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Anemia at hospital admission

And the severity of acute kidney injury

By

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Degree to be awarded: M.P.H.

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#### Abstract

<u>Background:</u> Acute Kidney Injury (AKI) is a common complication of hospitalized patient s that is associated with increase in hospital cost, length of stay and mortality. It is frequently associated with anemia. We seek to determine if anemia at hospital admission is associated with increased severity of AKI.

<u>Methods:</u> This was a retrospective chart review of all adult admissions with AKI during 2011 at a tertiary hospital. Cases of AKI were identified based on diagnosis code. Excluded were cases with chronic kidney disease. AKI was defined as an increase in serum creatinine of at least 0.3 mg/dl and classified into levels of severity as mild (increase in serum creatinine by 0.3-0.5), moderate (increase in serum creatinine by 0.5-1.0) and severe (increase in serum creatinine greater than 1.0). Anemia was defined as hemoglobin of less than 10 g/dl.

<u>Results:</u> There were 454 patients with 495 admissions in which AKI was one of the discharge diagnoses during 2011. 421 patients had single admissions while 33 patients had multiple admissions ranging from 2-4 admissions. Anemia was not associated with increased severity of AKI or dialysis. Anemia was however significantly associated with a two times increased odds of death in the unadjusted model (OR 2.01 95% CI 1.31-3.10). The presence of malignancy modified this association. Patients who had malignancy in addition to anemia and AKI had almost a 4 times increased odds of death at 3.61 (95% CI 1.75-7.47) while those without malignancy but with AKI and anemia had

iv

no increased odds of death OR 0.94 (95% CI 0.49-1.80). There was also a trend towards an increase in length of stay in anemic patients at admission. Lower hemoglobin, Black race and diabetes were factors associated with multiple admissions for AKI. Having multiple admissions for AKI was also significantly associated with increased odds of requiring dialysis. Anemia at hospital admission

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# Table of Contents

1.	Introduction	page 1
2.	Literature Review	page 4
3.	Methods	page 6
4.	Results	page 8
5.	Discussion	page 10
6.	Conclusion	page 12
7.	References	page 14
8.	Tables and Figures	page 16

Chapter 1: Introduction

Acute Kidney Injury (AKI) is a broad clinical syndrome of conditions resulting in an acute decline in kidney structure or function. It is defined as any of the following: (a) an increase in serum creatinine by  $\geq 0.3$  mg/dl within 48 hours; (b) an increase in serum creatinine to  $\geq 1.5$  times baseline, which is known or presumed to have occurred within the prior 7 days; or (c) a urine volume < 0.5 ml/kg/h for 6 hours [1]. AKI is a spectrum and varies in severity from mild impairment to severe impairment that requires organ support through renal replacement therapy (dialysis). AKI can be staged based on severity into 3 stages according to the Acute Kidney Injury Network (AKIN) staging system:

- Stage 1. An increase in serum creatinine by  $\geq 0.3$  mg/dl or an increase in serum creatinine to 1.5-1.9 times baseline or a urine volume < 0.5 ml/kg/h for 6-12 hours
- Stage 2. An increase in serum creatinine to 2.0-2.9 times baseline or a urine volume < 0.5 ml/kg/h for >=12 hours
- Stage 3. An increase in serum creatinine by  $\geq 4.0 \text{ mg/dl}$  or an increase in serum creatinine to  $\geq 3.0$  times baseline or initiation of renal replacement therapy or a urine volume < 0.3 ml/kg/h for  $\geq 24$  hours or anuria [1].

A clinical classification of each case of AKI into one of 3 groups: pre-renal, renal and post-renal helps in the understanding of the etiology of AKI. However, in some cases an exact etiology remains unknown and in other cases, may be due to multiple factors. The pre-renal group includes conditions resulting in reduced effective kidney perfusion such as volume loss, bleeding, sepsis and hepato-renal syndrome. The renal group is a much broader and heterogeneous group and includes conditions such as acute glomerulonephritis, acute interstitial nephritis and acute tubular necrosis. The post renal group includes urinary obstruction, both intrinsic and extrinsic.

