JKAU: Med. Sci., **Vol. 20** No. 1, pp: 97-110 (2013 A.D. / 1434 A.H.)
DOI: 10.4197/Med. 20-1.7

Intrapulmonary Hemorrhage in Small Vessel Vasculitis: A Case Series from Saudi Arabia

Fatima I. Al Beladi, SSCN**, Suzan M. Attar,** FRCP**, Afnan M. Samman[1], Alya A. Filfilan[1], Haneen A. Ismail[1],**
and **Mariam A. Al-Sheikah[1],** MBBS

Department of Internal Medicine, [1]Faculty of Medicine,
King Abdulaziz University,
Jeddah, Saudi Arabia
f_e_b@hotmail.com

Abstract. Intrapulmonary hemorrhage is a rare, yet serious condition. The aim was to determine the frequency of Intrapulmonary hemorrhage in small vessel vasculitis and to find out factors associated with low mortality. Retrospective study charts of patients hospitalized at King Abdulaziz University Hospital, Jeddah between January 2000 and December 2010 were reviewed. The review included the underlying diseases, laboratory and radiological investigations, treatment modalities and mortality. Data analysis was performed using the Statistical Package for the Social Sciences. Among 36 patients with small vessel vasculitis were followed up during the ten-year period, six (16.7%) fitted the criteria for intrapulmonary hemorrhage. The underlying diseases were systemic lupus erythematosus (n=2; 33.3%), Goodpasture's syndrome (n=1; 16.7%), Wegener's granulomatosis (n=1; 16.7%), and microscopic polyangiitis (n=1; 16.7%); hence, one patient was initially treated as a case of tuberculosis but was later found to have intrapulmonary hemorrhage. The incidence of intrapulmonary hemorrhage in patients with small vessel vasculitis was 16.7%, and the overall mortality rate was 33.3%. Further researches are warranted to explore the relation between mortality and current treatment options and the effect of underlying diseases on the outcome.

Keywords: Intrapulmonary hemorrhage, Goodpasture's syndrome, Pulmonary renal syndrome, Vasculitis, Wegener's granulomatosis.

Correspondence & reprint request to: Fatima I. Al Beladi
P.O. Box 80215, Jeddah 21589, Saudi Arabia
Accepted for publication: 16 October 2012. Received: 20 July 2012.

Introduction

Intrapulmonary hemorrhage (IPH) is an uncommon condition that results from the disruption of the pulmonary or bronchial circulation with subsequent bleeding into the alveolar spaces[1]. The study of several cases suggests that systemic vasculitis and Goodpasture's syndrome (GPS) caused by anti-glomerular basement membrane antibodies are the most common causes of IPH in non-infected/ non-cytopenic patients[2]. Other disorders that cause IPH include systemic lupus erythematosus (SLE), Wegener's granulomatosis and other causes of pulmonary-renal syndrome (PRS). The pathognomonic clinical triad consists of the 3 Hs-hemoptysis, hypoxia and a drop in the hemoglobin level. However, hemoptysis has been reported in only 60% of cases[3], and it might be absent at the time of presentation in up to a third of the patients[4].

Intrapulmonary hemorrhage is a medical emergency that necessitates prompt management. Increased mortality in IPH is still unclear. Although the mortality rates were reported to be as high as 70-90%, more recent reports indicate an improved survival[4]. The aim of this study was to determine the frequency of IPH in the presence of small vessel vasculitis and to find out factors associated with low mortality.

Patients and Methods

A retrospective review of all patients with small vessel vasculitis who were admitted to King Abdulaziz University Hospital (a tertiary care center) from January 2000 to December 2010 was performed. This included all patients who fulfilled the criteria of IPH. The following information was recorded: gender; age; underlying disease and its duration; onset of pulmonary hemorrhage; organs involved in the disease; diagnostic methods, including high resolution computed tomography (HRCT), chest X-ray, bronchoscopy, lung biopsy and pulmonary ultrasonography. Laboratory investigations, including the drop in hemoglobin level, antinuclear antibody (ANA), double-stranded deoxyribonucleic acid (dsDNA), cytoplasmic antineutrophil cytoplasmic antibodies (c-ANCA), perinuclear antineutrophil cytoplasmic antibodies (pANCA), anti-glomerular basement membrane antibodies (anti-GBM Ab), antiphospholipid antibody syndrome (APS), low C3 and low C4. Treatment; including pulse steroids, plasmapheresis, cyclophosphamide, intravenous immunoglobulin (IVIG) and mechanical ventilation; and mortality.

