Published in final edited form as:
Pediatr Blood Cancer. 2011 February ; 56(2): 262–266. doi:10.1002/pbc.22808.

Multi-Modal Intervention for the Inpatient Management of Sickle Cell Pain Significantly Decreases the Rate of Acute Chest Syndrome

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Abstract

Introduction—Pain in children with sickle cell disease (SCD) is the leading cause of acute care visits and hospitalizations. Pain episodes are a risk factor for the development of acute chest syndrome (ACS), contributing to morbidity and mortality in SCD. Few strategies exist to prevent this complication.

Methods—We performed a before-and-after prospective multi-modal intervention. All children with SCD admitted for pain during the two-year study period were eligible. The multi-modal intervention included standardized admission orders, monthly house staff education, and one-on-one patient and caregiver education.

Results—A total of 332 admissions for pain occurred during the study period; 159 before the intervention and 173 during the intervention. The ACS rate declined by 50% during the intervention period 25%, (39 of 159) to 12%, (21 of 173); p=0.003. Time to ACS development increased from 0.8 days (0.03–5.2) to 1.7 days (0.03–5.8); p=0.047. No significant difference was found in patient demographics, intravenous fluid amount administered, frequency of normal saline bolus administration, or cumulative opioid amount delivered in the first 24-hours. Patient controlled analgesia (PCA)-use was more common after the intervention 52%, (82 of 159) vs 73%, (126 of 173; p=0.0001) and fewer patients required changes in analgesic dosing within the first 24-hours after admission (26%, 42 of 159 vs 16%, 28 of 173; p=0.015).

Conclusions—A multi-modal intervention to educate and subsequently change physician behavior likely decreased the rate of ACS in the setting of a single teaching hospital.

Keywords

Acute Chest Syndrome; Sickle Cell Pain; Sickle Cell Disease; Children

Introduction

Sickle cell-related pain is the most common morbidity experienced by children and adults with sickle cell disease (SCD). Acute chest syndrome (ACS), though second to SCD-pain in frequency, contributes significantly to both morbidity and mortality in children with SCD.

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ACS is responsible for up to one-quarter of sickle cell-related mortality(1), and almost half of deaths due to ACS are in children less than 20 years of age(2). Furthermore, ACS during childhood negatively impacts long-term lung function(3), and predisposes children to subsequent episodes(4,5). A report from the Cooperative Study of Sickle Cell Disease found that three-quarters of patients who died from ACS initially presented with lower extremity pain due to sickle cell vaso-occlusion, and that death from ACS primarily occurred within 48 hours of symptom onset. For patients who did not die, 50% of adults and 11% of children presented with SCD-pain prior to the onset of ACS(2). Consequently, a close temporal relationship exists between ACS and SCD-pain.

The pathophysiology of ACS is not fully understood, yet multiple risk factors have been identified including splenectomy(6), abdominal surgery(7), asthma(4), infection(8), fat emboli and pain(9). Therapies to prevent the development of ACS are limited and risk factor dependent. For example, pre-operative blood transfusion reduces the rate of ACS in the surgical setting(10) as does the use of laparascopic rather than open abdominal procedures(11). The use of controller medications in patients with asthma(12) and hydroxyurea in patients with frequent episodes of ACS reduces ongoing risk, but these therapies are not used to treat an acute episode(13). Additionally, the use of incentive spirometry reduces the development of pulmonary complications in children admitted with pain(14). Lastly, in a retrospective study that examined the frequency of ACS development, patients who received nalbuphine, a mixed opioid agonist-antagonist, compared to morphine had a lower rate of ACS(15).

In this study, we evaluated the impact of a multi-modal intervention combining standardized orders and education for the inpatient management of SCD-pain on the rate of subsequent ACS episodes. The use of incentive spirometry substantially decreases the rate of ACS among individuals hospitalized for pain (15), though we observed that many patients are not compelled to consistently use incentive spirometry while hospitalized. We, therefore, tested the hypothesis that a standard treatment for pain episodes including specific orders to nursing staff regarding monitoring incentive spirometry would decrease the rate of ACS in patients admitted with SCD.

