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Experimental Study

The Role of Alpha Atrial Natriuretic Hormone in Pneumonectomy: An Experimental Study

Murat Akkuş,[1] Yener Yörük[2]

[1]Department of Thoracic Surgery, Mehmet Akif Ersoy Thoracic and Cardiovascular Surgery Training and Research Hospital, İstanbul, Turkey
[2]Department of Thoracic Surgery, Trakya University Faculty of Medicine, Edirne, Turkey

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Correspondence: Murat Akkuş, SBÜ İstanbul Mehmet Akif Ersoy Göğüs Kalp ve Damar Cerrahisi Eğitim ve Araştırma Hastanesi, Göğüs Cerrahisi Kliniği, İstanbul, Turkey
E-mail: akkusmdr@gmail.com

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ABSTRACT

Objective: The alpha atrial natriuretic hormone (α-ANH) is released in response to atrial distension and excessive fluid volume in the body. The aim of the present study was to evaluate α-ANH levels before and after pneumonectomy and to investigate the effects of normal and increased volume of crystalloid–colloid fluids on α-ANH following pneumonectomy in a rabbit model.

Methods: A total of 20 New Zealand rabbits were used in the study. The mean weight of the rabbits was 1.831 g. The subjects were divided into four groups with five in each group. The first group was given 3 mL/kg/h of crystalloid; the second group was given 10 mL/kg/h of crystalloid; the third group was given 3 mL/kg/h of colloid; the fourth group was given 10 mL/kg/h of colloid. Blood samples were preoperatively collected from the jugular vein. Posterolateral thoracotomy was applied to all subjects. The hilus was tied and cut en bloc with 2/0 silk, and pneumonectomy was performed. All groups received infusion for 3 h. Following infusion, blood samples from the contralateral jugular vein were collected at postoperative 3 h. Pre- and postoperative α-ANH levels were compared.

Results: There was no significant difference in the mean weight of the groups (χ^2=1.417, p=0.478). There was no significant difference in the pre- and postoperative α-ANH levels among all groups (Z=0.674, p=0.5 in the first; Z=0.405, p=0.686 in the second; Z=1.753, p=0.08 in the third; Z=0.944, p=0.345 in the fourth).

Conclusion: Our study results suggest that pneumonectomy alone appears not to change the α-ANH levels, and hypoxia, increased atrial pressure, and some neurohormonal factors may enhance α-ANH release.

INTRODUCTION

The concept of the heart as an endocrine organ has been increasingly adopted, mainly arising from the observation that the natriuretic hormones are secreted by atrial cardiomyocytes.[1,2] These include alpha atrial natriuretic hormone (α-ANH), brain natriuretic hormone, and C-type natriuretic peptide.[3] The α-ANH was first described and named by Flynn et al.[4] in a rat model in which the atrial extracts were infused to another rat group, leading to natriuresis.

The α-ANH is released in response to atrial distension and excessive fluid volume in the body. It plays a key role in the regulation of fluids and electrolytes. Several studies have shown that α-ANH exerts its effects through the relaxation of the vascular smooth muscles, mediating complex renal hemodynamic effects, inhibiting renin–aldosterone and vasopressin release, and hypotension.[5–8]

Pneumonectomy is a common surgical procedure in the thoracic surgery practice, mainly in lung cancer cases. Postpneumonectomy pulmonary edema (PPE) is one of the most frequent complications with high mortality rates. The literature claimed that PPE originally resulted from fluid overload.[9] However, in later studies, several factors including increased pulmonary capillary pressure, increased permeability, immune mediators, excessive transfusion of blood products, mediastinal lymph node dissection, exposure to mechanical ventilation, and hyperinflation have been blamed. Currently, the role of the fluid amount in pneumonectomy has been questioned.[10–12]

The aim of this experimental study was to evaluate the α-ANH levels before and after pneumonectomy and to investigate the effects of normal and increased volume of crystalloid–colloid fluids on α-ANH following pneumonectomy in a rabbit model.

