conditions. One possible mechanism is a sequela of hypoxia-induced pulmonary vasoconstriction, which reduces left atrial filling, and induces baroreceptors to stimulate release of ADH [4].

### Conclusions

As healthcare systems continue to adapt to the global pandemic, it is important to identify signs and symptoms of COVID-19 as early as possible to assist in triage, isolation, and appropriate treatment of patients. As such, awareness of abnormal or non-respiratory presentations is key. In this case, the patient presented with diarrhea, abdominal pain, and hyponatremia due to SIADH. Her respiratory failure and pulmonary sequelae of the infection manifested relatively late in her illness; earlier clinical suspicion from her primary care provider could have resulted in earlier presentation, isolation, and intervention.

### References

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## INTRODUCTION

### BACKGROUND

Palliative care continues to gain recognition among primary care providers, as patients suffering from chronic conditions may benefit from use of this growing service. Socioeconomic status (SES) and clinical indicators such as the Charlson Comorbidity Index (CCI) could help physicians identify patients for whom earlier referral to palliative care may be beneficial.

### OBJECTIVE

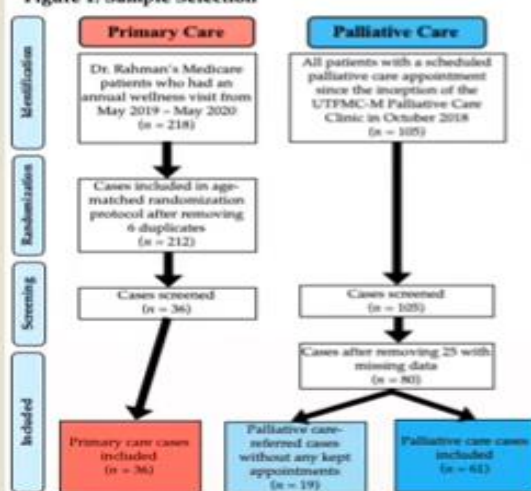
This single-institution quality improvement study investigates the clinical and socioeconomic characteristics of palliative care patients and identifies predictors of palliative care use.

### RESEARCH QUESTIONS

- Do primary and palliative care patients at the University of TN Family Medicine Center, Memphis, TN (UTFMC-M) differ in disease burden and socioeconomic status?
- Are there clinical and socioeconomic predictors of palliative care use at UTFMC-M?

## METHODS

Figure 1. Sample Selection



### RETROSPECTIVE CHART REVIEW

Clinical and socioeconomic data were collected for each patient using the NextGen electronic medical record. American Community Survey data were used to match patient ZIP codes with median household incomes.

### STATISTICAL ANALYSIS

Data were analyzed using SPSS and Microsoft Excel. Backward conditional variable selection was used to generate a Poisson regression model of palliative care use.

# Clinical and Socioeconomic Predictors of Palliative Care Use

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## RESULTS

Table 1. Patient Characteristics Stratified by Care Group\*

	Primary Care	Palliative Care-Referred Non-User	Palliative Care	Total	p-value
Indigency Rate (%)	34 (51.0)	19 (16.4)	61 (52.6)	114 (100.0)	0.790
Age (years), mean ± SD	60.5 ± 13.9	60.8 ± 16.7	63.3 ± 14.0	62.0 ± 13.8	0.495
Sex, n (%)					
Female	22 (61.1)	12 (60.2)	30 (54.1)	47 (57.6)	
Male	14 (38.9)	7 (34.8)	28 (45.9)	49 (42.2)	
Race, n (%)					0.330
Black	28 (77.4)	14 (73.7)	37 (63.8)	79 (64.9)	
White	8 (22.2)	5 (26.3)	21 (36.2)	34 (35.1)	
Distance to Clinic (mi), mean ± SD	6.7 ± 4.9	10.1 ± 8.5	12.8 ± 13.5	11.1 ± 10.8	0.226
Median Household Income (USD)	\$36,824 (\$31,499)	\$34,624 (\$31,499)	\$36,875 (\$37,446)	\$36,824 (\$36,659)	0.456
CCI (range)†, mean ± SD	30.2 ± 8.1	32.6 ± 11.7	25.9 ± 8.6†	28.3 ± 9.1	0.011*
CCI Risk Score, n (%)					<0.001*
Low	9 (22.2)	2 (11.1)	36 (62.2)	47 (45.7)	
High	18 (50.0)	7 (38.9)	11 (18.8)	36 (35.3)	
Very High	10 (27.4)	9 (50.0)	12 (20.0)	31 (27.0)	
CCI median (IQR)‡	0.00	0.00	0.00	0.00	<0.001*
CCI median (IQR)‡	4 (3.75)	4 (3.50)	4 (3.0)	4 (4.0)	0.002*
Chance of Survival (IQR)§	65% (50%)	25% (25%)	25% (25%)	25% (25%)	0.002*

\*ACG, Adjusted Clinical Groups; BMI, body mass index; CCI, Charlson Comorbidity Index; IQR, interquartile range; SD, standard deviation; \* denotes significant difference among all pairs (p < 0.05).