This study excludes patient with pulmonary infection, severe coagulopathy (international normalized ratio > 3.5), neoplasms, history of trauma, toxicity and autoimmune diseases other than small vessel vasculitis (SLE, Wegener's granulomatosis, GPS, APS, microscopic polyangiitis, Henoch-Schönlein purpura, Churg-Strauss syndrome, inflammatory bowel disease, cryoglobulinemia, rheumatoid arthritis, Sjögren's syndrome (SS), Behçet 's syndrome and PRS).

Intrapulmonary hemorrhage was diagnosed based on at least three of the following criteria: 1) Pulmonary symptoms that could be hemoptysis, dyspnea, hypoxemia, tachypnea or cough; 2) new infiltrates, which spared the apices, on chest radiographs or computed tomography scans; 3) bloody return with hemosiderin-containing macrophages on bronchoalveolar lavage[1,4]; and 4) a drop of hemoglobin of at least 1 g/dL or hemoglobin level < 6 g/dL. Diagnosis of Wegener's granulomatosis was based on the assessment of disease progress, serological assessment (ANCA antibody titers) and histopathological examinations[6]. Goodpasture's syndrome was diagnosed by the presence of anti-GBM antibodies[7]. Pulmonary-renal syndrome was suggested by pulmonary symptoms and accompanying hematuria or renal dysfunction[8].

To determine the prognostic factors of the outcomes, the patients were divided into two groups; the survivors and the non-survivors. The underlying diseases, treatment modalities and treatment initiation times were compared between the two groups.

Statistical Package for the Social Sciences (SPSS, version 18; Chicago, IL, USA) for data analysis was used. Numerical results were expressed as means and attribute results were expressed as frequencies and percentages.

Results

From January 2000 to December 2010, six cases of IPH out of 36 patients with small vessel vasculitis were admitted to King Abdulaziz University Hospital met the criteria for inclusion. The overall frequency of IPH in patients with small vessel vasculitis in this study was 16.7%. Table 1 summarizes the patients' demographic data, underlying diseases and involved organs. The mean age of the patients was 45.2 years (range, 17-71 years).

***Diagnoses*:** As shown in Table 1, the underlying diseases were systemic lupus erythematosus (SLE) in two cases (patients 1 and 2), GPS in one case (patient 6), Wegener's granulomatosis in one case (patient 4) and microscopic polyangiitis in one case (patient 3). Amongst the patients identified with IPH, PRS coexisted with GPS in one case (patient 6) and with microscopic polyangiitis in another case (patient 3). Patient 5 also fulfilled the criteria for IPH; however, he was initially managed as a case of TB because of his clinical presentation. This patient had no history of systemic diseases, neoplasm, toxicity or trauma. Further investigations revealed negative results for serial sputum acid fast bacilli tests, polymerase chain reaction for *Mycobacterium tuberculosis*, C-ANCA, and rheumatoid factor, but positive for pANCA. He died shortly before the final diagnosis was established.

Table 1. Demographics, underlying diseases and extrapulmonary involvement in the patients.

Patient	Age*	Sex	Underlying Disease	Associated Organs
1	22 years	Female	SLE	Kidneys + lungs
2	17 years	Female	SLE	Kidneys + lungs
3	54 years	Female	Microscopic polyangiitis	Kidneys + lungs
4	53 years	Male	Wegener's granulomatosis	Kidneys + brain
5	71 years	Male	Misdiagnosed as TB infection	Lungs
6	54 years	Male	Goodpasture's syndrome	Kidneys + lungs

Abbreviations: SLE, systemic lupus erythematosus; TB, tuberculosis
**Mean age: 45.2 years*

***Disease Duration and Onset of IPH*:** These were difficult to precisely analyze. Patient 1 had SLE for an uncertain duration before admission to our hospital. She presented with episodic hemoptysis and dyspnea prior to the diagnosis of IPH and was subsequently transferred to the intensive care unit (ICU). Similarly, patient 3 had microscopic polyangiitis for an uncertain duration was admitted to our hospital when she presented with massive hemoptysis that started on the day of admission in addition to fever, tachypnea, and hypoxemia. She was admitted to the ICU as a case of IPH. Patient 2 had an episode of IPH, and she received pulse steroids, cyclophosphamide and plasmapheresis. The patient with Wegener's granulomatosis (patient 4) had recurrent symptoms for 8 years. On admission, he presented with dyspnea, hypoxemia, and respiratory distress and was transferred to the ICU. Patient 5 was initially diagnosed based on his clinical presentation. Following his negative results for *Mycobacterium tuberculosis* in sputum

specimens, C-ANCA and pANCA were ordered 15 days later when he met the criteria for IPH; the results of pANCA were positive. The patient who had GPS and PRS (patient 6) complained of hemoptysis, coughs and dyspnea for 10 days prior to admission at our hospital.

