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Methylprednisolone, Dexamethasone or Hydrocortisone for Acute Severe Pediatric Asthma: Does it matter?

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Abstract

Objective: Various intravenous (IV) corticosteroids are available for acute severe asthma (ASA) treatment. The choice of IV corticosteroids varies broadly and depends on institution, country, or physician preferences. In this study, we compared the efficacy of IV Methylprednisolone, hydrocortisone and dexamethasone in ASA treatment during pediatric intensive care unit (PICU) admission.

Methods: The study was a prospective randomized clinical trial. We enrolled patients of 1-21 years after they were admitted to the PICU requiring continuous beta-2 agonist treatment. Patients were randomized into 3 groups: Group A: IV Methylprednisolone, Group B: IV Hydrocortisone and Group C: IV Dexamethasone. The primary outcomes measured were durations of beta-2 agonist continuous nebulization treatment. Secondary outcomes included PICU and hospital length of stay (LOS), pediatric asthma severity score (PASS), need for mechanical ventilation and maximum dose of beta-2 agonist treatment.

Results: 61 patients were included in the analysis. 22 patients recruited in Group A, 20 in group B and 19 group C. Median durations of beta-2-agonist treatment were 23 h (QR 16-38) for methylprednisolone, 27 h (QR 16-40) for hydrocortisone, and 32 h (QR 16-48) for dexamethasone ($p = 0.90$). There was no difference in PICU LOS, hospital LOS, PASS score, B2 agonist maximum dose, or need for ventilation support.

Conclusions: The use of IV methylprednisolone, hydrocortisone, and dexamethasone have equivalent efficacy when used at the appropriate doses. Studies with larger cohorts are needed to compare the effectiveness of IV corticosteroids in the management of ASA in the PICU setting.

Keywords asthma; acute severe asthma; pediatrics; corticosteroids; pediatric intensive care unit; treatment outcomes

Introduction

Asthma is the most common chronic disease in childhood, affecting more than 6 million children and estimated to cause 863 per 100 000 of deaths in 2017 in the United States.^{1,2} The exposure of the hypersensitive airway to environmental stimuli activates an inflammatory cascade that results in episodic airway obstruction and subsequent remodeling. Bronchial narrowing and airflow obstruction are due to bronchoconstriction, mucosal edema and mucous production. Initial treatment of ASA consists of oxygen, corticosteroids, and bronchodilators, including inhaled beta-2 agonists, anticholinergic agents, and magnesium sulfate.

Pediatric patients are admitted to the PICU for further management of asthma when they fail to respond adequately to initial intensified treatment with Beta 2 agonists, ipratropium bromide, magnesium sulfate and corticosteroids.²

Corticosteroids have been used to manage asthma since 1950³. They play an important role in management of ASA exacerbations and are the mainstay of treatment when used adjunctively with inhaled bronchodilators^{2,4}. Studies, mostly performed in the ED setting, have shown that the use of corticosteroids in ASA reduced admissions, hospital length of stay (LOS) and relapse.⁵⁻⁷ Still, despite decades of corticosteroids use in asthma patients, their use in the PICU setting has not been well studied or reported.

Corticosteroids are synthetic analogues of naturally occurring corticosteroid hormones which can yield both glucocorticoid and mineralocorticoid effects. In asthma, glucocorticoids provide beneficial immunosuppressive effects by reducing T-lymphocyte mediated hypersensitivity reactions, anti-proliferative effects by inhibiting DNA synthesis and epidermal cell turnover, and vasoconstrictive effects by counter-acting the action of histamine and other vasodilatory mediators.^{8,9}

Different types and forms of synthetic corticosteroids have been used for the management of ASA. Comparatively, the available corticosteroid formulations have different potency and affinity for glucocorticoid receptors, relative mineralocorticoid activity, and pharmacokinetic characteristics.¹⁰ The corticosteroid formulations and doses can be converted to one another based on their relative glucocorticoid potency and activity.¹¹

For children with ASA requiring admission to the PICU, the intravenous (IV) route is preferred, because the oral route may not be well tolerated and absorption may not be optimal.¹² The choice of IV steroids varies broadly and depends on institutional and physician preferences.

