

Antibiotic related acute kidney injury in patients treated for open fractures



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ABSTRACT

Objective: Antibiotic administration during the treatment of open fractures has been shown to reduce infection rates and is considered a critical step in the management of these injuries. The purpose of this study was to determine if aminoglycoside administration during the treatment of open fractures leads to acute kidney injury.

Methods: Patient records at a level I trauma centre were reviewed for adult patients who presented in 2014 with open fractures were screened for inclusion. Patients were excluded with fractures of the phalanges, metatarsals, and metacarpals, with isolated traumatic arthrotomies, or pre-existing renal dysfunction. Charts were reviewed for patient age, gender, race, past medical history, medication history, injury severity score, intravenous dye studies and fracture type. Patients were divided into those given cefazolin (Group A) and cefazolin with gentamicin (Group B). Laboratory values were used to determine which patients developed kidney dysfunction as measured using the RIFLE criteria. Wilcoxon–Mann–Whitney test and Chi-square were used to compare interval and categorical variables, respectively. Significance was set at $P < 0.05$.

Results: One-hundred and fifty-nine patients met inclusion criteria. Forty-one (25%) patients were given cefazolin alone and 113 (68%) patients were given cefazolin with gentamicin. Ten (18%) patients with Gustilo–Anderson type III fractures were given cefazolin alone and 67 (67%) patients with types I or II fractures were given a cefazolin with gentamicin. Baseline characteristics and risk factors for renal dysfunction did not vary between groups. Two (4.8%) patients in Group A and 5 (4%) patients in Group B developed acute kidney injury ($P = 0.599$).

Conclusions: Gentamicin use during the treatment of open fractures does not lead to increased rates of renal dysfunction when used in patients with normal baseline renal function.

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Introduction

Antibiotic administration during the treatment of open fractures has been shown to reduce infection rates and is considered a critical step in the management of these injuries [1–3]. While a 1st generation cephalosporin (e.g. cefazolin) is used for most patients with open fractures [4,5], adding an aminoglycoside (e.g. gentamicin) is generally recommended for patients with Gustilo–Anderson type III fractures to add coverage for gram-negative organisms [6,7]. While the Gustilo–Anderson fracture type has been shown to predict infection [6–8], final classification

is delayed until formal operative debridement [9]. Therefore decisions about antibiotic regimens that must be made upon admission rely on clinical judgement.

The Gustilo–Anderson classification has high interobserver variability, even after surgical debridement [10,11], so this preoperative judgement call may result in inappropriate antibiotic administration prior to definitive fracture type determination. While gentamicin may reduce infection rates in higher Gustilo–Anderson fracture types [6], it is unclear if routine use in open fractures increases complication rates. Prior studies have shown that gentamicin can cause renal dysfunction in patients undergoing elective joint arthroplasty [12–14], but our understanding of its impact on renal function in patients treated for open fractures is poorly understood. The primary aim of this study is to determine if gentamicin use during the treatment of patients with open fractures leads to increased rates of acute kidney injury.

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Patients and methods

After Institutional Review Board approval was obtained, all adult patients who presented to a level I trauma centre between January 1st and December 31st, 2014 with open fractures were screened for inclusion. Patients were excluded with open fractures of the phalanges, metatarsals, and metacarpals, as well as patients with isolated traumatic arthrotomies, estimated glomerular filtration rate (eGFR) less than 60 ml/min/1.73 m² on admission, patients seen greater than 24 h after injury, or with incomplete documentation. Charts were reviewed for patient age, gender, weight, race, past medical history, medication history, injury type, injury severity score, orthopaedic procedures, fracture type, drop in haemoglobin levels, intravenous dye studies, intensive care unit length of stay, and hospital length of stay. Medical history that was considered a risk factor for AKI included diabetes mellitus, hypertension, pregnancy, human immunodeficiency virus (HIV), nephrolithiasis, and hepatitis C. Current home medications that were considered a risk factor for AKI included angiotensin converting enzyme inhibitors, angiotensin receptor blockers, non-steroidal anti-inflammatories, and diuretics. Emergency department and inpatient medication administration records were used to divide patients into those given cefazolin alone (Group A) and cefazolin with gentamicin (Group B). Serum creatinine, gender, age, and race were used to calculate eGFR using the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation [15].