The incidence of AKI varies depending on the definition and clinical setting. In a study of nearly 20,000 admissions at a urban medical center, AKI as defined as a serum creatinine increase of 0.3 mg/dl or 1.5 times increase in baseline creatinine or initiation of dialysis was present in 22.7% of hospitalized patients and accounted for an independent increase in the odds of in hospital mortality of 4.43 (95% CI 3.68-5.35). [2] In another similar study of hospitalized patients using a different definition for AKI, an increase in serum creatinine by 0.5 mg/dl was present in 13% of patients and associated with a 6.5fold (95% CI 5.0 - 8.5) increase in the odds of death, a 3.5-day increase in length of stay (LOS), and nearly \$ 7500 in excess hospital costs. [3]. AKI is primarily a problem of hospitalized patients. The incidence of community-acquired AKI is much lower. In an older prospective study of hospital admissions, AKI was present in about 1% of patients on admission [4]. The incidence of AKI is however increasing; in a study of hospitalized Medicare beneficiaries between 1992 and 2001, the rates of AKI increased by approximately 11% per year [5]. Patients with AKI who are treated with dialysis have a mortality of 50-60%. Of surviving patients, 5-20% remain dialysis dependent at hospital discharge. [6]

There are many complications associated with AKI including fluid overload, electrolyte and metabolic derangements such as hyperkalemia and metabolic acidosis,

anemia, and death. The etiology of anemia in AKI is thought to be multifactorial and related at least in part to erythropoietin deficiency, sepsis, or uremia induced bone marrow toxicity and frequent blood sampling. [7-9]. Anemia is also a prevalent condition in hospitalized patients and is independently associated with excess mortality. In a 10 year retrospective review of hospitalized patients, anemia was present in 8.7% - 12.8% and was independently associated with 4.4 (95% CI 3.3-5.9) increased adjusted odds of mortality[10]. The co-existence of anemia and AKI is very common. In one observational study of 56 patients, anemia as defined as a hematocrit of less than 35% was present in 91% of patients with acute renal failure and was correlated with oliguria and rise in serum creatinine [11]. The conventional thinking is that anemia is at least in part a consequence of the AKI. In 25% of patients, however, anemia preceded the onset of rise in creatinine. [11] It will be informative to determine if anemia is associated with increased severity of AKI.

#### **Research Question**

Main Question: Is anemia at hospital admission associated with increase in the severity of AKI?

Null Hypothesis: Anemia at hospital admission is not associated with the severity of AKI.

Additional Questions: Is anemia at hospital admission associated with increase in the severity of AKI needing dialysis, hospital mortality, or an increase in length of stay?

Null Hypothesis: Anemia at hospital admission is not associated with the severity of AKI needing dialysis, hospital mortality, or an increase in length of stay.

#### Chapter 2: Literature Review

There are many predisposing factors to AKI and a recent systematic review and meta-analysis of 31 observational studies found significantly increased risk of AKI with older age, diabetes, hypertension, higher baseline creatinine, heart failure, sepsis/systemic inflammatory response syndrome, use of nephrotoxic drugs, higher severity of disease scores, use of vasopressors or inotropes, high risk surgery, emergency surgery, use of intra-aortic balloon pump, and longer time on cardiopulmonary bypass pump. [12]

Anemia is also being recognized in a few studies as a predisposing factor to the development of AKI, in addition to being one of the main complications of AKI. A retrospective cohort study of 2006 admissions to an Israeli hospital found that baseline anemia at admission significantly increased the adjusted odds of developing AKI defined as a 24% or more decline in eGFR at OR 1.5 (95% CI 1.4–1.6). There was also a graded increase in cumulative incidence of developing AKI with severity of baseline anemia. 5.9% of patients with admission hemoglobin greater than or equal to 12 developed AKI compared to 10.4% with hemoglobin 10-12 and 14.1% with hemoglobin less than 10. [13] At least 3 studies have shown that anemia may also predispose to development of AKI in cardiac surgery. A single-center prospective study of 1214 consecutive patients undergoing coronary artery bypass grafting found that pre-operative anemia of