MATERIALS AND METHODS

A total of 20 New Zealand rabbits were used in the present study. The mean weight of the rabbits was 1.831 (1.400–2.900) g. All animals were treated and cared for in accordance with the local Institutes of Health Guide for the care and Use of Laboratory Animals. The subjects were divided into four groups with five in each group. The first group was given 3 mL/kg/h of crystalloid (isotonic 0.9% NaCl^{R}; Fresenius Kabi AG, Bad Homburg, Germany); the second group was given 10 mL/kg/h of crystalloid (isotonic 0.9% NaCl^{R}; Fresenius Kabi AG); the third group was given 3 mL/kg/h of colloid (6% HES^{R} 450/0.7; Fresenius Kabi AG); the fourth group was given 10 mL/kg/h of colloid (6% HES^{R} 450/0.7; Fresenius Kabi AG). All groups received infusion for 3 h. The subjects were anesthetized with 10 mL/kg of ketamine (Ketalar 50 mg/mL, 10 mL flask; Pfizer, Kent, UK) and 0.3 mL/kg xylazine hydrochloride (Rompun 50 mL flask; Bayer, Leverkusen, Germany) and placed in a supine position. The jugular veins of the subjects were dissected using a vertical incision on the neck. They were catheterized using 24 F branule. One mg of ethylenediaminetetraacetic acid in 0.9 mL and 0.1 mL (TrasylolR 10,000 U/mL, 50 mL flask; Bayer) aprotinin-containing plastic tubes was prepared. One mL of blood was drained into these previously prepared and cooled tubes for α-ANH analysis. These blood samples were centrifuged at 2000g, −4 °C for 30 min. Blood plasma was separated and kept at −30 °C. After the blood was drawn, 3 mL/kg/h or 10 mL/kg/h crystalloid or colloid fluid was administered via a perfusion pump (Braun Perfusor; Melsungen, Germany).

Meanwhile, the trachea was reached and incised by expanding the incision to the neck. The subjects were intubated with a 3.0 intubation tube and were ventilated with continuous positive airway pressure with O_2 (tidal volume 4 mL/kg, respiratory rate 45–60/min, positive airway pressure 1–2, 30% O_2, Vokar-Sav 0301). The rabbits were placed in lateral decubitus according to the side to which pneumonectomy was to be applied. Posterolateral thoracotomy was applied to all subjects. The pulmonary inferior ligament was divided and then isolated, the hilus was tied and cut en bloc with 2/0 silk, and pneumonectomy was performed.

In the first group, left pneumonectomy and right pneumonectomy were performed in three and two subjects, respectively. In the second group, left pneumonectomy and right pneumonectomy were performed in two and three subjects, respectively. In the third group, left pneumonectomy and right pneumonectomy were performed in three and two subjects, respectively. Finally, in the fourth group, left pneumonectomy and right pneumonectomy were performed in three and two subjects, respectively. Blood samples from the contralateral jugular vein for α-ANH analysis were collected into plastic tubes, as described. The pneumonectomy procedure was repeated for the contralateral lung, and all subjects were sacrificed.

All plasma samples stored in the freezer were collectively analyzed in the Nuclear Medicine Center. The blood plasma was separated using the Alpha ANPAmprep 100 mg C8 colon kit (code: RPA 50129; Amershan Life Science, UK). The colon was washed with 2 mL methanol and 2 mL distilled water. Then, 0.25 mL 2 µHCl was added to each plasma at room temperature, acidified, and mixed. The plasma was treated with colons, and the colons were washed using 5 mL distilled water+0.1% (v/v) trifluoroacetic acid (TFA). Then, 60% methanol was added to 0.001 (v/v) TFA mixture, and the colons were re-washed with 4 mL of this mixture. The filtrate was collected in a glass tube, cooled, and dried under nitrogen. Then, 200 µL assay buffer was added, and the procedure was completed. Then, 100 µL of each sample was collected, and the α-ANH levels were examined using a gamma counter (DPC-Gammbyte) for 60 s in the Nuclear Medicine Center, per protocol.

The control group included blood samples that were obtained before the procedure and after fluid infusion following pneumonectomy; therefore, each subject could be used as both the experimental and the control subjects, and faulty results were avoided. For the α-ANH analysis, blood samples obtained before pneumonectomy were compared with the samples obtained after pneumonectomy, and the change in blood amount was evaluated between the experimental and control groups.