Methods

The protocol was approved by the Washington University School of Medicine Human Research Protection Office. A prospective cohort study was performed to assess the effect of a multi-modal intervention aimed at reducing 30-day hospital readmission for children admitted with SCD-pain. Secondary outcome measures obtained in the cohort study included the development of ACS and time to onset of ACS, a SCD-pain related morbidity. The results of the primary analysis of 30-day readmission rate and a detailed description of the multi-modal intervention including the specific order sets were published previously(16). The multi-modal intervention combined the use of standardized order sets and physician and patient/caregiver education. A physician with expertise in SCD was available daily and immediately via a phone call to answer house staff questions regarding the order sets. Pertinent to the current publication, the multimodal order sets included: specified patient monitoring with criteria for the initiation of supplemental oxygen; scheduled incentive spirometry; patient ambulation after the first 24 hours; opioid type and dosing schedule, maintenance and exacerbation asthma orders, and opioid titration and weaning guidelines.

All hospital admissions for children with SCD-pain were evaluated prospectively for 12 months from July 1, 2007 to June 30, 2008. Inclusion criteria were any diagnosis of SCD as confirmed by hemoglobin analysis; age > 12 months, as pain requiring intravenous opioid administration is rarely the primary reason for admission in young children; and admission

to the inpatient unit for further pain management. Exclusion criteria were the following: pain not requiring use of intravenous (IV) opioids, Group A β-hemolytic Streptococcus infection, costochondritis, or priapism as the primary reason for admission; and ≥ 12 hospitalizations for SCD-related morbidity in the previous 12 months. A similar seasonal time period prior to the intervention, July 1, 2006 to June 30, 2007, was chosen for the control cohort. The same inclusion and exclusion criteria were used to identify patients in the control cohort.

Definitions

Acute chest syndrome was defined as a new infiltrate on chest radiography in a patient with any form of SCD and one or more of the following symptoms: fever (T≥38.5°C), cough, chest pain, sputum production, respiratory distress (dyspnea, increased work of breathing, tachypnea), or hypoxia. SCD-pain was defined as acute pain in the extremities, back, abdomen, or chest with no other identified cause other than SCD. Disease severity was defined as patients with ≥ 3 hospitalizations for SCD-related morbidity in a one-year period. Asthma symptoms during admission were defined as the presence of chest pain, cough, hypoxia, wheezing, respiratory distress such as tachypnea or increased work of breathing, or decreased breath sounds in a patient with a history of asthma. Incentive spirometry was performed using a standard personal patient incentive spirometer. PEB and bubble therapy was not employed.

Covariates

Risk factors of interest were defined *a priori* and included four major categories: presence of asthma, presence of decreased oxygen saturation, the type and amount of opioid medications, amount of intravenous fluid (IVF). Specifically we considered a prior diagnosis of asthma and/or asthma symptoms on admission, as well as steroid treatment during hospitalization; type of opioid administered such as on demand administration by a nurse, intermittent scheduled, or patient controlled analgesia (PCA); escalation of opioid therapy in the first 24 hours, defined as positive change in opioid brand, type or schedule; cumulative opioids in morphine equivalents per kilogram administered in the first 24 hours; room air oxygen saturation on admission and initiation of supplemental oxygen therapy; the amount of IVF per m^2 received in the first 24 hours and administration of a 20ml/kg IVF bolus in the emergency department.

Data Collection Methods

Prospective data were obtained directly from inpatient medical records and from the hospital-based computer system using an a priori data collection tool by two extractors, one physician and one research assistant. Double-data entry was performed and 10% of the patient charts were evaluated for concordance between extractors, with 97% concordance found in demographic data and baseline characteristics. The concordance rate for ACS diagnosis was 89%. The physician extractor identified more cases of ACS than the research assistant. Each additional case was reviewed and the discrepancy found to be due to the interpretation of radiology reports and chest radiography images. Baseline characteristics including type of SCD, co-morbid diagnoses, steady-state hemoglobin level, and chronic medications were collected for each patient. For each admission, the following were collected: time to diagnosis of ACS, cumulative amount of intravenous fluids, presence of asthma symptoms, cumulative opioids, steroid administration, presence of fever, viral testing, antibiotic administration, chest X-ray findings, use of oxygen, and discharge oxygen saturations.