hemoglobin less than 11 independently predicted AKI of at least 25% decline in eGFR or 1.5 times increase in serum creatinine with an OR of 2.06 (95% CI 1.14–3.70) [14] A multi-center cohort study of cardiac surgery patients reached a similar conclusion with a peri-operative hemoglobin of less than 10 being associated with a 1.99 increased odds (1.29–3.08) of developing AKI as defined by a 25% decrease in eGFR or dialysis[15]. In a single center prospective cohort study of tricuspid valve surgery, each 1g increase in hemoglobin was protective against development of AKI with OR 0.76 (95% CI 0.70–0.83) in multivariate analysis [16] A prospective study of patients undergoing coronary angiography or percutaneous coronary intervention found that a lower pre procedure hemoglobin independently predicted increased risk of contrast induced AKI on multivariate analysis. [17]

Given that multiple observational studies are reporting anemia as a potentially modifiable risk factor for the development of AKI, at least 2 studies have examined the effect of blood transfusion as a possible intervention to reduce the risk of developing AKI. An under powered un-blinded, parallel-group, randomized pilot trial found a trend but no significant association between prophylactic transfusion before cardiac surgery and development of post-operative AKI. [18] Another case controlled study of blood transfusion to patients with acute lung injury found no effect on the development of acute kidney injury. [19] Chapter 3: Methods

### Study Design

This is a retrospective chart review of all adult admissions to Carle Foundation Hospital, a tertiary hospital in Urbana, IL for the calendar year 2011. Included were all admissions with International Classification of Disease 9 (ICD-9) codes for acute renal failure or AKI. Sampling was not done. Excluded were admissions with ICD-9 codes for chronic renal failure, chronic kidney disease or end stage renal disease. Also excluded were miscoded admissions in which the trend of the serum creatinine did not confirm AKI.

# Data Collection

Data was collected on patient characteristics such as demographics, laboratory data, comorbid conditions and dialysis from the hospital computerized database. Demographic information obtained included age at time of admission, gender, race, ethnicity, length of stay and status on discharge as either deceased or alive. Age was divided into eight 10 year categories. Race was classified into white, black or African American, Asian and other races. Laboratory data obtained were the hemoglobin (Hb) at time of admission and all the serial serum creatinine (SCr) values during that admission. Co-morbidities information was also obtained based on ICD-9 codes including congestive heart failure (CHF) (ICD-9 codes 428.00 – 428.90), sepsis (ICD-9 codes 995.90-995.94), diabetes mellitus (DM) (ICD-9 codes 250.00 – 250.93). There were multiple ICD-9 codes for cancer and the discharge diagnoses with codes with descriptive

names with the words cancer, carcinoma, malignant neoplasm, lymphoma, leukemia or multiple myeloma was used. Data on dialysis during the calendar year was obtained.

#### Variable Definition

- AKI was defined as increase in SCr of 0.3 during the hospital stay. Individuals
  with only one SCr value were considered not to have developed AKI. For
  individuals with more than two SCr measurements, the lowest was considered to
  reflect the baseline value, and the highest SCr compared to the baseline was used
  to calculate the differential and cases with a differential of 0.3 were considered to
  have AKI. For practical purposes I further sub-divided AKI into 3 classes:
  - a. Class 1 (Mild): increase in serum creatinine by 0.3-0.5
  - b. Class 2 (Moderate): increase in serum creatinine by 0.5-1.0
  - c. Class 3 (Severe): greater than 1.0 increase in serum creatinine.
- Preexisting anemia, as detected at hospital admission, was defined as Hb level below 10 g/dl. The first Hb reading during hospital entry was used to identify anemic patients at hospital admission
- 3. Outcomes :
  - a. Mortality: deceased or alive at discharge
  - LOS: difference in days between admission day and discharge or deceased date. This was divided into 4 categories: 1-2 days, 2-4 days, 5-10 days and 11-79 days.
  - c. Dialysis: initiation of hemodialysis for acute renal failure.

