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Effect of Sevoflurane Pretreatment on Inflammatory Response and Lung Ventilation in Neonates Undergoing Thoracoscopic Correction of Type III Esophageal Atresia

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HIGHLIGHTS

- To research the influence of sevoflurane pretreatment on neonatal inflammatory response and pulmonary ventilation.
- 60 neonates randomized into 2 groups, group C, group S.
- A concentration of 2% sevoflurane pretreatment may exist a protective influence on neonatal inflammatory response.

Abstract: Neonatal thoracoscopic correction of type III esophageal atresia requires the establishment of an artificial carbon dioxide pneumothorax in the affected thoracic cavity. At present, there is no suitable double-lumen endotracheal tube or bronchial occluder to be used in neonatal thoracoscopic surgery. Objectives: To research the influence of sevoflurane pretreatment on neonatal inflammatory response and pulmonary ventilation. Sixty neonates were randomized into 2 groups, group C, group S. Neonates in group S received sevoflurane inhalation at a concentration of 2% after endotracheal intubation until the establishment of pneumothorax, followed by a 10-min washout period, while those in group C only inhaled pure oxygen. Venous blood samples were collected from the subjects in the 2 groups from at 10 min before induction of anesthesia to 24 h after pneumothorax relief to determine the concentrations of serum interleukin (IL)-6, IL-8, and tumor necrosis factor α. Arterial blood gases were drawn from the radial artery at the above time points. The alveolar-arterial oxygen partial pressure difference, and respiratory index were calculated. Concentrations of IL-6, IL-8, and tumor necrosis factor α in serum were all lower in group S. Alveolar-arterial oxygen and respiratory index were also lower in group S. Duration of surgical intensive care unit stay, time of mechanical ventilation, and length of stay were all lower in group S. A concentration of 2% sevoflurane

pretreatment may have a protective influence on the neonatal inflammatory response and pulmonary ventilation, which correct type III esophageal atresia by suppressing the inflammatory response under the thoracoscope.

Keywords: Sevoflurane; Pretreatment; Inflammatory response; Oxygenation; Pulmonary gas exchange.

INTRODUCTION

Thoracoscopic correction of esophageal atresia (EA) with tracheoesophageal fistula (TEF) was first performed live during an International Pediatric Endosurgery Group meeting in 1999, and then described by Zhang and coauthors [1]. Since then, this approach has become more widespread4–10 and, even if controversy exists among pediatric surgeons as to which is the best approach (thoracotomy versus thoracoscopy). In our opinion the minimally invasive repair of EA/TEF is expected to become the standard technique. Even though this approach is increasingly widespread, the procedure remains one of the most advanced pediatric surgical skills: there is a steep learning curve; it requires great experience in endocorporeal knotting. Particularly early in the surgeon's experience, it requires a longer operative time than the open approach [1].

Neonatal thoracoscopic correction of type III esophageal atresia requires the establishment of an artificial carbon dioxide pneumothorax in the affected thoracic cavity. At present, there is no suitable double-lumen endotracheal tube or bronchial occluder to be used in neonatal thoracoscopic surgery. Single-lumen endotracheal tube is still used for endotracheal intubation for one-lung ventilation in such surgery. This unsatisfactory mechanical ventilation will cause some adverse influence on neonatal respiratory function during the perioperative period [1]. Therefore, it is especially necessary to take appropriate preventive measures to protect the respiratory function of neonates. Sevoflurane is a new halogenated fluorine inhalation anesthetic that has been widely used in pediatric anesthesia. Previous evidence has revealed that sevoflurane has a certain organ protective effect on the lungs [2]. However, it has not been reported in clinical practice whether there was a protective role of sevoflurane pretreatment on type III esophageal atresia correction by thoracoscopy.

The objective of our research was to assess the influence of sevoflurane pretreatment on neonatal inflammatory response and pulmonary ventilation. Type III esophageal atresia was corrected with thoracoscopy by measuring the concentration of IL-6, IL-8, and TNF-α and the tension difference change of alveolar-arterial oxygen [P (A-a) DO2] and respiratory index (RI) before and after one-lung ventilation in children, providing a reference for better guidance of perioperative anesthesia management in children.