Few studies have compared optimal forms and doses of synthetic IV corticosteroid for ASA in the PICU setting. The aim of our study was to compare the efficacy of 3 different IV corticosteroids in ASA patients admitted to the PICU.

Materials and Methods

Study Design

The study was approved by our institution's IRB (#855082-4). It was a prospective non-blinded clinical trial, which included pediatric patients between ages 1–21 years admitted to the PICU at our institution for ASA. Informed consents were obtained from the patients' legal guardian, as well as assent for minors aged 7 years and older at the time of PICU admission. Subjects with cardiovascular, neurological, psychiatric, neoplastic, or renal disease, and pregnant patients were excluded from the study. Patients with complications or comorbidities such as pneumonia, pneumothorax, or pneumomediastinum were also excluded. Demographic data was collected from all patients including age, sex, race, weight, and body mass index (BMI). Vital signs including respiratory rate, oxygen saturation, heart rate and blood pressure were obtained during the enrollment period. Asthma severity was assessed using the Global initiative for asthma guidelines.⁴ Patients were randomized using randomization in block sizes of 3. Treatment allocation was provided in sequential opaque sealed envelopes. Patients were divided into 3 groups; Group-1 received IV methylprednisolone (2 mg/kg/day, divided q6h, max dose 60 mg/day), Group 2 received IV hydrocortisone (5 mg/kg/day divided q8h, max dose 400 mg/day) and Group 3 received IV dexamethasone (0.6 mg/kg/day divided every 6 h, max. dose of 16 mg/day for 3 days). When determining the doses to utilize, consideration was given to the comparative glucocorticoid potency and the half-lives of the corticosteroids used, as well as published literature describing the use of

these formulations for asthma. Methylprednisolone was dosed using recommendations adapted from the published asthma treatment guidelines.^{2,4} Hydrocortisone dosing was determined using the equivalent dose with respect to methylprednisolone and based on the doses used in adult asthma studies.¹³⁻¹⁶ Dexamethasone dosing was adapted from several studies, as well as from a 2014 meta-analysis reporting 0.6 mg/kg as the most commonly used dosing. Due to its long half-life, the duration of therapy for dexamethasone was capped at 3 days.^{17,18} Methylprednisolone and hydrocortisone were discontinued with the discontinuation of continuous beta-2 agonist based on the clinical pharmacist recommendations. Adverse effects of corticosteroids were monitored throughout the study using glucose level, heart rate and blood pressure.

All subjects enrolled received continuous beta-2 agonist (minimum dose 5 mg/hr and maximum 20 mg/hr) and ipratropium bromide (500 mcg/dose) nebulization every 6 h for 48 h. Oxygenation and ventilatory support were determined based on disease severity and at the attending physician's clinical discretion. Corticosteroids received in the ED prior to study enrollment were collected. The decision to deescalate management was based on a systematic approach using improved pediatric asthma severity (PASS) scores. PASS is a validated clinical scoring tool and was used to assess severity of patient symptoms and clinical improvement.¹⁹ Patients were transferred out of the PICU once continuous beta-2 agonist treatment was no longer needed, and transitioned to beta 2- agonist nebs every 2 h. Data on duration of continuous beta-2 agonist treatment, maximum dose of beta-2 agonist used, type of mechanical ventilation required, PICU and hospital length of stay (LOS) were collected for all subjects. Median PASS scores were calculated for each group at 6, 12, and 24 h from enrollment. Bedside glucose values were collected at 6 h intervals until discharged from the PICU.