***Associated Organs*:** Table 1 shows that the most commonly involved organs were the kidneys and lungs. Brain involvement was detected in one patient, who was diagnosed with Wegener's granulomatosis (patient 4).

***Diagnostic Criteria*:** Table 2 summarizes the methods used to diagnose IPH in this study. The hemoglobin level was assessed in all patients, and the mean was 3.8 g/dL (range, 1.2-5.7). The mean drop in hemoglobin level was 1.7 g/dL (range, 1.2-2.6), and the mean duration of the drop in hemoglobin was 18.3 days (range, 2.0-46.0). Intrapulmonary hemorrhage was diagnosed by HRCT in four patients (66.7%). Chest X-rays were done in all patients. In patient 4, HRCT did not show bilateral ground glass appearance and a mobile X-ray was ordered for him. The radiographs were of poor quality, thus, the examination was not repeated. Only one patient (16.7%) had bronchoscopy, which revealed hemosiderin-containing macrophages. Regarding pulmonary symptoms, five patients (83.3%) presented with hemoptysis; dyspnea was observed in four cases (66.7%) (Fig. 1 and Table 2).

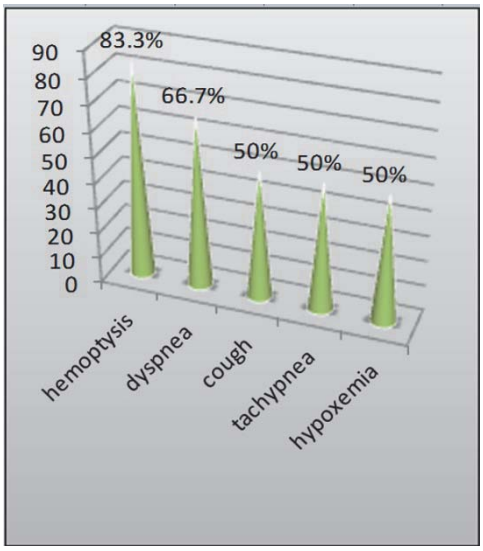

Fig. 1. The frequency of presenting symptoms (in percentages) is shown. The most frequent presenting symptom was hemoptysis, observed in 5 patients (83.3%).

Table 2. Diagnostic methods and presentation in the patients.

Patient	Pulmonary Presentation	Hb Drop or Hb[a] Level	HRCT findings	CXR findings	Bronchoscopy Findings	Lung Biopsy	Pulmonary US
1	Hemoptysis + dyspnea	L = 5.70 g/dl	Yes: ground glass appearance	Yes: multiple bilateral opacities	No	No	No
2	Dyspnea + hemoptysis + hypoxemia + cough + tachypnea	D= 4.31 g/dl	No	Yes: bilateral ground glass appearance	No	No	No
3	Hemoptysis + hypoxemia + tachypnea + dyspnea + fever	L= 4.20 g/dl	No	Yes: bilateral opacities	No	No	No
4	Dyspnea + hypoxemia + tachypea	D= 2.60 g/dl	Yes: peribronchovascular mild opacity	Yes: bad quality (mobile CXR) [b]	No	No	No
5	Hemoptysis	D= 1.20 g/dl	Yes: significant diffuse peribronchial thickening bilaterally and biopsy was recommended for differential diagnosis.	Yes: bilateral nodular air space disease	No	No	No
6	Hemoptysis	L= 4.60 g/dl	Yes: bilateral ground glass appearance	Yes: bilateral infiltrate /hemorrhage	Yes: bronchoalveolar lavage showed hemosiderin - containing macrophages	No	No

Abbreviations: CXR chest X-ray; D, drop from baseline; Hb, hemoglobin; HRCT, high resolution computed tomography; L, level; US, ultrasound.

[a] *Mean Hb drop: 1.7 g/dL after a mean of 18.3 days.*
Mean Hb level: 3.8 g/dL

[b] *This patient's mobile chest X-ray was of poor quality as the patient couldn't be transferred to the radiology department for better radiographs. Given his history, IPH could not be excluded.*

Laboratory Data: Antinuclear antibody (ANA) was ordered in 5 patients (83.3%). The results are presented in Table 3. Double-stranded DNA (ds-DNA) was ordered in 2 (33.3%) patients and the results were negative. C-ANCA was investigated in five patients; the results were negative in 3 (50.0%) patients and positive in 2 (33.3%) cases. pANCA was also checked in five patients. The results were negative in three patients and positive in the other cases. Anti-GBM was done in three patients; the result was positive in one case (patient 6). Complement 3 (C3) was tested in four patients (66.7%) and two of them (33.3%) had low levels. Complement 4 (C4) was also investigated in these four patients and only one had a low level. Subsequently, only one of these four patients had combined low complements (Table 3). Rheumatoid factor was ordered for one patient, and the result was negative.

