ORIGINAL ARTICLE

# Spontaneous rectus sheath hematoma in cardiac in patients: a single-center experience

**Ebubekir Gündeş, M.D., Durmuş Ali Çetin, M.D., Ulaş Aday, M.D., Hüseyin Çiyiltepe, M.D., Kamuran Cumhur Değer, M.D., Orhan Uzun, M.D., Aziz Serkan Senger, M.D., Erdal Polat, M.D., Mustafa Duman, M.D.,**

Department of Gastroenterological Surgery, Kartal Koşuyolu High Speciality Training and Research Hospital, İstanbul-*Turkey*

## ABSTRACT

**BACKGROUND:** This study presents the relationship between mortality and spontaneous rectus sheath hematoma (RSH) in inpatients receiving anticoagulant and antiaggregant treatment for cardiac pathology at cardiology and cardiovascular surgery clinics.

**METHODS:** Within the scope of our study, the cases of 27 patients who were diagnosed with spontaneous RSH between January 2010 and December 2015 at Kartal Kosuyolu High Speciality Training and Research Hospital were retrospectively evaluated.

**RESULTS:** Of the 27 patients, 19 (70.4%) were female and 8 (29.6%) were male. The mean age was 63±12 (32–84) years. All the patients had at least one comorbidity that necessitated follow-up. Fourteen patients received only anticoagulant treatment, 8 received only antiaggregant treatment, and the remaining 5 received both types of treatment. Physical examination of all patients revealed painful palpable masses in the lower quadrants of the abdomen. According to the results of computed tomography (CT) scans, which showed the size and localization of the masses, 7 of the cases were classified as Type I, 6 as Type II, and 14 as Type III. Although 23 of the cases received medical treatment, the remaining 4 patients received surgical treatment. Eight (29.6%) patients suffered mortality.

**CONCLUSION:** RSH is rare, but its prevalence is increased among patients receiving anticoagulant and antiaggregant treatment for cardiac reasons. The mortality rate markedly increased among patients who contracted RSH during hospitalization for cardiac reasons, had comorbidities, and experienced additional complications due to extended hospitalization.

**Keywords:** Anticoagulant treatment, antiplatelet treatment; rectus sheath hematoma.

## INTRODUCTION

Although rectus sheath hematoma (RSH) is a rare condition that has been reported in a limited number of studies, it has well-defined pathogenesis, treatment clinics, and treatment modalities.[1] It is an uncommon cause of acute abdominal pain. Despite the fact that RSH is mostly self-limiting, it may lead to unnecessary surgical procedures or even death if the patient is not diagnosed. RSH very rarely occurs in children and can develop at any age among adults.[1,2] It most frequently appears on the right side beneath the umbilicus and is almost always unilateral.[3]

Rectus muscular sheath hematoma is 2 to 3 times more prevalent in women than men. Although there are many possible risk factors, it has been reported that an increase in the number of spontaneous RSH cases occurred at the same time as that in anticoagulant treatments. Patients undergoing this type of treatment may suffer from hemodynamic instability because the treatment increases the likelihood of hemorrhage during RSH formation.[4,5]

Treatment of RSH is conservative because the illness is mostly self-limiting, but surgical treatment is recommended for complicated and large hematomas, which can cause hemodynamic disorders (such as rupture opening up to the peritoneum and infection).[1,2,5,6]

This study describes the relationship between mortality and spontaneous RSH in inpatients receiving anticoagulant and antiaggregant treatment for a cardiac pathology at cardiology and cardiovascular surgery clinics.