## Data Analysis

Dichotomous variables were described as frequencies. Means and standard deviations were calculated for continuous variables. Crude odds ratios with 95% confidence interval, confounding and effect modification were calculated by stratified (Mantel-Haenszel) analysis and logistic regression of the main exposure variable, anemia and potential confounders including age, sex, race, co-morbidities and procedures. Two group comparisons were done using t-test comparison for 2 classes and one way analysis of variance was done for multiple classes. Effect modification was assessed by testing the significance of the cross product terms at the 0.01 level of significance and meaningful confounding was assessed by the 10% rule of difference between the adjusted and the unadjusted odds ratio. A backward elimination approach with a level of significance at 0.01 was used in proportional ordinal logistic regression to select a multiple adjusted model. Data was analyzed using SAS 9.3 software.

#### Chapter 4: Results.

#### **Baseline characteristics**

There were 454 patients with 495 admissions for AKI during 2011. 421 patients had single admissions while 33 patients had multiple admissions ranging from 2-4 admissions for the calendar year. 55% (252) were males and 84.4% (383) of all AKI admission was in patients over the age of 50 years. The mean age was 66.5 years with a standard deviation (S. D) of 17.1 years. 84.4% (383) identified as White, 9.3% (42) as Black, 5.6% (25) as Other races and 0.4% (2) as Asians. Only 0.9% (4) self-identified as

Hispanic Ethnicity. Co-morbidities to AKI were common: 34.8% (158) had associated cancer diagnosis; 41.9% (190) had a congestive heart failure (CHF) diagnosis; 44.7% (203) had a diabetes mellitus (DM) diagnosis and 44.9% (204) had a sepsis or systemic inflammatory response syndrome diagnosis. Anemia was present in 30.1% of hospital admissions with mean hemoglobin at admission of 11.2 g/dl and a S.D of 2.1g/dl. The mean increase in serum creatinine was 1.3 with a S.D of 1.5 and dialysis was required in 3.5% (16) of the patients. 24.3% (110) had mild AKI, 35.0% (158) had moderate AKI and 40.7% (184) had severe AKI. The length of stay (LOS) ranged from 1 to 79 days with a mean of 8.4 days and S.D of 8.9 days. 11.2% (52) had a LOS less than 2 days, 24.7% (112) had a LOS of 3-4 days, 40.3% (183) had a LOS of 5-10 days and 23.6% (107) had a LOS of greater than 10 days. 28.4% (128) died. (Table 1)

Anemia was not significantly associated with increased severity of AKI in the unadjusted and adjusted models and there was no significant 2 way interaction between AKI severity and any of the potential confounders including age, sex and co-morbidities (Figure 1, Table 2). However, anemia was significantly associated with increased mortality, unadjusted OR (2.00 95% CI 1.29-3.07). The presence of malignancy modified this association with an OR of 0.94 (95% CI 0.49-1.80) in those without malignancies and OR of 3.61 (95% CI 1.75-7.47) in those with a history of malignancy. Anemia was not significantly associated with dialysis in both the unadjusted and adjusted models. In both the unadjusted and adjusted models, anemia was significantly associated with a length of stay of 5-10 days compared to 1-2 days as baseline. There was no significant association with other length of stay category groups for both unadjusted and unadjusted

models but overall there was a significant trend of anemia at admission associated with an increase length of stay. (Table 3)

421 people had one admission with AKI while 33 patients had multiple admissions ranging from 2-4 admissions for the calendar year. The age and length of stay was similar between the single admit and multiple admit groups at 66.5 years (S.D 17.4 years) and 66.7 (S.D 12.5 years) and 8.6 days (S.D 9.1 days) and 6.4 days (S.D 4.7 days), respectively. The 2 groups were similar in most characteristics such as 10 year age categories, sex distribution, ethnicity, co-morbidities such as cancer, CHF, sepsis, anemia, severity of AKI and mortality. However the group with multiple admits had significantly more people with diabetes (69.7% vs. 42.8% p value <0.01), were more likely to have had dialysis, (9.1% vs. 3.1% p value <0.01), more likely to be Black (24.2% vs. 8.1% p value <0.01) and had a higher hemoglobin 11.2g/dl vs. 10.8g/dl (p value 0.02) (Table 4)