***Treatment*:** Five patients (83.3%) were treated with pulse steroids, while four patients (66.7%) received plasmapheresis (Table 4). Cyclophosphamide was administered to three patients (50.0%). Patient 5 received anti-TB medications based on his initial diagnosis prior to the suspicion of an autoimmune pathology.

***Mortality*:** Two patients died. Patient 6 died six months after he was diagnosed with GPS that was complicated by IPH, while patient 5 died shortly before the diagnosis was established. The overall mortality rate in this report was 33.3%.

Discussion

The frequency of IPH among patients with small vessel vasculitis admitted to our hospital was 16.7%. However, comparing the results of our report with the literature was not possible as most of the studies on IPH were on a single small vessel vasculitis disease or on collagen vascular diseases. In addition, the small size of our sample did not permit us to make relevant comparisons.

In this report, IPH was the first manifestation of the underlying disease in two patients. This was the case for patients 5 and 6 who presented initially with hemoptysis. After thorough investigations, patient 6 was diagnosed with GPS and PRS that manifested initially as IPH. On the other hand, patient 5 was initially diagnosed and managed as a case of TB. Further investigations were negative for TB and positive

Table 3. Laboratory investigations performed in the patients.

Patient	ANA		ds-DNA	C-ANCA	pANCA	Anti-GBM Ab	APS	Low C3	Low C4	Combined low C3 and C4	Others
	Titer	Pattern of immunofluorecence									
1	1/80	Not specified	-ve	-ve	-ve	-ve	Not done	Yes	No	No	No
2	1/60	Not specified	Not done	Not done	Not done	Not done	Not done	Yes	Yes	Yes	No
3	1/40	Not specified	Not done	-ve	+ve	-ve	Not done	No	No	No	No
4	1/320	Not specified	-ve	+ve	-ve	Not done	Not done	No	No	No	No
5	1/80	Not specified	Not done	-ve	+ve	Not done	Not done	Not done	Not done	Not done	No
6	Not done		Not done	+ve	-ve	+ve	Not done	Not done	Not done	Not done	No

Abbreviations: ANA, anti-nuclear antibody; ds-DNA, double-stranded DNA; C-ANCA, cytoplasmic antineutrophil antibody; pANCA, perinuclear antineutrophil antibody; anti-GBM Ab, anti- glomerular basement membrane antibody; APA, anti-phospholipid antibody; C3, complement 3; C4, complement 4; -ve, negative; +ve, positive.

Table 4. Underlying diseases, treatment and outcome in the patients.

Patient	Underlying Disease	Treatment								Mortality
		Pulse Steroids		Plasmapheresis		Cyclophosphamide		IVIG	MV	
			Initiation After Bleeding/hr		Initiation After Bleeding /hr		Initiation After Bleeding/hr			
1	SLE	Yes	120 mins	Yes	192	Yes	264	No	Yes	No
2	SLE	Yes	2 hours	Yes	Unknown	Yes	Unknown	No	No	No
3	MPA	Yes	Immediately	Yes	Unknown	Yes	72	No	Yes	No
4	WG	Yes	96 mins	No	-	No	No	No	Yes	No
5	MIS-Dx: TB (#)	Managed as TB								Yes
6	GPS	Yes	72 days	Yes	312	No	-	No	No	Yes

Abbreviations: GPS, Goodpasture's syndrome; Hr, hour; IVIG, intravenous immunoglobulins; MV, mechanical ventilation; mins, minutes; Mis-Dx, misdiagnosed as; MPA, microscopic polyangiitis; PRS, pulmonary renal syndrome; TB, tuberculosis; WG, Wegener's granulomatosis.
(#): Serial sputum acid fast bacilli tests and Mycobacterium bovis polymerase chain reaction results came out negative. The patient was negative for cytoplasmic antineutrophil cytoplasmic antibodies but positive for perinuclear antineutrophil cytoplasmic antibodies.

for pANCA. Given the history of this patient, he was included in this study as it was possible that IPH was the first manifestation of small vessel vasculitis in this case. This patient might have had any of the small vessel vasculitides that went unrecognized given his negative medical history of toxicity, iatrogenic, infections and other systemic diseases. Moreover, the clinical findings and investigations pointed towards a possible undergoing autoimmune pathology and hence, IPH. Intrapulmonary hemorrhage can be the initial presentation of a severe illness that can go unrecognized for months[9]. According to the literature, when pulmonary hemorrhage is the only condition and there is no systemic finding, iatrogenic cause, toxicity or infection, the following conditions should be considered: GPS, pulmonary-limited microscopic polyangiitis, isolated pauci-immune pulmonary capillaritis and idiopathic pulmonary hemosiderosis[10].