Statistical analysis

Statistical analysis was performed using the Excel software (Microsoft Corp., Seattle, WA, USA). Descriptive data were expressed as mean and standard deviation for continuous variables and number and percentage (%) for categorical variables. The Kruskal–Wallis test was used to compare the mean weight of the subjects. The Wilcoxon test was used to analyze significant differences in the pre- and postoperative ANH levels. A p value of <0.05 was considered statistically significant.

RESULTS

There was no significant difference in the mean weight of all groups (χ^2=1.417, p=0.478).

Pre- and postoperative α-ANH levels

In the first group (3 mL/kg/h of crystalloid), the α-ANH levels before and after pneumonectomy are shown in Table 1. The mean preoperative α-ANH level was 24.10±9.77 fmol/mL, whereas the mean postoperative α-ANH level was 19.85±5.67 fmol/mL. The mean change from baseline is illustrated in Fig. 1.

In the second group (10 mL/kg/h of crystalloid), the α-ANH levels before and after pneumonectomy are shown in Table 2. The mean preoperative α-ANH level was 19.85±3.90 fmol/mL, whereas the mean postoperative α-ANH level was 16.12±3.45 fmol/mL. The mean change from baseline is illustrated in Fig. 2.

In the third group (3 mL/kg/h of colloid), the α-ANH levels before and after pneumonectomy are shown in Table

Table 1. The α-ANH levels in pneumonectomy before and after 3 mL/kg/h of crystalloid perfusion in the first group

No	Pneumonectomy	Pre-ANH	Post-ANH
1	Left	29.31	20.55
2	Left	32.00	15.35
3	Left	11.41	14.63
4	Right	15.80	19.85
5	Right	32.00	28.84

α-ANH: Alpha atrial natriuretic hormone; Pre-ANH: Preoperative atrial natriuretic hormone; Post-ANH: Postoperative atrial natriuretic hormone.

Table 2. The α-ANH levels in pneumonectomy before and after 10 mL/kg/h of crystalloid perfusion in the second group

No	Pneumonectomy	Pre-ANH	Post-ANH
1	Left	13.33	15.84
2	Left	14.39	15.32
3	Right	16.58	17.05
4	Right	12.31	11.41
5	Right	22.13	21.02

α-ANH: Alpha atrial natriuretic hormone; Pre-ANH: Preoperative atrial natriuretic hormone; Post-ANH: Postoperative atrial natriuretic hormone.

Table 3. The α-ANH levels in pneumonectomy before and after 3 mL/kg/h of colloid perfusion in the third group

No	Pneumonectomy	Pre-ANH	Post-ANH
1	Right	15.23	13.27
2	Right	19.79	11.57
3	Left	31.94	12.62
4	Left	13.70	13.76
5	Left	15.76	11.84

α-ANH: Alpha atrial natriuretic hormone; Pre-ANH: Preoperative atrial natriuretic hormone; Post-ANH: Postoperative atrial natriuretic hormone.

Table 4. The α-ANH levels in pneumonectomy before and after 10 mL/kg/h of colloid perfusion in the fourth group

No	Pneumonectomy	Pre-ANH	Post-ANH
1	Left	18.74	13.33
2	Left	15.41	10.15
3	Left	15.85	14.31
4	Right	12.65	17.75
5	Right	13.72	14.45

α-ANH: Alpha atrial natriuretic hormone; Pre-ANH: Preoperative atrial natriuretic hormone; Post-ANH: Postoperative atrial natriuretic hormone.

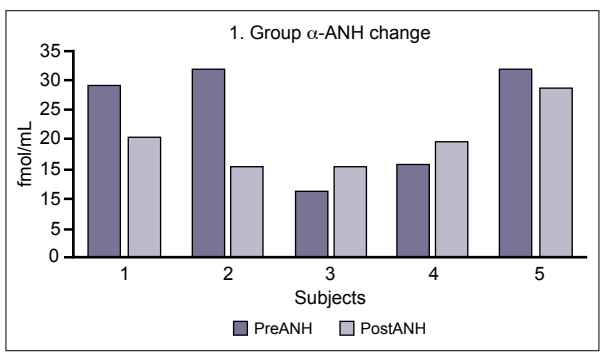

Figure 1. The mean change from baseline in the α-ANH levels in the first group. α-ANH: Alpha atrial natriuretic hormone.