Statistical Analysis

Descriptive statistics including mean, median, standard deviation, and 25th-75th interquartile ranges were used to compare the groups. Univariable associations were statistically assessed using Pearson Chi-square test for categorical variables including gender, race, asthma severity and prior use of controller medications. ANOVA and Kruskal Wallis tests were used to compare parametric and non-parametric continuous variables including age, BMI, PICU and hospital LOS, PASS and duration of continuous beta-2 agonist. Kaplan Meier time to event, multivariate Cox proportional hazard models and log rank test were computed to compare time to discontinuation of continuous beta-2 agonist treatment. The study primary outcomes were duration of beta-2 agonist and duration PICU LOS. All tests were two-sided, with statistical significance defined as $p < 0.05$. All statistical analyses were performed using programming language R and RStudio.^{20,21}

A sample size of 21 was estimated to reach 80% power to detect a difference between the 3 groups. Sample size was based on the primary outcome of duration of continuous beta-2 agonist treatment to be 24 h (± 4), 28 h (± 4) and 32 h (± 4) for the Methylprednisolone, Hydrocortisone and Dexamethasone groups, respectively

Results

A total of 64 patients were enrolled in the study. Three patients were excluded from the study: one had complicated pneumonia with empyema and the other 2 had neurocognitive comorbidity (figure 1). The mean age of the enrolled patients was 7.35 ± 4.06 years ranging from 16 months to 17 years old. The majority of patients were African American ($N = 54$, 88.5%). 40% of the patients had intermittent asthma, 24% had mild persistent asthma, 29% had moderate persistent asthma and 7% had severe persistent asthma. 62 (97%) patients received corticosteroids in the emergency department. Overall median PICU and hospital LOS were 36 (Quartile Range (QR) 24,48) hours and 68 (QR 46 100) hours, respectively.

Group A received IV methylprednisolone ($N = 22$), Group B received IV hydrocortisone ($N = 20$), and Group C received IV dexamethasone ($N = 19$). All 3 groups were comparable in terms of age, sex, and race/ethnicity. Patients receiving IV methylprednisolone had a higher BMI compared to patients receiving IV hydrocortisone and IV dexamethasone (mean 22 ± 7.3 , 20.9 ± 5.8 and 17.86 ± 6.6) respectively, $p = 0.02$). Patient in all 3 groups were comparable at admission in terms of asthma severity, prior use of controllers and median PASS scores at study entry (Table 1).

The vast majority of patients ($>90\%$) received 5-10 mg/hour of beta-2 agonist treatment, and the median length of continuous nebulization did not differ among the 3 groups (Table 2). There was no significant difference between the 3 groups in duration of continuous beta-2 agonist treatment (Hazard Ratio Group A vs. B; 0.92, 95% CI (0.50, 1.70) and A vs. C (0.82 95% CI (0.4,1.5), p values = 0.5) (Figure 2)

Patients receiving IV methylprednisolone had shorter PICU and hospital LOS compared to those receiving IV hydrocortisone and IV dexamethasone (median PICU LOS (QR); 30 (20,38.3) vs. 36.5 (29.8,53.5) vs. 36 (28,49) hours, $p = 0.25$, median hospital LOS (QR); 62 (40 112) vs. 68.5 (47.5,93.75) vs. 71(50 102.5), $p = 0.92$). However, the results were not statistically significant (Table 2).

Patients' vital signs during study enrollment showed significant differences in mean arterial pressure (MAP) between the 3 groups. Patients receiving IV hydrocortisone had a higher MAP compared to those that received IV methylprednisolone or IV dexamethasone (MAP mean 71.6 ± 6.5 vs. 76.5 ± 8.2 vs. 71.3 ± 6.7), $p = 0.036$). There was no difference in mean heart rate between the 3 groups ($p = 0.82$) (Table 2).

Discussion

We found great variability in corticosteroid choice and dosing in the management of ASA in the PICU setting. we assessed the relative efficacy of dexamethasone, hydrocortisone, and methylprednisolone in critically ill children with ASA admitted to the PICU. We compared the duration of continuous nebulization with beta-2 agonist, the improvement in PASS scores and the PICU and hospital LOS and found that all 3 IV corticosteroids were comparable in terms of each variable measured.