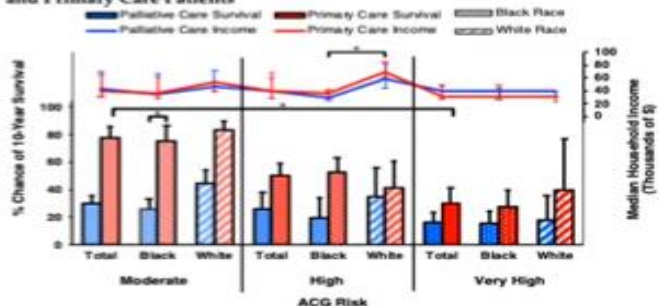
† denotes significant difference from Primary Care group (p < 0.05).

‡ denotes significant difference from Palliative Care-Referred Non-User group (p < 0.05).

§ denotes significant difference from Palliative Care-Referred Non-User group (p < 0.05).

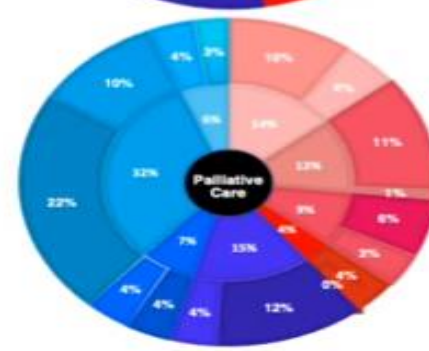
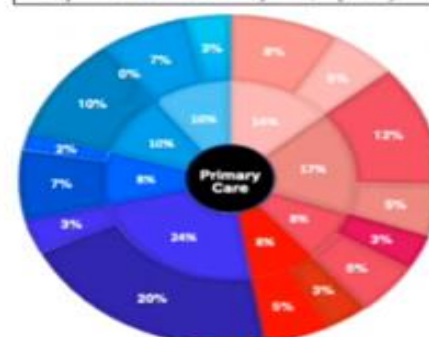
Palliative care patients had a lower chance of 10-year survival (p = 0.002) and tended to have lower ACG risk scores (p < 0.001) than primary care patients. Palliative care-referred non-users tended to have very high risk.

Figure 2. Disease Burden and Income Across Risk Groups for Palliative and Primary Care Patients



While chance of survival for very high-risk primary care patients was lower than for moderate-risk patients (30% vs. 78%; p = 0.019), it did not differ across risk groups for palliative care patients (p = 0.678).

Figure 3. Diagnoses by Race



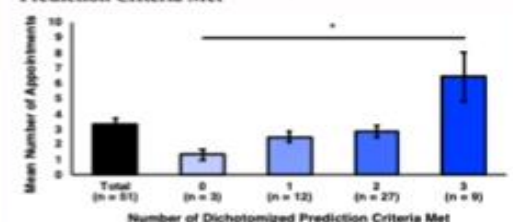
Palliative care patients differed from primary care patients in comorbidity distribution with a higher prevalence of cancer ( $\chi^2 = 14.648$ , df = 7, p = 0.041).

## RESULTS, CONTINUED

Table 2. Poisson Regression Model of Palliative Care Use

Predictor	Wald Statistic	Exp(B)	p-value
Race (1 = Black)	3.187	1.372	0.074
Referral from Hospital (1 = Referred)	6.267	1.671	0.009
Charlson Comorbidity Index	8.930	0.907	0.003
Morphine Milligram Equivalents	3.803	0.998	0.051
Number of Medications	8.705	1.043	0.003
Systolic Blood Pressure	8.234	0.989	0.004

Figure 4. Palliative Care Use by Number of Dichotomized Prediction Criteria Met



Significant predictors of palliative care use are referral from hospital (p = 0.039), a greater number of prescribed medications (p = 0.003), a greater chance of survival (p = 0.003), and a lower systolic blood pressure (p = 0.004).

## DISCUSSION

Figure 5. Recommendations



### LIMITATIONS

- Moderate degree of overdispersion in regression model
- Limited sample sizes in race-risk cross-tabulation
- Patient recall bias when reporting prescribed medications
- Height and weight were unavailable for patients in wheelchairs, so data are biased toward patients with greater mobility and potentially higher survival chance.

### FUTURE DIRECTIONS

- Identify patients' reasons for visiting palliative care.
- Explore why some patients who are referred to palliative care do not keep their appointments.
- Investigate why patients who make more palliative care appointments do not significantly differ in chance of 10-year survival and socioeconomic status.

CONGRATULATIONS