MATERIAL AND METHODS

Patient collection and grouping

The study was approved by our hospital ethics committee, and all the families of the neonates signed the informed consent. From February 2017 to June 2018, 60 neonates (39 males and 21 females) aged 1-28 d, weighed 1.4-3.9 kg, were classified Grade II-III according to the American Society of Anesthesiologists (ASA). Inclusion criteria: No preoperative pneumothorax; no serious metabolic disease; no immune system disease; no heart, lung, liver, kidney, or other organ dysfunctions; no recent cold, fever, cough, or expectoration; no history of drug allergy; no conversion to thoracotomy. All neonates were randomly divided into two groups, one was the control group (group C) and the other was the sevoflurane pretreatment group (group S). There were 30 patients in each group.

Exclusion criteria: Patients had the following diseases: preoperative pneumothorax, severe metabolic disease, and no immune system disease; Patients combined with cardiopulmonary, liver, kidney, or other organ dysfunction; Patients develop a cold, fever, cough, sputum and other symptoms; The patient had chest surgery and had a history of drug allergy，Patients with the above conditions weren't included of the group.

Anesthetic method the neonates were visited the day before surgery, and their families were instructed to perform fasting and abstain from drinking for 6 h. The neonates were given 0.02 mg/kg of penehyclidine hydrochloride intramuscularly 30 min before surgery. Ketamine (5 mg/kg) was injected intramuscularly before entry into the surgery room, followed by low-flow mask oxygen inhalation at 2-3 L/min. Electrocardiogram, pulse oximetry (SpO2), mean arterial pressure (MAP), and heart rate (HR) of patients were regularly monitored. A gas collection tube was placed at the nostril to monitor the end-tidal carbon dioxide partial pressure (PETCO2). After entering the surgery room, the peripheral vein was opened. Radial artery puncture and catheterization were performed. Arterial blood pressure was continuously monitored. The induction of

intravenous anesthesia was started after trocar puncture. Induction of anesthesia: Propofol 2.5 mg/kg, Sufentanil 1.0 μg/kg, rocuronium 0.45 mg/kg. After satisfactory anesthesia induction, endotracheal intubation and mechanical ventilation were performed. Ventilator parameters: tidal volume 8-10 ml/kg, ventilation frequency 30-40 times/min, inspiratory/expiratory ratio = 1:1.5, inspired oxygen concentration 100%. A concentration of 2 mg kg-1h-1 of propofol and 0.5 to 1.0 μg kg-1·min -1 remifentanil were given to maintain anesthesia. An artificial pneumothorax was established on one side of the procedure and the pressure was maintained at 6 mmHg, then the one-lung ventilation parameters during the artificial pneumothorax were set: tidal volume of 8-10 ml/kg, ventilation frequency of 30-40 times/min, I:E ratio = 1:1.5, positive end-expiratory pressure (PEEP) of 4-6 cmH2O (1cmH2O=0.098kPa), inspired oxygen of 100%, peak airway (Pmax) of 30 cmH2O, until the surgeon was satisfied with the collapse of the affected lung. After the surgeon sutured and severed the esophagotracheal fistula or returned the diaphragmatic hernia under the microscope, the neonates were immediately given cisatracurium 0.15 mg/kg intravenously, followed by 0.1-0.2 mg kg-1 min-1 pump to maintain muscle relaxation. The urine volume and body temperature of the neonates were monitored in real time during the operation, and the feeding rate and volume were adjusted according to the neonates and surgical conditions. When necessary, atropine 0.01-0.02 mg/kg was intravenously injected and dopamine 3-5 μg kg-1 min-1 was intravenously pumped to keep the neonate's intraoperative hemodynamics stable so that the fluctuation of HR and MAP did not exceed 20% of the baseline value. All neonates were transferred to the surgical intensive care unit (SICU) after surgery and continued on mechanical ventilation by usingendotracheal tube in the weasand. The endotracheal tube was removed until the newborn's lungs were in good condition.

Methods Neonates in group S received inhalation of sevoflurane at a concentration of 2% (batch number: 8Z02, Maruishi Pharmaceutical Co., Ltd., Japan) after endotracheal intubation until the establishment of pneumothorax, followed by a 10-min washout period, while those in group C received pure oxygen only. Methods of endotracheal intubation in the two groups of neonates: first insert the endotracheal tube into one side of the common bronchus, then auscultate the breath sounds of both lungs to just symmetry while withdrawing, i.e., close to the tracheal carina, and at the same time, try to make the tip of the endotracheal tube cross the esophageal and tracheal fistula.