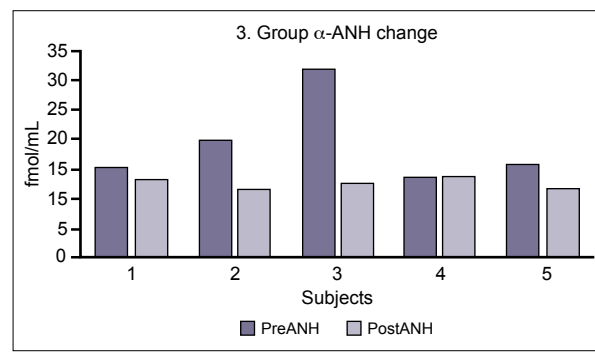

Figure 3. The mean change from baseline in the α-ANH levels in the third group. α-ANH: Alpha atrial natriuretic hormone.

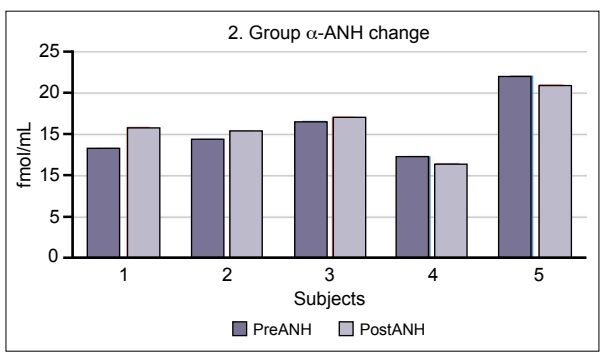

Figure 2. The mean change from baseline in the α-ANH levels in the second group. α-ANH: Alpha atrial natriuretic hormone.

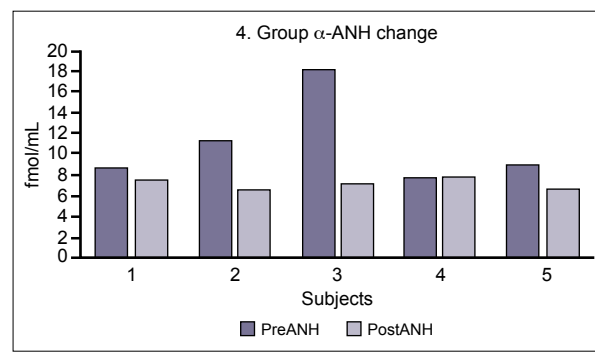

Figure 4. The mean change from baseline in the α-ANH levels in the fourth group. α-ANH: Alpha atrial natriuretic hormone.

3. The mean preoperative α-ANH level was 19.28±7.42 fmol/mL, whereas the mean postoperative α-ANH level was 12.61±0.92 fmol/mL. The mean change from baseline is illustrated in Fig. 3.

In the fourth group (10 mL/kg/h of colloid), the α-ANH levels before and after pneumonectomy are shown in Table 4. The mean preoperative α-ANH level was 15.27±2.32 fmol/mL, whereas the mean postoperative α-ANH level was 13.99±2.72 fmol/mL. The mean change from baseline is illustrated in Fig. 4.

There was no statistically significant difference in the α-ANH levels before and after the procedure (Z=0.674, p=0.05 in the first group; Z=0.405, p=0.686 in the second group; Z=1.753, p=0.08 in the third group; Z=0.944, p=0.345 in the fourth group) in all groups.

DISCUSSION

In this experimental study, the α-ANH levels before and after pneumonectomy were evaluated, and the effects of normal and increased volume of crystalloid–colloid fluids on α-ANH following pneumonectomy in a rabbit model were examined. New Zealand rabbits were used in the present study, as these materials are readily available, inexpensive, and particularly amenable for laboratory testing. Similarly, there are many studies on this subject using rabbit models in the literature.[6] The levels of α-ANH are shown to be higher in pulmonary resection cases with pneumonectomy than in lobectomy cases in some experimental study.[13]

In our study, all subjects underwent chest X-ray to rule out any pathology. Jugular catheterization and tracheostomy with positive ventilation were conducted in accordance with the techniques described in previous studies.[14] In addition, the gamma camera for radioimmunoassay was operated in compliance with the previous studies.[4,6,15,16]

Matsubara et al.[17] evaluated the α-ANH levels in patients with acute myocardial infection and prior myocardial infarction using blood samples collected simultaneously from the brachial vein, right atrium, pulmonary artery, and femoral vein. The authors found no significant difference between the groups. However, they reported that the α-ANH was degraded in the pulmonary circulation, although it had little clinical significance in the α-ANH measurement. Similarly, blood samples were collected from the jugular veins for the measurement of the α-ANH.