Our study was performed in an urban setting in which 89% of participants were African American children. The characteristics of ASA in African American children is not well studied in literature. Published studies predominantly focus on a white pediatric population.²² African American pediatric patients are at higher risk for asthma and have different response to corticosteroids therapy.^{23,24} Our study is one of the first to provide insight into the comparative efficacy of IV corticosteroids used to treat ASA in African American children in the PICU setting. Although IV corticosteroids are used in many intensive care units as the standard of care in ASA management, their efficacy has not been as well studied in this setting. There is even less research comparing the efficacy of different IV corticosteroids in PICU patients.²⁵

A survey of pediatric intensivists in North America showed that methylprednisolone was their preferred choice (96%) in the management of ASA in the PICU. Eighty-five percent of responders reported that their choice of dosing was purely based on their clinical experience. The doses of methylprednisolone reported by pediatric intensivists in this survey were 4 mg/kg/d (66%), 2 mg/kg/d (31%) and 1 mg/kg/d (3%).²⁶ A Cochrane review by Maser et al. analyzed the data of 344 adult patients with ASA treated with corticosteroids. Included in the analysis were methylprednisolone, hydrocortisone, dexamethasone, prednisone, prednisolone, betamethasone and triamcinolone delivered via different routes such as oral, intramuscular (IM) or IV. Doses used in the

analysis were made equivalent to methylprednisolone for a 24 h period and divided into 3 groups: Low dose ($\leq 80\text{mg/day}$), medium dose ($80\text{--}360\text{mg/day}$) and high dose ($>360\text{ mg/d}$). Predicted FEV1 was assessed in each group at 24, 48 and 72 h. Results showed no clinical differences between low, medium, and high dose corticosteroid groups. The side effect profile and respiratory failure rates were similar amongst the 3 dosing groups.²⁷ Lung function tests (FEV1 or PEF) recommended by the 2007 NIH Asthma Guidelines for children 5 years and older are useful tools to monitor the clinical response during an exacerbation. However, their determination in critically ill asthmatic children is not practical due to lack of cooperation which makes the test difficult to perform.²

Dexamethasone has been studied in recent years, gaining popularity in the ED setting and may become a valid option in the PICU as well, although data on its use in the PICU is limited. ED pediatric patients with asthma exacerbation who receive dexamethasone orally or IM constitute a different population, with the majority of these children being discharged and treated as outpatients.

There is scarce literature on the use and effectiveness of hydrocortisone for ASA. In a study published in 1974, Pierson et al. used injectable hydrocortisone, dexamethasone and betamethasone for ASA in children and found a beneficial effect of all 3 corticosteroids compared to a placebo group in terms of improvement in arterial hypoxemia. All 3 corticosteroids were well tolerated with no significant adverse effects.¹⁷ Raimondi et al. compared high (80 mg/kg/day) versus moderate (6 mg/kg/day) dose IV hydrocortisone given to 40 adult patients with ASA every 6 h for 5 days. Their results showed no significant differences between the 2 study groups with spirometric evaluation.²⁸

The lack of differences in the effectiveness seen among the 3 agents in our study could be explained by the fact that a sufficient anti-inflammatory effect was achieved by all 3 agents when given at the doses and duration utilized. Hydrocortisone, the most structurally similar to endogenous cortisone, is the weakest agent in terms of glucocorticoid potency. The statistically significant higher MAP in the hydrocortisone group, compared to the methylprednisolone and dexamethasone groups ($p = 0.036$), could be explained by the accompanying higher mineralocorticoid effect of hydrocortisone. The stronger mineralocorticoid effect of hydrocortisone makes it a less desirable option for the treatment of ASA when compared to methylprednisolone and dexamethasone. Dexamethasone may be gaining more popularity going forward because of its longer half-life. It has the advantage of requiring only 3 days of therapy to achieve a similar response, compared to a longer duration with methylprednisolone and hydrocortisone.