**Address for correspondence:** Ebubekir Gündeş, M.D.
Kartal Koşuyolu Yüksek İhtisas Eğitim ve Araştırma Hastanesi, Gastroenteroloji Cerrahisi Kliniği, İstanbul, Turkey
Tel: +90 216 - 500 15 00 E-mail: ebubekir82@hotmail.com

Submitted: 27.05.2016
Accepted: 06.04.2017

Ulus Travma Acil Cerrahi Derg
2017;23(6):483–488
doi: 10.5505/tjtes.2017.67672

## MATERIALS AND METHODS

Within the scope of our study, data of 27 patients who were diagnosed with spontaneous RSH between January 2010 and December 2015 at Department of Gastroenterological Surgery, Kartal Kosuyolu High Speciality Education and Training Hospital were retrospectively evaluated after consent was obtained from Dr. Lütfi Kırdar Kartal Training and Research Hospital's Board of Clinical Research Evaluation (Registration No;89513307/1009/518). The study included inpatients at cardiology and cardiovascular surgery clinics who contracted spontaneous RSH during hospitalization and after evaluation received conservative or surgical treatment.

The patients' anamnesis, age, sex, comorbidities, and reason for starting antiaggregant and/or anticoagulant treatment were recorded following investigation of the hospital's archive files and electronic archive system. Patients were classified in line with Berná et al.'s[7] classification for hematomas according to the radiological diagnostic method utilized at the diagnostic stage and the size and localization of the hematoma. Patients' international normalized ratio (INR), prothrombin time (PT), and activated partial thromboplastin time (aPTT) and hematocrit (hct), platelet, and leukocyte levels were recorded at the time of diagnosis. The day of hospitalization and decrease in hemoglobin and hct levels at the time of diagnosis were identified. The medical and surgical treatment methods for hematoma following diagnosis, amount of blood and blood product replacement, morbidity and mortality rates of the patients in the early phase of the disease, and duration of hospitalization were also investigated.

### Statistical Analysis

Statistical Package for the Social Sciences (SPSS 21 Inc., Chicago, IL, USA) was used for biostatistical analysis. The collected data were expressed as means (±SD), minimums and maximums, or percentages. The hct levels at hospitalization and at the time of RSH diagnosis were compared using a paired t-test. Spearman test was performed to analyze the mortality correlations. P values of <0.05 were considered to be statistically significant.

## RESULTS

### Patients

During the study, 27 patients with spontaneous RSH were treated at our clinic. Of these, 19 (70.4%) were female and 8 (29.6%) were male. The mean age was 63±12 (32–84) years. All the patients had at least one comorbidity that necessitated follow-up. The clinical and demographic characteristics of the patients are summarized in Table 1.

At the time of diagnosis, no patient had a history of trauma or abdominal surgery within the previous month or pregnancy or peritoneal dialysis at any point in their lives. All patients contracted RSH due to cardiac reasons while they were being treated in inpatient clinics. When the patients were evaluated with regards to abdominal injection, it was observed that insulin was not administered, although 8 of the patients were diabetic, and the arm was used as the injection site for patients who had low-molecular-weight heparin.

**Table 1.** Clinical and demographic characteristics of the patients

| | |
|---|---|
| Sex (n=27) | |
| Female | 19 (70.4%) |
| Male | 8 (29.6%) |
| Age (years) | 63±12 (32–84) |
| Anticoagulant treatment (n=14; 51.8%) | |
| Oral anticoagulant | 10 |
| Low-molecular-weight heparin | 4 |
| Antiplatelet treatment (n=8; 29.6%) | |
| Acetylsalicylic acid | 4 |
| Klopidogrel | 2 |
| Acetylsalicylic acid + Klopidogrel | 2 |
| Anticoagulant + antiplatelet treatment | 5 (18.5%) |
| Comorbidities | |
| Hypertension | 8 |
| Diabetes mellitus | 8 |
| Atrial fibrillation | 9 |
| Coronary artery disease | 7 |
| Chronic obstructive pulmonary disease | 4 |
| Chronic renal failure | 4 |
| Cerebrovascular disease | 3 |
| Other (Cirrhosis, PVT, PE) | 3 |
| Indications for anticoagulant and antiplatelet treatments | |
| Atrial fibrillation | 8 |
| Valve replacement | 5 |
| Coronary artery bypass graft | 5 |
| Acute myocardial infarction | 5 |
| Coronary angiography-stent placement | 4 |

PVT: Portal vein thrombosis, PE: Pulmomary embolism.