Statistical Methods

The statistical software package, SPSS 16.0.0, was used to perform statistical analysis. Dichotomous primary and secondary outcomes were analyzed using Chi square and Fisher's Exact Test to compare the outcomes of interest between groups before and after the intervention. Continuous variables were analyzed using Student's T-test. Baseline characteristics of the cohort are presented in descriptive tables. Odds ratios and 95% Confidence Intervals were calculated where appropriate. Multivariable analysis using logistic regression modeling was performed with development of ACS as the categorical dependent variable. All covariates postulated *a priori* to be important in the development of ACS defined in the methods section were included(17). The Hosmer-Lemeshow goodness of fit was used to assess the model. Initial model building was performed using all covariates that were identified a priori as potential risk factors for ACS. Covariates that met criteria for exclusion were removed individually by backward selection creating the final model. Criteria to exclude a covariate was a significance of 0.2(18). Reduction in the rate of ACS after the intervention was a secondary outcome defined *a priori*. Thus a p value of less than 0.05 was considered marginal evidence, a p value of less than 0.05 and greater than 0.01 was consider moderate evidence, and a p value of less than 0.01 was considered strong evidence to support our hypothesis.

Results

Study Population for Prospective and Control Cohorts

The control cohort was composed of 159 inpatient admissions for SCD-pain, occurring among 88 individual patients. The prospective cohort included a total of 173 inpatient admissions for pain among 102 individual patients. Standard order sets were used in 27% (43 of 159) of admissions in the control cohort due to overlap with a three-month pharmacy run-in and 94% (162 of 173) of admissions in the prospective cohort during the multi-modal intervention. Demographic data for the individual patients in the intervention and control cohorts are presented in Table I. No significant difference in baseline characteristics was found between the two cohorts.

Characteristics of Acute Chest Syndrome Development Between Cohorts

The rate of acute chest syndrome declined by 50% between the control and intervention time periods, falling from 25% (39 of 159) to 12% (21 of 173; p=0.003). Time to development of ACS increased between the control and intervention cohorts, from 0.8 days (0.03–5.2) to 1.7 days (0.03–5.8; p=0.047; 95% CI −1.9373 to −0.0118), but the difference did not reach statistical significance. The frequency of chest x-ray acquisition was 71% (113 of 159) in the control cohort and 76% (131 of 173; p=0.4) in the prospective cohort.

Assuming oxygen therapy and transfusion as markers of severity of ACS episode, no significant difference existed in severity of ACS between the two time periods. Oxygen therapy was administered in 85% (33 of 39) of control patients with ACS versus 67% (14 of 21) of prospective cohort, (95% CI −0.0364– 0.6667; p=0.2). Simple transfusion was given to 49% (19 of 39) of patients in the control cohort versus 43% (9 of 21) in the prospective control cohort, (95% CI -0.1953- 0.2964; p=0.86). Only one patient in the control and one in the prospective cohort received erythrocytapheresis; data are presented in Table II. The rate of ACS per month is depicted in Figure 1. The peak rate of ACS development in the control cohort occurred in April, August, and September, while the peak rate in the intervention cohort occurred in March, June and July. Seasonal influenza peaked in February for both time periods for Region 7 (Iowa, Kansas, Missouri, and Nebraska) as reported by the Centers for Disease Control and Prevention(19).

Risk Factors for the Development of Acute Chest Syndrome

The following covariates met *a priori* criteria for exclusion: supplemental oxygen use, asthma symptoms on admission, amount of IVF received in the first 24 hours, type of opioid administration, amount of opioids per kg received, administration of a bolus in the ED, and steroid administration. The final model included baseline oxygen saturation, typically greater than 92% on room air (p=0.01; OR 0.85, 95% CI 0.75–0.96), a history of asthma (p=0.002; OR1.3, 95% CI 1.1–1.6), and an escalation of pain medications within the first 24 hours of admission (p=0.47; OR 2.6 95% CI 1–7.1). R square was 0.22.