Observation indicators Venous blood samples were collected from two groups of neonates at 10 min before induction of anesthesia (T0), 10 min after tracheal intubation (T1), pneumothorax establishment (T2), 60 min after pneumothorax establishment (T3), pneumothorax relief (T4), 4 h after pneumothorax relief (T5), and 24 h after pneumothorax relief (T6), and the concentrations of IL-6, IL-8, and TNF-α were measured using a double-antibody one-step sandwich enzyme-linked immunosorbent assay (ELISA, kits were purchased from Besancon Cedex, France). Radial artery blood gas (blood gas analysis 7 + Chips were purchased from i-STAT Abbott Portable Blood Gas Analyzer, USA), and P(A-a)DO2 were calculated = (atmosphere - PH2O) × FiO2 - PaCO2/R-PaO2, respiratory index RI = P(A-a)DO2/PaO2 (Vapor pressure PH2O = 47 mmHg, respiratory quotient R = 0.8, 760 mmHg for uniform atmospheric pressure, FiO2 is the inspired oxygen concentration). The duration of SICU stay, mechanical ventilation, hospital stay, incidence of postoperative pulmonary complications, such as atelectasis, pneumothorax, hypoxemia, and re-endotracheal intubation, of the subjects in the 2 groups were recorded.

Statistical analysis

All statistical data were analyzed using the SPSS 16.0 software. Normally distributed continuous data were shown as mean ± standard deviation (± s). Continuous data of non-normal distribution were shown as median and the quartiles Q1, Q3. Data distribution was analyzed by the Shapiro-Wilk method. The Mann-Whitney test was applied to compare the difference between the two groups for data with non-normal distribution. Unpaired or paired t test was used for comparison between the two groups of normally distributed data. Rates were compared by Chi-square test. $P < 0.05$ was considered statistically significant.

RESULTS

There is no significantly difference in all demographic indices and basic clinical characteristics, including age, sex ratio, ASA grade, body weight, operation time, and anesthesia time, between the two groups (Table 1).

Table 1. Comparison of general clinical data between the 2 groups (n = 30)

Group	**Group S**	**Group C**	**t or χ2**	**df**	**P**
Sex ratio (male/female)	19/11	18/12	0.071	1	0.791
ASA classification (II/III)	17/13	19/11	0.278	1	0.598
Body weight (kg, $\bar{x}$ ± s)	2.6±1.2	2.5±1.3	0.4660	58.0000	0.6430
Operation time (min, $\bar{x}$ ± s)	144.6±14.5	146.2±15.3	-0.4160	58.0000	0.6790
Group	Group S	Group C	U	Z	P
Age (d，M(Q1-Q3))	15.5 (6-18)	7 (6-20)	444.00	-0.089	0.929

Compared with T0 in the same group, the serum concentrations of IL-6, IL-8, and TNF-α in T1-T6 were significantly increased in both groups. Compared with group C, the serum concentrations of IL-6, IL-8, and TNF-α were remarkably lower in group St all different time points of T1~T6 (Table 2).

Table 2. Comparison of serum IL-6, IL-8, and TNF-α concentrations at different time points between the 2 groups ($\bar{X}$ ± s, n = 30)