Of the patients, 14 received only anticoagulant treatment, 8 received only antiaggregant treatment, and the remaining 5 received both types of treatment. Anticoagulant and antiplatelet treatments are the most common indicators of atrial fibrillation (n=8).

### Diagnosis

Physical examination of all the patients revealed painful palpable masses in the lower quadrants of the abdomen (Fig.

**Figure 1.** Ecchymosis in the upper, lower, and pelvic abdominal area.

1). Abdominal ultrasonography (USG) was initially performed for 8 patients, and then all 27 patients underwent abdominal computed tomography (CT). According to the results of the CT scans, which revealed the size and localization of the masses, the majority of cases were classified as Type III (n=14, 51.8%) (Fig. 2a-c). The largest hematomas revealed by the CT scans averaged 130±68 (40–300) mm. The majority of hematomas were located in the right quadrant of the abdomen (n=16, 59.2%). The patients' radiological results are summarized in Table 2.

There was a significant relationship between hematoma size and tomographic type (p=0.003). Based on the level of significance of the groups, there is no difference between Type I and Type 2. However, there was a significant difference between these types and Type 3. There was not a significant relationship between the number of transfusions and type of hematoma (p=0.222).

According to the laboratory values obtained at the time of diagnosis, the INR levels of all patients using warfarin were above 2. The hct levels of patients with levels of 35.48±3.87 (30.1–44.6) at the time of hospitalization were 27.07±4.19 (16.9–36.6) on the day of RSH diagnosis, a decrease that was statistically significant (p<0.05). The laboratory results of the patients are summarized in Table 3.

**Table 2.** Computed tomography findings of patients

| | n | % | Mean±SD |
|---|---|---|---|
| Type | | | |
| 1 | 7 | 25.9 | |
| 2 | 6 | 22.2 | |
| 3 | 14 | 51.8 | |
| Size (mm) | | | 130±68 (40–300) |
| Localization | | | |
| Right | 16 | 59.2 | |
| Left | 8 | 29.6 | |
| Bilateral | 3 | 11.1 | |

SD: Standard deviation.

**Table 3.** Laboratory values of patients

| | Mean±SD |
|---|---|
| International normalized ratio | 2.02±1.5 (1–7) |
| Prothrombin time | 20.5±10.7 (12.7–61) |
| Activated partial thromboplastin time | 36.1±15.4 (20.7–96.1) |
| Hematocrit | 27.07±4.19 (16.9–36.6) |
| Hematocrit level at first | 35.48±3.87 (30.1–44.6) |
| Platelet count $10^9$/L | 249.1±89.06 (83–459) |
| Leukocyte | 10866±4016 (4500–23900) |

SD: Standard deviation.

## Treatment

Only 4 of the patients who were diagnosed with RSH did not have to undergo blood transfusion at follow-ups. Accord-

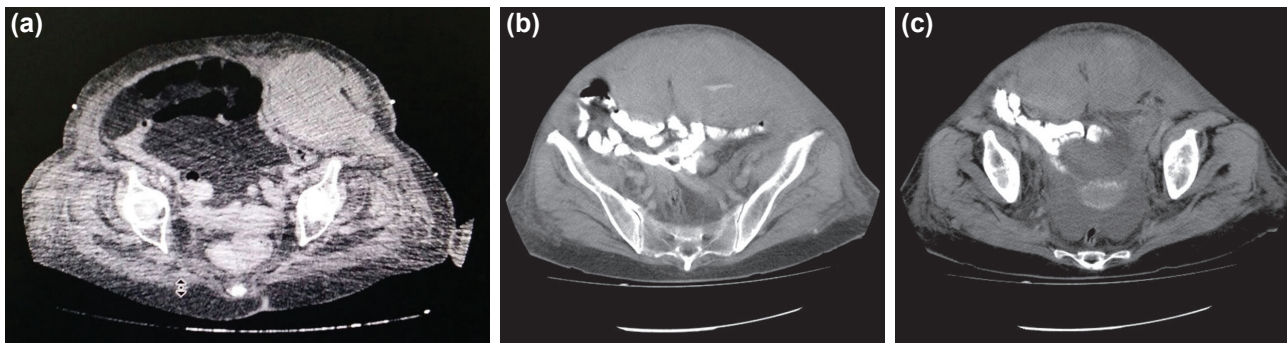


**Figure 2. (a)** Computed tomography scan showing Type I rectus sheath hematoma. **(b)** Type II hematoma. Computed tomography scan showing hyperdense bilateral hematoma. **(c)** Computed tomography scan showing Type III hematoma.