Discussion

The etiology of ACS in children with SCD is multi-factorial. Recent evidence strongly suggests that medical care at the bedside with the use of incentive spirometry and choice of opioids can significantly decrease the rate of ACS. We have identified yet another modifiable risk factor for ACS, the implementation of a multi-modal program to improve the inpatient treatment of SCD-pain. The intervention employed evidence-based standardized SCD-pain admission orders and systematic education to promote physician and patient and caregiver behavior change. The goal of the admission orders was to prevent delays in pain relief due to physician inexperience and provide more rapid and adequate pain control early in the hospital admission.

The standardized orders were created following the most recent evidence-based guidelines for inpatient pain management and included treatments that have been shown previously either to decrease ACS development during pain such as incentive spirometry(20) or have been hypothesized to be based on the underlying pathophysiology of SCD, such as asthma(4). However, the impact of incentive spirometry is limited by physician ordering as noted by Co et al., who found only 40% of patients received incentive spirometry compared to 100% of patients admitted after implementing a clinical pathway prompting physicians to order spirometry(21). Supportive therapy was tailored based on the diagnosis of asthma as lower airway obstruction has been linked to increased rates of pain and ACS(22). Our results provide further validation of asthma as a risk factor for ACS(4,22,23). We have provided evidence that reduction in ACS rate was likely to be a result of the multi-modal nature of the intervention and the underlying improvement in the inpatient treatment of SCD-pain.

highlight the first 24 hours of hospitalization, at least in our hospital, as a critical time period for intervention to prevent the development of ACS above and beyond the standardized therapies included in our admission orders. This time to onset of ACS has previously been described as occurring much later than the first 24 hours. Vichinsky et al. described the development of ACS a mean of 2.5 days after admission for pain(9) which parallels an earlier report by Bellet et al. which found an abnormal chest x-ray 2.4 days after admission for pain in patients not receiving incentive spirometry(14). Similarly, low oxygen saturations are associated with an increase risk of ACS, as oxygen requirement and increased work of breathing are considered part of the definition of ACS.

Our study has several limitations. During the both the control and interventional study period, nursing staff were not required to directly observe incentive spirometry use by patients. However, incentive spirometry was an “opt out” order as part of the standardized order sets which lead to its automatic ordering. The reduction of ACS may entirely be related to the fact that incentive spirometry was consistently ordered from the time of admission during the intervention cohort. In addition, the study was continued for only one year and the decrease in ACS rate may have been due to differences in the prevalence of viral infections between the consecutive time periods, though influenza rate was the same between groups. Further, the intent of the original intervention was to decrease the rate of re-

hospitalizations for pain within 30 days of the admission and not to decrease the rate of ACS episodes. Given the possibility that our secondary analysis would provide spurious results(24), prior to the start of the study, we selected a more conservative level of significance of 0.01. The actual p value for the hypothesis was 0.003, well below the more conservative level of significance chosen.

The use of a multi-modal intervention to improve SCD-pain management successfully altered patient outcomes by reducing the rate of ACS development during pain treatment. The feasibility of continuing such a program as well as the application of a time-intensive program in smaller institutions has not been determined. The investment needed to maintain the intervention may be cost-effective by decreasing morbidity related to SCD-pain.

Acknowledgments

Melissa Frei-Jones, MD MSCI participated in the following NIH funded programs at Washington University School of Medicine.

UL1RR024992 - (09/17/2007 to 06/30/2008) Polonsky (PI)

TL1RR024995 - (09/17/2007 to 06/30/2008)

K30 RR022251 - (07/01/2006- 09/16/2007) Evanoff (PI)

NIH/NCRR

Washington University Institute of Clinical and Translational Sciences (PI: Polonsky)

Research Education, Training and Career Development (Co-PI: Fraser)

The Clinical and Translational Science Award provides support to establish the W U Institute of Clinical and Translational Sciences (ICTS). By implementing 15 key Program Functions, the ICTS operationally reinvents clinical and translational research and clinical research training at WU and its regional partners. The Education sub-project of the ICTS integrates and enhances existing training programs, develops new clinical and translational courses, promotes multidisciplinary team training and provides improved tracking and evaluation for all clinical research training programs.