In the present study, the mean pre- and postoperative α-ANH levels are consistent with previous reports in all the four groups.[4,5,14,18,19] The first and third groups received crystalloid (3 mL/kg/h, 0.9% NaCl) and colloid (3 mL/kg/h, 6% HES 450/0.7) in normal volume. The mean pre- and postoperative α-ANH levels were 24.10 fmol/mL and 19.84 fmol/mL and 19.84 fmol/mL and 12.61 fmol/mL, respectively. Despite a slight decrease in the postoperative level, it did not reach statistical significance. These decrease can be explained by minimal hemorrhage and hypovolemia during the procedure. The second and fourth groups received crystalloid (10 mL/kg/h, 0.9% NaCl) and colloid (10 mL/kg/h, 6% HES 450/0.7) in an increased volume. The mean pre- and postoperative α-ANH levels were 15.74 fmol/mL and 16.12 fmol/mL and 15.27 fmol/mL and 13.99 fmol/mL, respectively. Despite a slight increase in the third group and decrease in the fourth group postoperative level, both of them did not reach statistical significance. An increase in the third group can be explained by high-volume crystalloid infusion. A decrease in the fourth group can be explained by increased intravascular volume of the colloids that did not lead to atrial strain. Based on these findings, pneumonectomy appears to have no effect on the α-ANH levels.

During pneumonectomy, right or left pulmonary artery ligation increases the pulmonary artery pressure; however, it is not associated with atrial distension in patients with normal cardiac functions without an increase in the α-ANH levels.[20] On the contrary, there are experimental studies in the literature showing increased α-ANH levels in pneumonectomy cases.[21-23] In an experimental study, Chen et al.[23] showed that not only atrial distension and strain but also hypoxia plays a central role in the α-ANH release. Hypoxia directly stimulates the α-ANH release from cardiac myocytes and prevents hypoxic pulmonary hypertension. Similarly, the lack of any increase in the α-ANH levels after pneumonectomy can be attributed to the absence of hypoxemia in our study.

There are a number of studies showing increased α-ANH levels with fluid replacement in the literature. In a rat model, Garcia et al.[6] found that the α-ANH levels increase by 4 to 5 fold with increased atrial pressure when the total blood volume was increased using 50% isotonic solutions. In their study, the authors used volume loading to increase the blood pressure in the rats undergoing right and left atrial appendectomy and performed the α-ANH measurement. Compared with the control group, the α-ANH release originated from the right atrium, which was mainly associated with the blood pressure regulation. In addition, the authors reported a decrease in the α-ANH levels proportional to the volume loss due to hemorrhage during surgery. In our study, although there was no significant difference in the α-ANH levels, a slight decrease was observed after pneumonectomy in normal volume in both groups.

Amano et al.[24] reported a significant and prolonged increase in the α-ANH levels with hypertonic solutions compared with glucose and isotonic solutions. On the other hand, in our study, only a slight increase was found in the postoperative α-ANH level in the high crystalloid volume group, although it did not reach statistical significance. An increase was not obtained in the high-volume colloid group.

In a study conducted on a dog model, Ledsome et al.[25] inserted a balloon catheter into the left atrium and catheters into three pulmonary veins. They inflated the balloon until the left atrial pressure reached 11 cm H_2O to induce mitral valve obstruction. They found a mild in-

crease in the pulmonary artery pressure with pulmonary vein distension, leading to a significant increase in heart rate. However, there was no significant change in left atrial or arterial pressure. Despite a slight increase in the α-ANH levels, it did not reach statistical significance.