**Table 4.** Follow-up and treatment

| | n | % | Mean±SD |
|---|---|---|---|
| Blood transfusion | | | |
| Yes | 23 | 85.1 | |
| No | 4 | 14.81 | |
| Mean number of transfusion | | | 10±12 (1–45) |
| Treatment | | | |
| Medical | 23 | 85.1 | |
| Surgery | 4 | 14.9 | |
| Mortality | 8 | 29.6 | |
| Length of stay in hospital | | | 23.2±20.8 (5–92) |
| The time of diagnosis after hospitalization | | | 13±16 (3–70) |

SD: Standard deviation.

ing to the radiology results, all the cases not requiring blood transfusion were classified as Type I. Twenty-three patients enrolled in the study received blood transfusions, which averaged to 10±12 (1–45) units. Moreover, mortality was significantly correlated with the requirement of blood transfusion (p=0.001). Ten of these 23 patients underwent cardiac surgery during their hospitalization.

Of the 27 patients, 23 received medical treatment and the remaining 4 received surgical treatment. For patients who underwent surgery, the need for surgery was identified when the hematoma ruptured into the peritoneum. The mean duration of hospitalization was 23.2±20.8 (5–92) days, and the mean time of RSH diagnosis was 13±16 (3–70) days. Eight (29.6%) patients suffered mortality. Five of these patients underwent cardiac surgery, and the other 3 patients attended follow-ups to check for acute myocardial infarction and atrial fibrillation. Of the 8 patients who suffered mortality, 7 died due to sepsis related to hospital-acquired pneumonia and 1 due to hemorrhagic shock after massive hemorrhage. The patients' follow-ups and treatments are summarized in Table 4.

## DISCUSSION

The rectus abdominis muscle, one of the structures that forms the anterior abdominal wall, is located within an aponeurotic sheath alongside the inferior and superior epigastric veins. Rupture of these veins or the rectus abdominis muscle leads to RSH.[8]

RSH is a rare but significant disease that can imitate an acute abdomen because of its clinical manifestation. It is often overlooked as a possibility in emergency rooms (ERs), and the causes of acute abdominal pain are investigated more commonly.[9,10] However, RSH should be taken into consideration during the evaluation of acute abdominal pain, particularly for inpatients receiving anticoagulant or antiaggregant treatment for reasons other than those that led them to visit the ER.

RSH mostly occurs in people in their 60s and twice as often in female patients because they have less rectus muscle mass.[1,2] In line with the literature, most of the patients in our study were older than 60 years, and 70% were female. This disease is more frequently seen in older age groups due to their higher use of anticoagulant and antiaggregant drugs. Moreover, more spontaneous RSH cases have been reported for senior individuals because the elasticity of their epigastric veins decreases with atheromatous mural changes.[11]

The probable risk factors for RSH include trauma, rapid and sudden changes in position, anticoagulant treatment, antiaggregant treatment, recent history of surgery, acute asthma and COPD exacerbations and coughing attacks, injections, and pregnancy.[12] Recently, the gradual increase in the use of antiaggregant and anticoagulant treatments, particularly at cardiology and cardiovascular surgery clinics, has led to an increase in the prevalence of RSH.[5] Hematomas related to anticoagulant treatment are generally formed 4–14 days after initiation of treatment.[13,14] All the patients in our study had been hospitalized for cardiac reasons or had been receiving antiaggregant treatment and on average had contracted RSH on the 13$^{th}$ day of hospitalization.