#### Chapter 5: Discussion

In this study of 454 patients hospitalized with AKI coded as one of their diagnoses at our tertiary center, anemia on hospital admission as defined as a hemoglobin less than 10g/dl did not play a role in the severity of AKI; patients who were anemic or non-anemic were both likely to have the same severity of AKI. Anemia in our study was present on admission and preceded the development of AKI as determined by at least a 0.3 mg/dl rise in creatinine. Anemia was significantly associated with a two times

increased odds of death in the unadjusted model (OR 2.01 95% CI 1.31-3.10). The presence of malignancy modified this association in that patients who had malignancy in addition to anemia and AKI had almost a 4 times increased odds of death at 3.61 (95% CI1.75-7.47) while those without malignancy but with AKI and anemia had no increased odds of death OR 0.94 (95% CI 0.49-1.80). The mean admission hemoglobin was 10.7 g/dl with a S.D of 1.9 g/dl for those with malignancy and 11.5 g/dl with a S.D of 2.1 g/dl for those without malignancy. This may be because of the multiplicative effect of 2 adverse risk factors of anemia and malignancy on mortality. Lower hemoglobin, Black race and having diabetes were associated with multiple admissions for AKI. Having multiple admissions for AKI was also significantly associated with increased odds of requiring dialysis. The mean peak creatinine was not significantly different in the group with multiple admits compared to the group with single admits (2.3 mg/dl versus 2.5 mg/dl T-Test p value = 0.4) This may be because of 2 reasons; chronic kidney disease needing dialysis may result from repeated renal insults leading to multiple admissions for AKI or a subsequent AKI admission may be severe enough to require dialysis for acute reasons. This is useful as at the time of index admission, we can identify those at risk for re-admission for AKI and plan interventions accordingly to prevent re-admission.

This study has a number of limitations. First, the data in the computerized data extraction was limited. There was no control group; a control group without AKI would have been useful to calculate the relative odds of anemia in both AKI and non AKI groups. Also, information on all 2011 admissions for the hospital would have been useful for calculating the incidence of AKI at the hospital. In particular, 2011 non AKI-related

admissions for the cases in this study could give more information on how many repeat admissions are associated with AKI. Other additional information that would have been helpful would be other potential confounders such as blood transfusion for anemia, nephrotoxic medications, and procedures associated with AKI. Second this was a small, single center, retrospective study with disadvantages including misclassification bias from the incorrect coding of diagnoses. This was evident from this data as only about 68% (495/728) of admissions initially extracted as AKI related admission by diagnoses code truly had AKI based on serum creatinine differential as defined by the study criteria. Also causality cannot be assumed from the retrospective nature of the study. Third, the racial and ethnic mix as well as health insurance of our study population could limit generalization of our study results to other populations. For future studies, a prospective study with a bigger sample size and additional information on other potential confounders could address some of the issues.

Contrary to previous studies [13-15] we did not find that anemia was associated with increased AKI severity. While this may indeed be the case, another explanation for our paradoxical results could be selection bias of our study population. The population was selected conditioned on development of outcome AKI and only about 30% had the exposure of interest, anemia. A different study design could lead to different observations.

Chapter 6: Conclusion

In summary, in a review of hospitalized patients with AKI during 2011 at our hospital, anemia at hospital admission was not associated with the severity of AKI that subsequently developed even when adjusting for potential confounders. Anemia was independently associated with mortality but not the severity of AKI. Certain risk factors such as diabetes, black race, and the need for dialysis were associated with recurrent admissions for AKI during that calendar year.