The eGFR on admission and throughout each patient's hospital stay were used to determine which patients developed acute kidney injury (AKI). Patients were separated by severity of AKI

based on decrease in eGFR from baseline using the RIFLE criteria, which has been shown to be predictive of in-hospital mortality in critically ill patients [16,17]. Patients with eGFR decrease >25%, >50%, and >75% from baseline were assigned RIFLE 1, 2, and 3, respectively [18]. No patients had AKI more severe than RIFLE 3. Based on prior literature, an a priori power analysis determined that 37 patients would be needed in each group to detect an 8% difference in AKI rate with 80% power (G*Power version 3.1.9.2). Shapiro–Wilk test was used to check interval variables for normality and either an independent sample t-test or Wilcoxon–Mann–Whitney test was then used. Chi-square or Fisher's exact tests were used to compare categorical variables. Spearman correlation was used to evaluate eGFR over time. Significance was set at $P < 0.05$. All statistical analysis was performed using STATA software (Version 13.1 MP; Stata-Corp, College Station, TX, USA).

Results

One-hundred and eighty six patients were identified on initial screening, of which 159 met inclusion criteria (Fig. 1). Forty-one patients were given cefazolin alone and 113 patients were given cefazolin with gentamicin. With the exception of a larger drop in haemoglobin seen in Group A, baseline characteristics and risk factors for renal dysfunction did not vary between groups (Table 1). Ten (18%) patients with type III fractures were given cefazolin alone and 67 (67%) patients with types I or II fractures were given cefazolin with gentamicin. The average dose of cefazolin given in group A was 3.0 g/day (SD 1.3 g) for 8 days (SD 7.5 days). The average dose of cefazolin given in group B was 3.2 g/day (SD 2.7 g) for 12 days (SD 14.4 days). The average dose of gentamicin given in

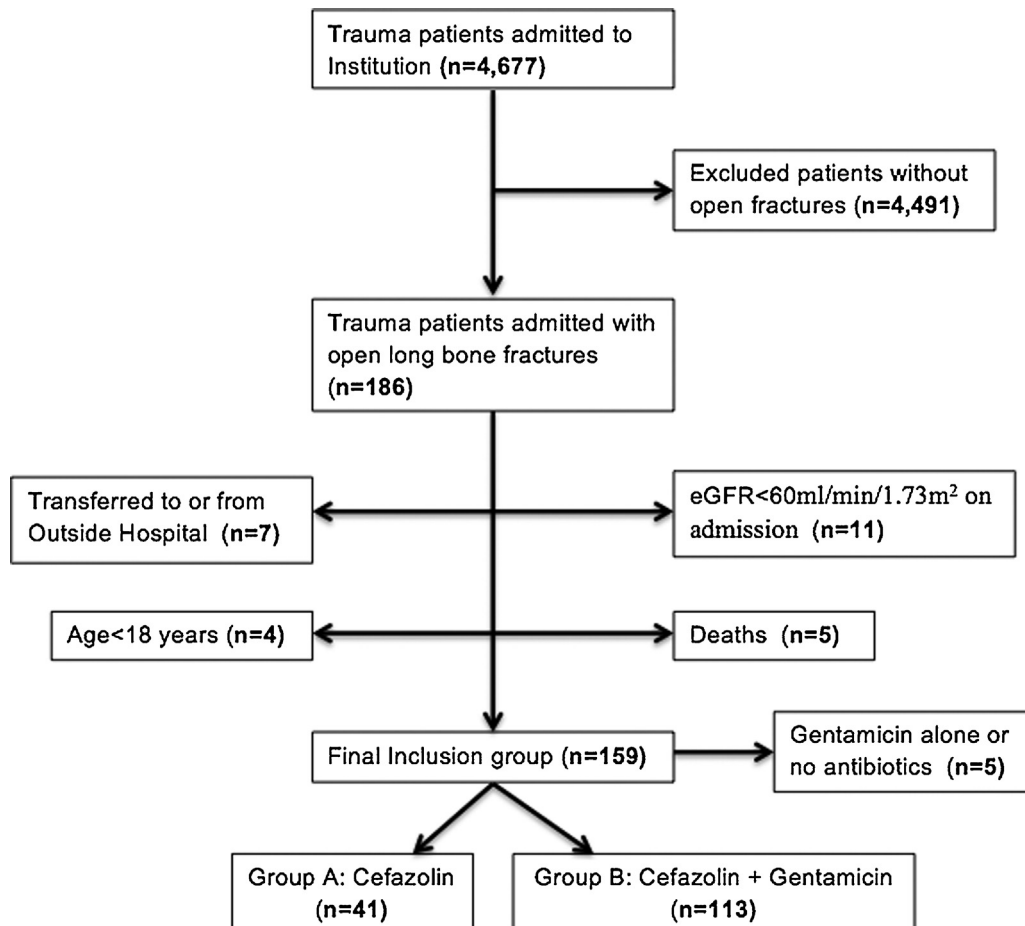


Fig. 1. Flowchart of patient selection.

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