USG, CT, and magnetic resonance imaging techniques are used to diagnose RSH. The sensitivity of USG, which is the easiest and most rapidly available diagnostic tool for initial investigation of patients suspected to have RSH, is between 80% and 90%. The results of USG generally reveal accurate information about the size and localization of the mass.[15,16] It may, however, be difficult to differentiate intra-abdominal lesions from extra-abdominal lesions. In our study, USG was used as the first diagnostic tool for only 8 patients, and CT was used for the final comprehensive diagnosis.

Abdominal CT is the standard imaging technique for diagnosis. CT is a diagnostic tool that is utilized for non-diagnostic

USG results, elimination of other intra-abdominal pathologies, and classification of RSH with sensitivity and authenticity rates of 100%.[6,17] Berná et al.[7] devised a system of classification for RSH that takes CT results into consideration. Final diagnoses of all the patients in our study were obtained using CT. According to the results, 7 of our patients were Type I, 6 were Type II, and 14 were Type III. Four of the patients with Type I RSH followed up without the need for blood transfusion. We believe that the reason why Type III was more common than the other types was related to the higher amount of hemorrhage in patients who had contracted RSH when taking anticoagulant or antiaggregant treatment.

RSH is mostly self-limiting, and thus, its treatment is conservative. Under such a treatment plan, it is advised that anticoagulant treatment be stopped in cases of anticoagulant-related bleeding, and vitamin K, factor replacement, or fresh-frozen plasma should be utilized when necessary. Application of ice on the hematoma area, non-steroidal anti-inflammatory analgesics, and bed rest can also be sufficient.[12,13,18,19] Surgical treatment, however, is recommended for complicated hematomas (showing rupture into the peritoneum, infection, etc.) and large hematomas causing hemodynamic disorders. Surgeons make an incision in the mass, drain the hematoma, wash it with saline, and if possible, ligate the bleeding vein (but the vein cannot be detected in most cases).[1,2] Approximately 85% of the patients in our study received conservative treatment. Surgical procedures were performed for 4 patients because their hematomas ruptured into the peritoneum. All the patients in our study had been receiving anticoagulant and/or antiaggregant treatment for major cardiac diseases. Because stopping these treatments was contraindicated for cardiac diseases, they were maintained for patients who had stable hemodynamic follow-up results, laboratory parameters, and hematoma sizes.

Interventional radiological techniques can be performed as an alternative to surgery in serious cases of active bleeding. Using the selective embolization technique to treat bleeding veins is not preferred because it requires significant experience to be performed correctly, it is not always feasible, and the hematoma remains in that area after bleeding is stopped.[20] Because of the lack of an experienced interventional radiological department at our hospital, embolization could not performed for any patient.

RSH-related mortality is rare.[2] However, the mortality rate increases for patients suffering from hematomas related to anticoagulant treatment.[8] In a clinical study conducted by Dağ et al.,[21] the authors found that 9% of patients with RSH who had been on anticoagulants suffered mortality.There are no studies on the factors affecting mortality in patients in which RSH was directly related to anticoagulant treatment. It has, however, been established that mortality related to bleeding in the upper gastrointestinal system occurring after anticoagulant treatment was to a large extent related to comorbidities.[22] Eight (29.6%) patients in our study suffered mortality, which was a higher rate than that reported in previous studies.[1,2] We believe that this was due to the high rate of comorbidities and the fact that our patients were diagnosed with RSH while they were undergoing medical and surgical treatment for serious cardiac problems. It was not possible to say that all deaths were related to RSH due to patients' pre-existing heterogeneous cardiac diseases and interventions. However, as mentioned above, the relationship between the number of blood transfusions and mortality rate in this heterogeneous group suggests that mortality increases in RSH patients who need more blood transfusions.

## Limitations

Our work has a few important limitations. The first and most important limitation is the retrospective nature of the study. Second, the study population is heterogeneous because it includes patients with various ailments that require hospitalization, such as atrial fibrillation, heart valve disease, and acute myocardial infarction. Despite these limitations, this study is important because it provides detailed follow-up information about patients who contracted RSH during hospitalization for cardiac reasons.