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# Tables

#### Table 1. Baseline characteristics of AKI cases

Characteristic					
	All				
	Frequency	%	Mean	S.D	
	N=454	100.0			
Age			66.5	17.1	
18-29	18	4.0			
30-39	18	4.0			
40-49	35	7.7			
50-59	74	16.3			
60-69	99	21.8			
70-79	95	20.9			
80-89	88	19.4			
90-101	27	6.0			
Sex					
Male	252	55.5			
Female	202	44.5			
P					
Kace White	383	81 1			
Black	42	0.3			
Asian	42	9.5 0.4			
Other	25	5.6			
Julei	25	0.0			
Ethinicity					
Hispanic	4	0.9			
Non-Hispanic	448	98.7			
Cancer					
Yes	158	34.8			
No	296	65.2			
CHF	100	41.0			
No	264	41.9			
110	204	00.2			
Diabetes					
Yes	203	44.7			
No	251	55.3			
Sensis					
Vac	204	11 0			
No	250	55.1			
Dialysis	10	0.5			
Yes	16	3.5			
IND	430	90. D			
Status					
Alive	322	71.6			
Deceased	128	28.4			
Langth of Star			0.4	0.0	
Length of Stay	50	11 5	ö.4	ö. 9	
1-2 days	52	017			
5-4 days	112	24.1			
3-10 days 11-79 days	103	40.5			
11-75 uays	107	20.0			
Anemia					
Yes	134	30.1			
No	311	69.9			
HaB			11.2	2.1	
- <del></del>					
AKI		04.0	1.3	1.5	
Wild U.3-U.5 Increase	110	24.3			
Woderate U.5-1.0 Increase	158	35.0			
Severe >1.0 Increase	184	40.7			

S.D = Standard Deviation If it doesn't add up then it is missing 454 people and 528 observations

# Table 2: AKI severity based on anemia at admission

Hb

		P value*	Trend**
			0.67
AKI 1 vs 2	1.11 0.65-1.91	0.7	
AKI 1 vs 3	1.26 0.75-2.13	0.38	
			0.67
AKI 1 vs 2	1.12 0.65-1.94	0.68	
AKI 1 vs 3	1.27 0.75-2.14	0.38	
			0.47
AKI 1 vs 2	1.13 0.65-1.96	0.67	
AKI 1 vs 3	1.37 0.81-2.34	0.24	
dities			0.71
AKI 1 vs 2	1.14 0.65-2.00	0.66	
AKI 1 vs 3	1.27 0.73-2.21	0.41	
	AKI 1 vs 2 AKI 1 vs 3 AKI 1 vs 2 AKI 1 vs 3 AKI 1 vs 3 AKI 1 vs 3 dities AKI 1 vs 2 AKI 1 vs 3	AKI 1 vs 2 AKI 1 vs 3 AKI 1 vs 3 AKI 1 vs 3 AKI 1 vs 2 AKI 1 vs 3 AKI 1 vs 2 AKI 1 vs 3 AKI 1 vs 3	P value* AKI 1 vs 2 1.11 0.65-1.91 0.7 AKI 1 vs 3 1.26 0.75-2.13 0.38 AKI 1 vs 2 1.12 0.65-1.94 0.68 AKI 1 vs 3 1.27 0.75-2.14 0.38 AKI 1 vs 3 1.27 0.75-2.14 0.38 AKI 1 vs 3 1.37 0.81-2.34 0.24 dities AKI 1 vs 2 1.14 0.65-2.00 0.66 AKI 1 vs 3 1.27 0.73-2.21 0.41

\*P value of Wald's Chi square \*\*Trend P value of Type 3 overall analysis of effects

Figure 1: Odds Ratio of AKI Severity on anemia adjusted for age sex and co-morbidities