Indicators	Time	Group S	Group C	ta	Pa	tb	Pb	tc	Pc
IL-6	T0	29.8±7.8	30.1±8.0	0.163	0.871	-	-	-	-
	T1	99.8±17.2	122.4±18.9	4.857	＜0.001	-20.271	0.001	-24.666	＜0.001
	T2	178.7±24.5	223.5±34.5	5.794	＜0.001	-31.696	＜0.001	-29.876	＜0.001
	T3	268.9±48.8	332.8±65.4	4.283	＜0.001	-26.478	＜0.001	-25.150	＜0.001
	T4	243.1±30.3	287.6±54.4	3.986	＜0.001	-37.322	＜0.001	26.217	＜0.001
	T5	132.3±20.2	176.5±23.8	7.723	＜0.001	-25.954	＜0.001	-31.718	＜0.001
	T6	63.5±12.4	95.7±16.8	8.446	＜0.001	-12.541	＜0.001	-19.310	＜0.001
IL-8	T0	36.3±8.9	35.6±9.2	-0.308	0.7590	-	-	-	-
	T1	124.3±19.7	148.6±25.3	4.154	＜0.001	-22.275	＜0.001	-23.005	＜0.001
	T2	189.4±34.3	233.1±40.2	4.531	＜0.001	-23.633	＜0.001	-26.222	＜0.001
	T3	229.8±39.6	288.8±53.2	4.868	＜0.001	-26.081	＜0.001	-25.668	＜0.001
	T4	200.5±36.2	249.5±43.4	4.747	＜0.001	-24.103	＜0.001	-26.389	＜0.001
	T5	154.4±24.6	188.6±33.2	4.539	＜0.001	-24.718	＜0.001	-24.356	＜0.001
	T6	87.5±24.3	124.2±20.1	6.378	＜0.001	-10.823	＜0.001	-21.948	＜0.001
TNF-α	T0	45.3±8.2	46.3±7.6	0.499	0.620	-	-	-	-
	T1	189.6±31.5	218.9±38.6	3.220	＜0.001	-24.312	＜0.001	-24.021	＜0.001
	T2	234.2±54.3	343.8±67.9	6.903	0.002	-18.835	＜0.001	-23.841	＜0.001
	T3	501.1±112.2	612.8±132.6	3.525	＜0.001	-22.200	＜0.001	-23.358	0.001
	T4	422.4±89.5	554.6±108.8	5.138	＜0.001	-22.978	＜0.001	-25.536	＜0.001
	T5	332.6±67.5	424.6±78.5	4.867	＜0.001	-23.130	＜0.001	-26.272	＜0.001
	T6	121.8±32.8	223.5±42.5	10.381	＜0.001	-12.408	＜0.001	-22.496	＜0.001

Note:

a: Group S compared with Group C at the same time (T0, T1, T2, T3, T4, T5, T6) using unpaired t test. If P<0.05, it is statistically significant.
b: The different time compared with T0 in the Group S using paired t test. According to Bonferroni correction, P should be less than 0.05/21=0.002, then it is statistically significant.
c: The different time compared with T0 in the Group C using paired t test. According to Bonferroni correction, P should be less than 0.05/21=0.002, then it is statistically significant.

Compared with T0 in the same group, P (A-a) DO2 and RI at T1 were increased from T1 to T6 in both groups. P (A-a) DO2 and RI in group S were significantly lower compared with group C at all different time points of T1~T6 (Table 3).

Table 3. Comparison of P (A-a) DO2 and RI at different time points between the two groups ($\bar{x}$ ± s, n = 30)

Indicators	Time	Group S	Group C	ta	Pa	tb	Pb	tc	Pc
P (A-a) DO2	T0	16.8±9.7	15.7±8.9	-0.471	0.639	-	-	-	-
	T1	285.5±32.2	352.4±28.5	8.527	＜0.001	-43.789	＜0.001	-61.776	＜0.001
	T2	422.8±41.5	466.8±36.1	-4.389	＜0.001	-52.204	＜0.001	-66.549	＜0.001
	T3	432.6±39.6	483.8±34.7	5.288	＜0.001	-55.808	＜0.001	-71.535	＜0.001
	T4	385.6±42.2	443.4±36.8	5.655	＜0.001	-46.689	＜0.001	-61.842	＜0.001
	T5	182.2±27.3	235.4±31.9	6.937	＜0.001	-31.299	＜0.001	-36.463	＜0.001
	T6	91.4±20.6	119.7±24.8	-4.804	＜0.001	-17.947	＜0.001	-21.602	＜0.001
RI	T0	0.24±0.19	0.21±0.12	-0.820	0.416	-	-	-	-
	T1	1.28±0.21	1.97±0.24	11.814	＜0.001	-20.131	＜0.001	-35.751	＜0.001
	T2	1.71±0.34	2.29±0.31	-6.853	＜0.001	-20.677	＜0.001	-33.783	＜0.001
	T3	1.82±0.26	2.59±0.32	10.155	＜0.001	-27.302	＜0.001	-33.783	＜0.001
	T4	1.35±0.25	1.96±0.26	9.180	＜0.001	-19.269	＜0.001	-37.725	＜0.001
	T5	0.88±0.20	1.03±0.25	2.687	0.009	12.708	＜0.001	-33.031	＜0.001
	T6	0.53±0.17	0.79±0.19	5.396	＜0.001	-6.272	＜0.001	-16.069	＜0.001