## Conclusion

Although RSH is rare, its prevalence increases among patients receiving anticoagulant and antiaggregant treatments for cardiac reasons. The mortality rates of RSH reported in literature are low, but in our study, the mortality rate markedly increased among patients who contracted RSH during hospitalization for cardiac reasons, had comorbidities, and suffered from additional complications due to extended hospitalization. Therefore, RSH is a condition that needs to be considered in differential diagnosis because it may cause serious morbidity and/or mortality in patients receiving anticoagulant and antiaggregant treatment for cardiac diseases who have low hct levels and newly developed abdominal pain.

Conflict of interest: None declared.

## REFERENCES

1. Linhares MM, Lopes Filho GJ, Bruna PC, Ricca AB, Sato NY, Sacalabrini M. Spontaneous hematoma of the rectus abdominis sheath: a review of 177 cases with report of 7 personal cases. Int Surg 1999;84:251–7.
2. Cherry WB, Mueller PS. Rectus sheath hematoma: review of 126 cases at a single institution. Medicine (Baltimore) 2006;85:105–10. [CrossRef]
3. Klingler PJ, Wetscher G, Glaser K, Tschmelitsch J, Schmid T, Hinder RA. The use of ultrasound to differentiate rectus sheath hematoma from other acute abdominal disorders. Surg Endosc 1999;13:1129–34.
4. Salemis NS, Gourgiotis S, Karalis G. Diagnostic evaluation and management of patients with rectus sheath hematoma. A retrospective study. Int J Surg 2010;8:290–3. [CrossRef]
5. Alla VM, Karnam SM, Kaushik M, Porter J. Spontaneous rectus sheath hematoma. West J Emerg Med 2010;11:76–9.
6. Fitzgerald JE, Fitzgerald LA, Anderson FE, Acheson AG. The changing

nature of rectus sheath haematoma: case series and literature review. Int J Surg 2009;7:150–4. [CrossRef]
7. Berná JD, Garcia-Medina V, Guirao J, Garcia-Medina J. Rectus sheath hematoma: diagnostic classification by CT. Abdom Imaging 1996;21:62–4.
8. Hildreth DH. Anticoagulant therapy and rectus sheath hematoma. Am J Surg 1972;124:80–6. [CrossRef]
9. Siu WT, Tang CN, Law BK, Chau CH, Li MK. Spontaneous rectus sheath hematoma. Can J Surg 2003;46:390.
10. Maharaj D, Ramdass M, Teelucksingh S, Perry A, Naraynsingh V. Rectus sheath haematoma: a new set of diagnostic features. Postgrad Med J 2002;78:755–6. [CrossRef]
11. Verhagen HJ, Tolenaar PL, Sybrandy R. Haematoma of the rectus abdominis muscle. Eur J Surg 1993;159:335–8.
12. Berná JD, Zuazu I, Madrigal M, García-Medina V, Fernández C, Guirado F. Conservative treatment of large rectus sheath hematoma in patients undergoing anticoagulant therapy. Abdom Imaging 2000;25:230–4.
13. Bownik H, Afsar-manesh N, Jakoi A. A growing problem: A case of rectus sheath hematoma. Proceedings of UCLA Healthcare 2010;14:1– 4.
14. DeLaurentis DA, Rosemond GP. Hematoma of the rectus abdominis muscle complicated by anticoagulation therapy. Am J Surg 1966;112:859–63. [CrossRef]
15. Moreno Gallego A, Aguayo JL, Flores B, Soria T, Hernández Q, Ortiz S, et al. Ultrasonography and computed tomography reduce unnecessary surgery in abdominal rectus sheath haematoma. Br J Surg 1997;84:1295–7.
16. Fukuda T, Sakamoto I, Kohzaki S, Uetani M, Mori M, Fujimoto T, et al. Spontaneous rectus sheath hematomas: clinical and radiological features. Abdom Imaging 1996;21:58–61. [CrossRef]
17. Luhmann A, Williams EV. Rectus sheath hematoma: a series of unfortunate events. World J Surg 2006;30:2050–5. [CrossRef]
18. Ozucelik DN, Neslihan Y, Emet M, Coskun S. Spontaneous rectus sheath hematoma presenting with acute abdominal pain: a case series and review of the literature. Ann Saudi Med 2005;25:250–4.
19. Dubinsky IL. Hematoma of the rectus abdominis muscle: case report and review of the literature. J Emerg Med 1997;15:165–7. [CrossRef]
20. Magill ST, del Prado G, Chiovaro J. Embolization of hemorrhaging rectus sheath hematoma. J Gen Intern Med 2014;29:408–9. [CrossRef]
21. Dağ A, Ozcan T, Türkmenoğlu O, Colak T, Karaca K, Canbaz H, et al. Spontaneous rectus sheath hematoma in patients on anticoagulation therapy. Ulus Travma Acil Cerrahi Derg 2011;17:210–4. [CrossRef]
22. Thomopoulos KC, Mimidis KP, Theocharis GJ, Gatopoulou AG, Kartalis GN, Nikolopoulou VN. Acute upper gastrointestinal bleeding in patients on long-term oral anticoagulation therapy: endoscopic findings, clinical management and outcome. World J Gastroenterol 2005;11:1365–8. [CrossRef]