References

1. Platt OS, Brambilla DJ, Rosse WF, Milner PF, Castro O, Steinberg MH, et al. Mortality in sickle cell disease. Life expectancy and risk factors for early death. N Engl J Med 1994;330(23):1639–1644. [PubMed: 7993409]
2. Vichinsky EP, Styles LA, Colangelo LH, Wright EC, Castro O, Nickerson B. Acute chest syndrome in sickle cell disease: clinical presentation and course. Cooperative Study of Sickle Cell Disease. Blood 1997;89(5):1787–1792. [PubMed: 9057664]
3. Sylvester KP, Patey RA, Milligan P, Rafferty GF, Broughton S, Rees D, et al. Impact of acute chest syndrome on lung function of children with sickle cell disease. J Pediatr 2006;149(1):17–22. [PubMed: 16860119]
4. Boyd JH, Macklin EA, Strunk RC, DeBaun MR. Asthma is associated with acute chest syndrome and pain in children with sickle cell anemia. Blood 2006;108(9):2923–2927. [PubMed: 16690969]
5. Castro O, Brambilla DJ, Thorington B, Reindorf CA, Scott RB, Gillette P, et al. The acute chest syndrome in sickle cell disease: incidence and risk factors. The Cooperative Study of Sickle Cell Disease. Blood 1994;84(2):643–649. [PubMed: 7517723]
6. Ghantous S, Al Mulhim S, Al Faris N, Abushullaih B, Shalak F, Yazbeck S. Acute chest syndrome after splenectomy in children with sickle cell disease. J Pediatr Surg 2008;43(5):861–864. [PubMed: 18485954]
7. Kokoska ER, West KW, Carney DE, Engum SE, Heiny ME, Rescorla FJ. Risk factors for acute chest syndrome in children with sickle cell disease undergoing abdominal surgery. J Pediatr Surg 2004;39(6):848–850. [PubMed: 15185210]

8. Neumayr L, Lennette E, Kelly D, Earles A, Embury S, Groncy P, et al. Mycoplasma disease and acute chest syndrome in sickle cell disease. Pediatrics 2003;112(1 Pt 1):87–95. [PubMed: 12837872]
9. Vichinsky EP, Neumayr LD, Earles AN, Williams R, Lennette ET, Dean D, et al. National Acute Chest Syndrome Study Group. Causes and outcomes of the acute chest syndrome in sickle cell disease. N Engl J Med 2000;342(25):1855–1865. [PubMed: 10861320]
10. Vichinsky EP, Haberkern CM, Neumayr L, Earles AN, Black D, Koshy M, et al. The Preoperative Transfusion in Sickle Cell Disease Study Group. A comparison of conservative and aggressive transfusion regimens in the perioperative management of sickle cell disease. N Engl J Med 1995;333(4):206–213. [PubMed: 7791837]
11. Alwabari A, Parida L, Al-Salem AH. Laparoscopic splenectomy and/or cholecystectomy for children with sickle cell disease. Pediatr Surg Int 2009;25(5):417–421. [PubMed: 19370356]
12. The Childhood Asthma Management Program Research Group. Long-term effects of budesonide or nedocromil in children with asthma. N Engl J Med 2000;343(15):1054–1063. [PubMed: 11027739]
13. Charache S, Terrin ML, Moore RD, Dover GJ, Barton FB, Eckert SV, et al. Effect of hydroxyurea on the frequency of painful crises in sickle cell anemia. Investigators of the Multicenter Study of Hydroxyurea in Sickle Cell Anemia. N Engl J Med 1995;332(20):1317–1322. [PubMed: 7715639]
14. Bellet PS, Kalinyak KA, Shukla R, Gelfand MJ, Rucknagel DL. Incentive spirometry to prevent acute pulmonary complications in sickle cell diseases. N Engl J Med 1995;333(11):699–703. [PubMed: 7637747]
15. Buchanan ID, Woodward M, Reed GW. Opioid selection during sickle cell pain crisis and its impact on the development of acute chest syndrome. Pediatr Blood Cancer 2005;45(5):716–724. [PubMed: 15926170]
16. Frei-Jones MJ, Field JJ, DeBaun MR. Multi-modal intervention and prospective implementation of standardized sickle cell pain admission orders reduces 30-day readmission rate. Pediatr Blood Cancer 2009;53(3):401–405. [PubMed: 19422031]
17. Harrell, FJ. Regression Modeling Strategies: with applications to linear models, logistic regression, and survival analysis. New York: Springer; 2001.
18. Mantel N. Why step-down procedure in variable selection. Technometrics 1970;12:621–625.
19. Atlanta: Centers for Disease Control and Prevention; 2009. The Flu Season.
20. Merrill WW. Incentive spirometry in sickle cell crisis. N Engl J Med 1996;334(2):124–125. [PubMed: 8531956]
21. Co JPT, Johnson KB, Duggan AK, Casella JF, Wilson M. Does a clinical pathway improve the quality of care for sickle cell anemia? Joint Com J Qual and Safety 2003;29(4):181–190.
22. Boyd JH, DeBaun MR, Morgan WJ, Mao J, Strunk RC. Lower airway obstruction is associated with increased morbidity in children with sickle cell disease. Pediatr Pulmonol 2009;44(3):290–296. [PubMed: 19205057]
23. Knight-Madden JM, Forrester TS, Lewis NA, Greenough A. Asthma in children with sickle cell disease and its association with acute chest syndrome. Thorax 2005;60(3):206–210. [PubMed: 15741436]
24. DeBaun MR, Field JJ. Limitations of clinical trials in sickle cell disease: a case study of the Multi-center study of Hydroxyurea (MSH) trial and the Stroke Prevention (STOP) trial. Hematology Am Soc Hematol Educ Program 2007:482–488. [PubMed: 18024668]