Cheung et al.[5] found that despite high-dose α-ANH in healthy individuals, there is no significant change in blood pressure, heart rate, and glomerular filtration. The lack of hypotension and tachycardia was attributed to the presence of other neuronal and hormonal factors that are responsible for the vasomotor effects of natriuretic peptides, leading to fluid escape from the intravascular space to the extravascular space.[4] In addition, Matsubara et al. demonstrated the effect of certain catecholamines on the α-ANH release and renin–angiotensin–aldosterone system. These neuronal and hormonal regulatory factors may also play a role in the slight change in the α-ANH levels.[16]

Furthermore, Richards et al.[8] examined the effects of sodium loading with diet on the α-ANH levels in volunteers and found decreased blood pressure and urinary electrolyte excretion. However, the renin activity and aldosterone, anti-diuretic hormone, or noradrenalin levels remained unchanged.[7] On the other hand, some research showed that the α-ANH and renin–angiotensin–aldosterone system are closely associated with another.[19,26] These findings can be another explanation for the non-increased α-ANH levels after pneumonectomy and overload fluid administered.

Omari et al.[18] evaluated the effect of cannulation below from the atrial appendix during right atrial cannulation for elective coronary artery bypass grafting on the α-ANH levels. The authors found that the α-ANH levels in the atrial appendix cannulation were significantly lower. In addition, the α-ANH release occurred when the right atrial pressure reached ≥8 mmHg with 7.5 mL/kg 5% albumin loading. The limitation of our study is the lack of an atrial pressure measurement.

CONCLUSION

In conclusion, our study results showed that pneumonectomy was not associated with increased α-ANH levels in rabbits, and crystalloid and colloid fluids in normal or increased volume did not affect the α-ANH levels following pneumonectomy. Based on these findings, pneumonectomy alone appears not to change the α-ANH levels, and hypoxia, increased atrial pressure, and some neurohormonal factors may enhance α-ANH release.

Ethics Committee Approval

Approved by the local ethics committee.

Peer-review

Internally peer-reviewed.

Authorship Contributions

Concept: M.A., Y.Y.; Design: M.A.; Data collection&/or processing: M.A.; Analysis and/or interpretation: M.A, Y.Y.; Literature search: M.A.; Writing: M.A.; Critical review: M.A., Y.Y.

Conflict of Interest

None declared.

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Pnömonektomide Alfa Atrial Natriüretik Hormonun Rolü: Deneysel Çalışma

Amaç: Atrial natriüretik hormon (α-ANH) vücutta aşırı sıvı birikimi ve atrial distansiyona cevap olarak salgılanır. Biz, deneysel tavşan modelimizde pnömonektomi öncesi ve sonrası normal ve yüksek volüm kristaloid ve kolloid sıvı verilmesinin alfa atrial natriüretik hormon seviyesine etkisini değerlendirmeyi amaçladık.

Gereç ve Yöntem: Çalışmada ortalama ağırlığı 1831 g olan toplam 20 Yeni Zelanda tavşanı kullanıldı. Örnekler her biri beş denekten oluşan dört gruba ayrıldı. Birinci gruba 3 mL/kg/sa kristaloid, ikinci gruba 10 mL/kg/sa kristaloid, üçüncü gruba 3 mL/kg/sa kolloid, dördüncü gruba 10 mL/kg/sa kolloid sıvı başlandı. Ameliyat öncesi juguler venden kan örnekleri alındı. Tüm deneklere posterolateral torakotomi uygulandı. Hilus 2/0 ipek ile en blok bağlandı ve kesildi, pnömonektomi yapıldı. Bütün gruplara üç saat infüzyon yapıldı, ameliyat sonrası üçüncü saat karşı taraf juguler venden kan örnekleri alındı. Ameliyat öncesi ve sonrası α-ANH seviyeleri karşılaştırıldı.

Bulgular: Gruplar arasında ortalama ağırlık açısından farklılık yoktu. Ameliyat öncesi ve sonrası gruplar arasında α-ANH seviyeleri arasında anlamlı farklılık saptanmadı (birinci grup Z=0.674; p=0.5; ikinci grup Z=0.405; p=0.686; üçüncü grup Z=1.753; p=0.08; dördüncü grup Z=0.944; p=0.345).

Sonuç: Çalışmamızda sonuçlar pnömonektominin α-ANH seviyesi değişimine yalnız başına etkisi olmadığını göstermektedir. Hipoksi, artmış atrial basınç ve bazı nörohormonal faktörler α-ANH salınımının artmasına yol açabilir.

Anahtar Sözcükler: Alfa atrial natriüretik hormon; atriyal natriüretik peptidler; pnömonektomi; postpnömonektomik pulmoner ödem.