Note:

a: Group S compared with Group C at the same time using unpaired t test (T0, T1, T2, T3, T4, T5 ,T6). If P<0.05, it is statistically significant.
b: The different time compared with T0 in the Group S using paired t test. According to Bonferroni correction, P should be less than 0.05/21=0.002, then it is statistically significant.
c: The different time compared with T0 in the Group C using paired t test. According to Bonferroni correction, P should be less than 0.05/21=0.002, then it is statistically significant.

Compared with group C, the duration of SICU admission, mechanical ventilation and hospital stay in group S were all markedly decreased (Table 4).

Table 4. Comparison of SICU admission time, mechanical ventilation time, and hospital stay between the two groups ($\bar{x}$ ± s, n = 30)

Group	Duration of SICU admission (d)	Duration of mechanical ventilation (h)	Hospital stay (d)
Group S	6.3 ± 1.8	64.2 ± 7.1	12.4 ± 2.3
Group C	8.8 ± 2.3	75.5 ± 6.5	15.6 ± 2.5
t	-4.6720	-6.427	-5.122
df	58.0000	58.0000	58.0000
P	＜0.001	＜0.001	＜0.001

Note: Compared with group C using unpaired t test.

For total occurrence rate of atelectasis, pneumothorax, hypoxemia, and re-endotracheal intubation complication after surgery, group S also showed significantly lower rate than group C (Table 5).

Table 5. Comparison of postoperative pulmonary complications between the 2 groups [n (%)]

Group	Total	Atelectasis	Pulmonary infection	Pneumothorax	Hypoxemia	Re-endotracheal intubation
Group S (n=30)	3 (10.00)	0 (0)	1 (3.33)	0 (0)	2 (6.67)	0 (0)
Group C (n=30)	11 (36.67)	2 (6.67)	3 (10.00)	1 (3.33)	5 (16.7)	0 (0)
χ^2		19.880				
P		<0.001				

Note: Rates were compared by Chi square test. The complications were compared as a whole.

DISCUSSION

Hypercapnia and/or hypoxemia are common complications of thoracoscopic surgeries in neonates. Therefore, timely prevention and treatment of hypercapnia and/or hypoxemia have become one of the primary tasks of anesthesia management in this kind of surgery. In this non-ideal ventilation mode, both mechanical compression and lateral decubitus position can lead to severe uneven ventilation and blood redistribution in the lungs, resulting in an imbalance in the ventilation/blood flow ratio and increased intrapulmonary shunt, which in turn leads to abnormal changes in respiratory function in neonates [3]. During an artificial pneumothorax, the neonate's bilateral lungs are mechanically compressed and artificially pushed by an artificial CO2 pneumothorax, which leads to incomplete inflation of the affected lung and a decrease in the area of the respiratory membrane, causing insufficient alveolar ventilation and oxygenation [4]. CO2 pneumothorax, lateral decubitus position, and hypoxic pulmonary vasoconstriction (HPV) are all factors that affect intraoperative hypercapnia and/or hypoxemia [5, 6]. Among them, HPV can constrict small arteries in the non-ventilated lung and reduce intrapulmonary shunt. How to improve intraoperative hypoxemia is still one of the problems to be solved in clinical anesthesia. Continuous hypoxia in the alveoli during one-lung ventilation can induce some vasoactive substances with strong vasoconstrictive effects such as endothelin, thromboxane A and leukotrienes, stimulate HPV, reduce blood flow in the non-ventilated lung, and then improve hypoxemia [7]. It is currently believed that the mechanism of HPV is mainly that a large number of vasoconstrictor substances, such as histamine, catecholamines, bradykinin, and leukotrienes are released in the non-ventilated lung to stimulate HPV [7]. Hypoxia promoted the energy metabolism in cells of pulmonary vascular smooth muscle, accelerated the production of ATP, and maintained HPV. In addition, hypoxia could induce increased production of oxygen radical, insufficient production of lipid peroxides, shrink of Pulmonary small vessels, and production of HPV [8].