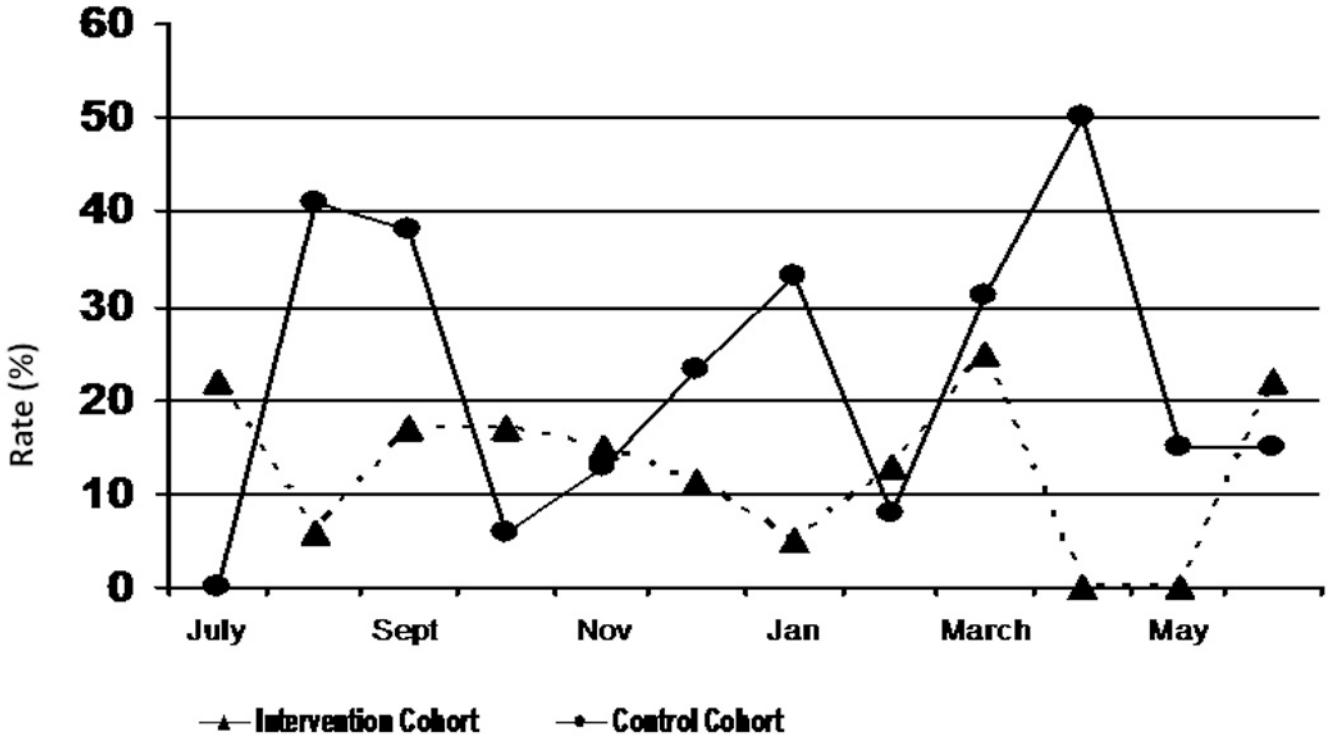

Figure 1.

Table I

Demographics and clinical characteristics of the intervention and control cohort of children with sickle cell disease admitted to St. Louis Children's Hospital for an initial painful episode during two separate 12 month periods

	Intervention Cohort n=102	Control Cohort n=88	p-value
Mean Age (range in years)	11.4 (1–20)	11.5 (1–20)	0.84
Gender (males)	48 (47%)	45(51%)	0.66
SCD Genotype			
SS	65 (64%)	64 (72%)	0.21
SC	29	18	0.24
$S\beta^+$ Thalassemia	4	2	0.69
$S\beta^o$ Thalassemia	3	4	0.71
SHPFH	1	0	1
Co-morbidities			
Asthma	46 (45%)	43 (49%)	0.66
Stroke	14 (14%)	10 (11%)	0.67
Hydroxyurea	16 (16%)	8 (9%)	0.2
Baseline Labs			
Hemoglobin (gm/dl)	9.6	9.2	0.09
Reticulocyte count	10.5%	8.9%	0.05
Room Air Oxygen Saturations	98%	96.5%	0.13
Mean Hospitalizations in Previous 12 months (range)	2.1 (0–10)	2.3 (0–10)	0.55
Insurance Status			
Medicaid or other State-sponsored Insurance	73 (72%)	66 (75%)	0.63

Table II

Demographics and clinical characteristics of the intervention and control cohort of children with sickle cell disease admitted to St. Louis Children's Hospital for an initial painful episode and subsequently developed acute chest syndrome during two separate 12 month periods.