In terms of the cost-benefit analysis for the 3 IV corticosteroids we used, the average wholesale price for the 3 agents is comparable per vial and is of relatively low cost.²⁹ We also did not find a difference among the 3 groups in the terms of the median PICU LOS and hospital LOS (1.4 days and 2.8 days respectively) which are the main drivers of hospital associated costs. Both PICU LOS and hospital LOS in our study were comparable to Hartman's published review of inpatients trends of children admitted for ASA.³⁰

A limitation of our study is it being a prospective non-blinded study, which could have resulted in bias. Future studies with double blinded designs are needed to solidify the results. Additionally, the power study was done on duration of beta 2 agonist and future studies with higher power is needed to detecting difference among the 3 IV corticosteroids in the secondary variables. Furthermore, our study did not control for corticosteroids received in the ED, which may have impacted the PICU course. Future multidisciplinary studies would be required to control for management beyond the PICU. The determination and change of FEV1 would be more objective in assenting response to treatment but was not practical given the age of most of our patients.

Conclusion

To date, there is no objective evidence of the superiority of one IV corticosteroid over the other. Based on our study, IV methylprednisolone, dexamethasone and hydrocortisone are safe and have equivalent efficacy when used at the appropriate doses. The choice of which of these IV corticosteroids are to be used in the treatment of ASA can continue to be based of the physician's preference. However, in the case of hydrocortisone, special attention needs to be given to the risk for higher blood pressure, due to its higher mineralocorticoid effects. Given common practice, it is likely that methylprednisolone, and dexamethasone to a lesser extent, will continue to be the preferred choices for the management of ASA in the PICU setting. Larger studies with a more diverse population are needed to compare the effectiveness of IV corticosteroids in the management of ASA in the PICU setting.

- We received no funding for our research study.
- This study was performed in our institution's pediatric intensive care unit.
- All study participants provided informed consent, and the study design was approved by the appropriate ethics review board. We have read and understood your journal's policies, and we believe that neither the manuscript nor the study violates any of these.

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Table 1: Comparison of the baseline demographics and clinical characteristics of Group A (Intravenous Methylprednisolone), Group B (Intravenous Hydrocortisone) and Group C (Intravenous Dexamethasone), n = number of patients, % = Percentage, SD = Standard deviation, Body Mass Index (BMI) percentile calculated using CDC growth for age and sex chart.

Patient Characteristics	A (n = 22)	B (n = 20)	C (n = 19)	P-value
Number of patients (%)	22 (36.1)	20 (32.8)	19 (31.1)	
Age in years, median (QR)	7.29 (3.82, 10.5)	7.13 (4.9, 10)	6 (3.7, 8.6)	0.550
Gender (female) n (%)	8 (36)	12 (60)	9 (47)	0.318
Race, n (%)				0.229
• African American, n (%)	21 (95.5)	16(80)	17 (89.5)	
• Asian, n (%)	0	0	1 (5.2)	
• Hispanic, n (%)	1 (4.5)	4 (0.2)	1 (5.2)	
Body Mass Index, median (QR) (kg/m²)	21.35 (15.9, 25.5)	20.3 (16.9, 22)	16.3 (15, 18.5)	0.024
BMI percentile, median (QR)	96.1 (55.9, 98.8)	92.5 (59.5,99.3)	58 (24.1,90.7)	0.068
Asthma characteristics				
Newly diagnosed and intermittent asthma, n (%)	9 (40.9)	10 (50)	9 (47.4)	0.965
Persistent asthma (mild, moderate and severe), n (%)	13 (59.1)	10 (50)	10 (52.6)	0.761
Use of controllers, n (%)	11 (50)	11 (55)	5 (26.3)	0.157
Pediatric asthma severity score at admission, median (QR)	10.5(9,12)	11.5(10,12)	12(10,13)	0.355

Table 2: Comparing outcomes of patients with acute severe asthma in Group A (Intravenous Methylprednisolone), Group B (Intravenous Hydrocortisone) and Group C (Intravenous Dexamethasone). QR= Quartile range 25–75 percentile, n = Number of patients, % = Number of patients in percentage, PICU = Pediatric Intensive Care Unit, LOS = Length of Stay, bpm = Beats per Minute).