ORİJİNAL ÇALIŞMA - *ÖZET*

## Kardiyak nedenlerle hastanede yatan hastalarda spontan rektus kılıf hematomu: Tek merkez deneyimi

**Dr. Ebubekir Gündeş, Dr. Durmuş Ali Çetin, Dr. Ulaş Aday, Dr. Hüseyin Çiyiltepe, Dr. Kamuran Cumhur Değer, Dr. Orhan Uzun, Dr. Aziz Serkan Senger, Dr. Erdal Polat, Dr. Mustafa Duman**

Kartal Koşuyolu Yüksek İhtisas Eğitim ve Araştırma Hastanesi, Gastroenteroloji Cerrahisi Kliniği, İstanbul

AMAÇ: Bu yazıda kardiyak patoloji nedeniyle kardiyoloji ve kalp damar cerrahisi kliniklerinde antikoagülan ve antiagregan tedavisi altında spontan gelişen rektus kılıf hematomunun mortalite ile ilişkisi sunuldu.
GEREÇ VE YÖNTEM: Ocak 2010 ve Aralık 2015 tarihleri arasında Kartal Koşuyolu Yüksek İhtisas Eğitim ve Araştırma Hastanesi'nde spontan rektus kılıf hematom tanılı 27 hasta geriye dönük olarak değerlendirildi.
BULGULAR: Hastaların 19'u (%70.4) kadın, sekizi (%29.6) erkek olup yaş ortalamaları 63±12 (dağılım, 32- 84) yıldı. Hastaların tamamında en az bir olmak üzere takip gerektiren ek hastalık mevcuttu. Hastaların 14'ü sadece antikoagülan tedavi, sekizi sadece antiagregan tedavi ve geri kalan beş hasta ise hem antikoagülan hem de antiagregan tedavi almaktaydı. Fizik muayenede olguların tümünde karın alt kadranlarında ağrılı palpabl kitle vardı. Bilgisayarlı tomografideki boyut ve lokalizasyona göre tiplendirildiğinde olguların yedisi Tip 1, altısı Tip 2, 14'ünün ise Tip 3 olduğu saptandı. Olguların 23'üne tıbbi tedavi uygulanırken geriye kalan dört hastayada cerrahi tedavi uygulandı. Sekiz (%29.6) hastada mortalite izlendi.
TARTIŞMA: Sonuç olarak, rektus kılıf hematomu nadir olup kardiyak nedenlerle antikoagülan ve antiagregan alan hastalarda sıklığı artmaktadır. Kardiyak nedenlerle hastane yatışı esnasında rektus kılıf hematomu gelişen, ek hastalıkları olan ve hastanede kalış süresinin uzaması nedeniyle ek komplikasyonlar gelişen hastalarda mortalite önemli ölçüde artmaktadır.
Anahtar sözcükler: Antiagregan tedavi; antikoagülan tedavi; rektus kılıf hematomu.

Ulus Travma Acil Cerrahi Derg 2017;23(6):483–488 doi: 10.5505/tjtes.2017.67672