	Intervention Cohort n=21	Control Cohort n=39	p-value
Mean Age (range in years)	11.7 (1–19)	10.1 (1–20)	0.27
Gender (males)	10 (48%)	19 (49%)	1
SCD Genotype			
SS	18 (86%)	34 (87%)	1
SC	2 (10%)	2 (5%)	1
Sβ+ Thalassemia	0	2 (5%)	0.54
Sβ^o Thalassemia	1 (4%)	1 (3%)	1
Co-morbidities			
Asthma	10 (48%)	19 (49%)	1
Sleep Apnea	0	2 (5%)	0.54
Nocturnal Oxygen at Home	1 (4%)	2 (5%)	1
Stroke	5 (24%)	7 (18%)	0.74
Hydroxyurea	4 (19%)	4 (10%)	0.43
Baseline Labs			
Hemoglobin (gm/dl)	8.5	8.5	0.97
Reticulocyte Count	12.3%	12.1%	0.94
Room Air Oxygen Saturations	98%	95%	0.32
Mean Hospitalizations in Previous 12 Months (range)	3 (0–10)	2.7(0–10)	0.67
ACS Episode			
Asthma Symptoms	10 (48%)	16 (41%)	0.82
Hypoxia	14 (67%)	33 (85%)	0.2
Fever	15 (71%)	30 (78%)	0.76
Influenza	0	0	1
Simple Transfusion	9 (43%)	19 (49%)	0.86
Erythrocytapheresis	1 (5%)	1 (3%)	1
Mean Length of Stay in Days (range)	7 (3.8–10)	6.1 (1.9–10.3)	0.44
Mean Intravenous Fluid (ml/m^2)	1520	1550	0.64
Mean Admission Pain Score	8.7 (6–10)	8.4 (3–10)	0.65