Variables	A (n = 22)	B (n = 20)	C (n = 19)	p-value
Duration of beta-agonist (hours), median (QR)	23 (15.75,37.75)	26.75 (16, 39.75)	32 (16,47.5)	0.900
Weaning beta-agonist dose from 10 to 5 mg/hour, median time (hours) (QR)	18 (18,33)	18 (12,24)	21 (13.5,24)	0.517
PICU LOS (hours), median (QR)	30 (20,38.25)	36.5(29.75,53.5)	36 (28,49)	0.247
Hospital LOS (hours), median (QR)	62 (40,112)	68.5 (47.5,93.75)	71(50,102.5)	0.922
Glucose level, median (QR) (mg/dl)	137.4(124-163)	141(129,151)	150(138,175)	0.343
Vital Signs				
Heart rate (bpm), mean (SD)	131 (16)	133 (12.3)	136 (19.3)	0.573
Systolic blood pressure (mmHg), mean (SD)	109 (8.22)	112 (7.95)	107 (8.33)	0.107
Diastolic blood pressure (mmHg), mean (SD)	53.1 (7.23)	58.8 (9.57)	53.7 (7.03)	0.054
Mean arterial pressure (mmHg), mean (SD)	71.6 (6.54)	76.5 (8.21)	71.3 (6.71)	0.041
Maximum dose of continuous beta-2 agonist treatment				
5-10 mg/hour, n (%)	20 (90.9)	18 (90)	18 (94.7)	0.931
15 –20 mg/hour, n (%)	2 (9.1)	2 (1)	1 (5.3)	0.819
Need for respiratory support n (%)				
No respiratory support	16 (72.7)	11 (55)	6 (31.6)	0.103
High flow nasal cannula	0	1 (5)	3 (15.8)	0.317
Bi-level positive airway pressure	6 (27.3)	8 (40)	10 (52.6)	0.607