IL-6 can stimulate the release of endogenous inflammatory mediators as an essential proinflammatory factor, which will result in response of acute inflammatory after its concentration increased. Thus, IL-6 is a kind of sensitive factors at early stage of tissue injury. TNF-α as an essential regulator of proapoptosis will be lot released by inflammatory cells when abnormal pathophysiological changes occur in lung tissue. It indicates that TNF-α induces apoptosis in lung tissue to enhance permeability of lung tissue, and cause damage of lung tissue. TNF-α can promote the production of IL-6. They have a synergistic effect, which plays a synergistic role in promoting the adhesion of neutrophils to endothelial cells. Therefore, IL-6 and TNF-α have exist an essential effect on the adjustment of inflammatory cascade [9]. IL-8 is a chemotactic cytokine that promotes chemotaxis of inflammatory cells (mainly neutrophils), mediates cytotoxicity and local inflammation, and can cause a local inflammatory response, thereby acting as a bactericidal and damaging cell. In our research, the concentration of TNF-α, IL-6, and IL-8 in serum were higher than those before one-lung ventilation. It indicated that one-lung ventilation could accelerate the release of inflammatory mediators to induce acute lung injury.

Since sevoflurane has aromatic fruit flavor and is easily accepted by children, it is an ideal inhalation anesthetic for infants and young children with stable induction of anesthesia and short time. At present, the inhalation concentration of sevoflurane used in clinical anesthesia is mostly 1-2 minimum effective alveolar concentration (MAC), i.e., 1.7%-3.5%. In cardiovascular surgery anesthesia, 2% end-tidal concentration (i.e., 1.2 MAC) of sevoflurane is often used to inhibit the inflammatory response and play a certain role in organ protection [10]. Therefore, 2% was selected as the end-tidal concentration for sevoflurane in this study. The results of this study showed that after pretreatment with 2% sevoflurane, the serum concentrations of IL-6, IL-8, and TNF-α in the neonates in group S were decreased after one-lung ventilation as compared with the those in the neonates in group C, suggesting that sevoflurane pretreatment could reduce the release of IL-6, IL-8, and TNF-α in lung tissues and reduce lung tissue injury, and the mechanism may be that sevoflurane inhibited the release of inflammatory mediators, alleviated the inflammatory response, and inhibited the apoptosis of lung tissue cells by inhibiting the adhesion of neutrophils to endothelial cells [11,12]. It is not clear in which pathway sevoflurane affects the inflammatory response. Therefore, the specific mechanism remains to be further explored.

Clinically, P(A-a) DO2 and RI are the main indicators to evaluate the oxygenation function and pulmonary ventilation function of patients. The larger their values, the worse the oxygenation function and pulmonary ventilation function, and the more severe the pulmonary function damage [13]. The results of P(A-a) DO2 and RI in two groups of neonates were obviously higher than those before one-lung ventilation. P(A-a) DO2 and RI in group S after sevoflurane pretreatment were obviously lower than of group C. It indicated that sevoflurane pretreatment could reduce lung damage and enhance respiratory function after one-lung ventilation. Based on the comprehensive analysis, the reasons why sevoflurane pretreatment exerts lung

protective effects may be considered as follows: sevoflurane can reduce the degree of hypoxia in lung tissues and improve pulmonary microcirculation; scavenge oxygen free radicals, combat lipid peroxidation, and enhance the antioxidant capacity of the body [14]. It inhibits the expression of pro-inflammatory factors, regulates the balance between pro-inflammatory and anti-inflammatory factors, and reduces the damage of systemic inflammatory response to lung tissue [15]. The duration of SICU admission, time of mechanical ventilation, and days of hospital stay in group S were lower than that in group C due to the pulmonary protective role of sevoflurane pretreatment above. The occurrence rate of atelectasis, pneumothorax, hypoxemia, and re-endotracheal intubation postoperative pulmonary complication was also lower in group S. It is also indicated that the concentration of 2% sevoflurane pretreatment on neonates undergoing thoracoscopic correction of type III esophageal atresia is safe and effective.

CONCLUSION

Sevoflurane pretreatment at a concentration of 2% may have a protective effect on oxygenation and pulmonary ventilation in neonates, which conduct a correction of type III esophageal atresia undergoing thoracoscopy by inhibiting inflammatory responses.

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