		Odds Ratio	95% C.I.	P-value *	Trend**
Mortality	Crude	2.00	1.29-3.07	0.002	
	Interaction with malignancy (at 0.01)			0.007	
	No malignancy	0.94	0.49-1.80	0.000	
	Malignancy	3.61	1.75-7.47		
		0101			
	Adjusted Mortality (at 0.001) ***				
	with sex	2.15	1.38-3.34	<0.01	
	with age	1.79	1.15-2.79	0.01	
	with sex/age/comorbidities	1.67	1.04-2.67	0.03	
	with sex/age/comorbidities/AKI	1.68	1.05-2.69	0.03	
Dialysis	Crude	1.84	0.67-5.04	0.24	
	Adjusted				
	with sex	1.90	0.69-5.24	0.22	
	with age	2.07	0.73-5.83	0.17	
	with sex/age/comorbidities	2.13	0.70-6.44	0.18	
	with sex/age/comorbidities/AKI	2.05	0.66-6.38	0.22	
LOS	Crude				0.01
	LOS 2v1	1.73	0.72-4.14	0.22	
	LOS 3v1	3.08	1.36-6.97	0.01	
	LOS 4v1	1.91	0.80-4.56	0.15	
	Adjusted				
	with sex				0.02
	LOS 2v1	1.73	0.72-4.15	0.22	
	LOS 3/1	3.06	1.36-6.94	0.01	
	LOS 4v1	1.96	0.82-4.69	0.13	
	with age				0.03
		1 68	0 70-4 06	0 24	0.00
	105 3/4	2 90	1 27-6 59	0.24	
	LOS 4v1	1.90	0.79-4.57	0.01	
	with any/aga/agmarhidition				0.00
		1 50	0 60 0 70	0.26	0.09
		1.55	0.02-3.70	0.36	
		2.30	1.01-5.54	0.05	
	LOS 4V1	1.48	0.59-3.70	0.41	
	with sex/age/comorbidities/AKI				0.09
	LOS 2v1	1.53	0.62-3.82	0.36	
	LOS 3v1	2.35	0.99-5.59	0.05	
	LOS 4v1	1.42	0.56-3.64	0.46	

#### Table 3. Secondary Oucomes; Anemia on mortality, dialysis and LOS

\*P value of Wald's Chi square \*\*Trend P value of Type 3 overall analysis of effects

\*\*\*No interaction at 0.001 level

Figure 2a: Secondary Outcomes: Anemia on mortality adjusted for age, sex and co-morbidities with effect modification by the presence or absence of malignancy



Figure 2b: dialysis



# Figure 2c:



5	Single Ad	missions f	or AKI		Multiple Admissions for AKI				
F	Frequency	%	Mean	S.D	Frequency	%	Mean	S.D	P value
	N=421	100.0			N=33	100.0			
Age			66.5	17.4			66.7	12.5	0.81
18-29	17	4.0			1	3.0			
30-39	18	4.3			0	0.0			
40-49	33	7.8			2	6.1			
50-59	69	16.4			5	15.2			
60-69	89	21.1			10	30.3			
70-79	82	19.5			13	39.4			
80-89	86	20.4			2	6.1			
90-101	27	6.4			0	0.0			
Sex									0.35
Male	235	55.8			17	58.6			
Female	186	44.2			16	41.4			
Race									<0.01
White	360	85.5			23	69.7			
Black	34	81			8	24.2			
Asian	2	0.5			õ	0.0			
Other	22	5.5			2	6.1			
Other	23	5.5			2	0.7			
Ethinicity		4.0			0				0.71
Hispanic	4	1.0			0	0.0			
Non-Hispanic	415	98.6			33	100.0			
Cancer									0.85
Yes	147	34.9			11	33.3			
No	274	65.1			22	66.7			
CHF									0.84
Yes	176	41.8			14	42.4			
No	245	58.2			19	57.6			
Diabetes									<0.01
Yes	180	42.8			23	69.7			
No	241	57.2			10	30.3			
Sepsis									0.07
Yes	186	44.2			18	54.6			
No	235	55.8			15	45.5			
Dialysis									<0.01
Yes	13	3.1			3	9.1			50.01
No	408	96.9			30	90.9			
Status									0.29
Alive	296	71.0			26	78.8			
Deceased	121	29.0			7	21.2			
Length of Stav			8.6	91			64	47	0 12
1-2 days	48	11 1	0.0		4	12 1	<b>9</b> .1		0.12
3.4 days	40	23.5			4	30 /			
5-4 days	172	23.3			10	20.2			
5-10 days	1/3	41.1 24.0			10	18.2			
11-75 days	101	24.0			0	10.2			
Anemia	400	00.0							0.06
Yes No	290	29.8 70.2			21	34.4 65.6			
							40.0		
пов			11.2	2.1			10.8	2.0	0.02
AKI									0.09
Mild 0.3-0.5 increase	93	22.2			17	51.5			
And the second sec									
Moderate 0.5-1.0 increase	145	34.6			13	39.4			

#### Table 4: Comparison of single admissions versus multiple admissions for AKI

S.D = Standard Deviation

P. value of difference in means