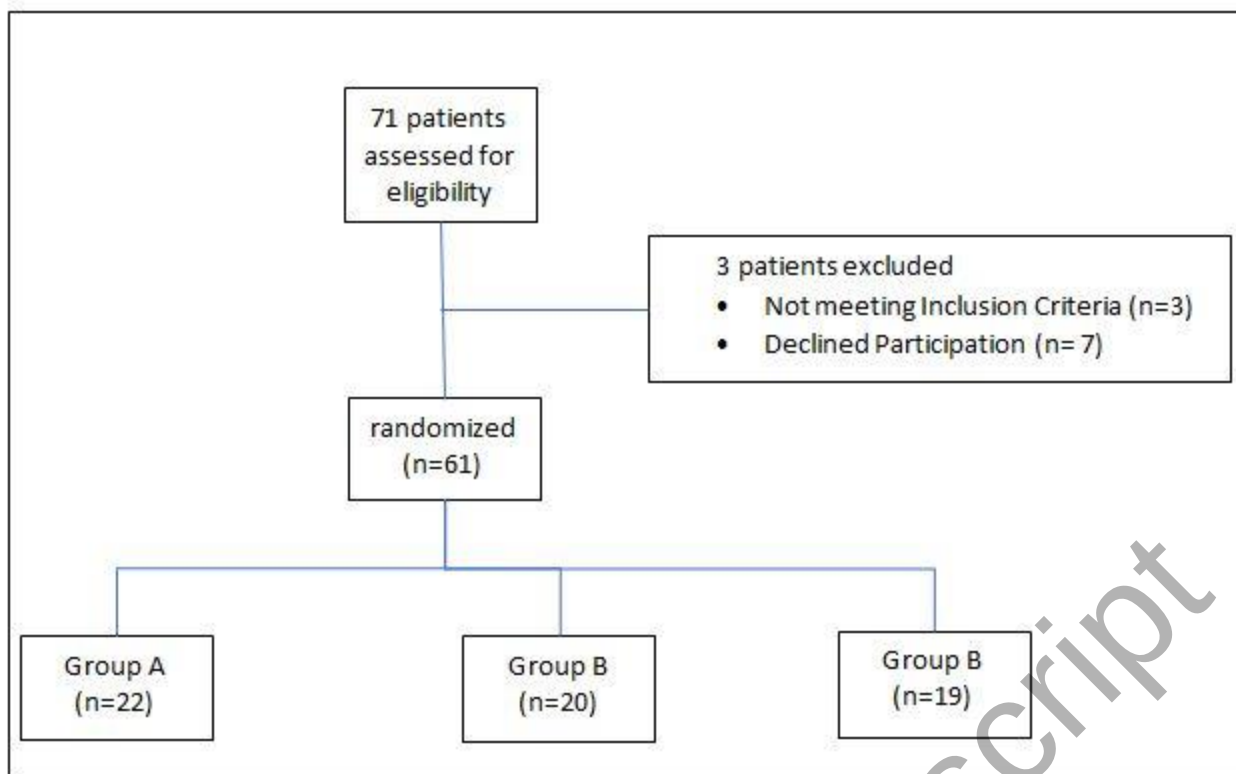


Figure 1: Consort Diagram.

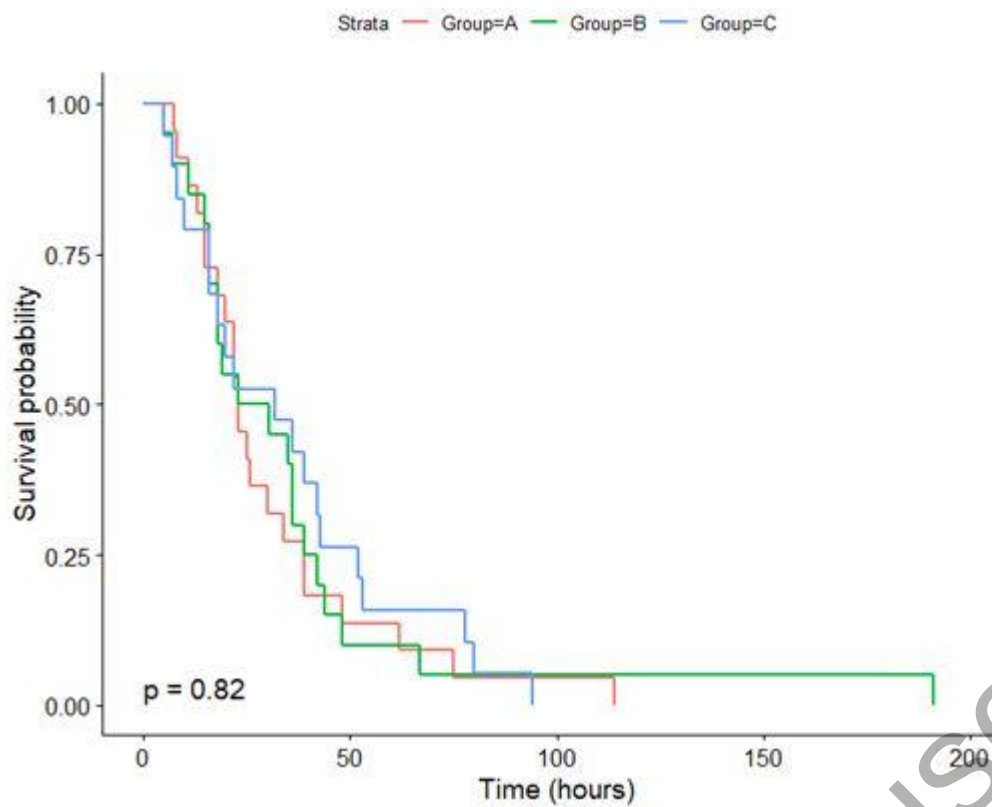


Figure 2: Kaplan Meier curve and log rank test for time to discontinuation of continuous beta 2 agonist treatment.