The most common underlying small vessel vasculitis disease in our study was SLE, diagnosed in 2 patients (33.3%). The most common histological lesions described in IPH were Wegener's granulomatosis, GPS, and microscopic polyangiitis[9]. The duration of the underlying diseases was not precisely determined. In some cases, the underlying diseases were diagnosed concomitantly with IPH at initial presentation, and some of the patients had the condition for several years before diagnosis. Therefore, IPH should be suspected in any patient with small vessel vasculitis who fits the criteria for IPH regardless of disease chronicity.

Similar to an earlier report[10], hemoptysis was the most common complaint in our study. Other studies reported renal involvement as the most common extrapulmonary manifestation[4,11]; a finding that was also observed in our study.

Regarding the diagnostic methods, chest X-ray was undertaken in all of our patients, and the results corresponded with the findings of IPH (bilateral new infiltrates, bilateral opacities or air space diseases). In patient 4, chest HRCT findings did not point towards IPH. The performance of a chest X-ray was also not helpful because of the poor quality of the radiographs. Despite the presence of only two criteria for IPH in this patient (pulmonary symptoms and 2.6 g/dL hemoglobin level drop from the baseline), IPH couldn't be excluded. Moreover, patients with intrapulmonary hemorrhage due to GPS may have normal findings on chest radiographs despite the presence of extensive pulmonary

hemorrhage[12]. This was looked into consideration in the assessment of all our patients. Radiologically, it might be difficult to differentiate IPH from pulmonary embolism or chest infection. Notwithstanding, findings of edema or infection in a patient with GPS usually mean that the patient is bleeding and a chest X-ray cannot exclude hemorrhage[13]. Therefore, it is not recommended to depend only on radiographs or symptoms in establishing the diagnosis of IPH since the symptoms are not specific. Laboratory investigations accompanied with bronchoscopy findings, pulmonary function tests and pulmonary biopsy should be taken into consideration as well.

The overall mortality rate among patients with small vessel vasculitis who had IPH in our study was 33.3%. Just as in the case of frequency, the present results could not be compared with those of the literature as a small vessel vasculitis was investigated as a whole. In addition, a valid conclusion regarding the factors associated with mortality was not possible due to our small sample size.

Our study has some limitations because in addition to the small sample size, it is a single-center retrospective study. The small number of subjects; however, was due to the rarity of the condition.

In conclusion, IPH is a relatively rare condition at King Abdulaziz University Hospital. The frequency of IPH in patients with small vessel vasculitis is 16.7%, and the mortality rate is 33.3%. Intrapulmonary hemorrhage usually presents with nonspecific symptoms and radiological findings and it should be strongly suspected in patients with small vessel vasculitis presenting with anemia and pulmonary symptoms. Radiological investigations along with bronchoscopy, pulmonary function tests and lung biopsy should be considered for confirmation. Intrapulmonary hemorrhage in the absence of systemic disease, toxicity, iatrogenic and infections should raise the suspicion of a possible underlying small vessel vasculitis that manifests for the first time as IPH. Further researches are required to test the hypothesis of a relation between mortality and the current available treatment options. In addition, the relation between unfavorable outcomes and underlying diseases should be investigated.

Disclosure of benefit

The authors have no conflict of interest to declare.

References

[1] **Deane KD, West SG.** Antiphospholipid antibodies as a cause of pulmonary capillaritis and diffuse alveolar hemorrhage: A case series and literature review. *Semin Arthritis Rheum* 2005; **35**(3): 154-165.

[2] **Schnabl A, Reuler M, Csernok E, Richter C, Gross WL.** Subclinical alveolar bleeding in pulmonary vasculitides: Correlation with indices of disease activity. *Eur Respir J* 1999; **14**(1): 118-124.

[3] **Albelda SM, Gefter WB, Epstein DM, Miller WT.** Diffuse pulmonary hemorrhage: a review and classification. *Radiology* 1985; **154**(2): 289-297.

[4] **Chang MY, Fang JT, Chen YC, Huang CC.** Diffuse alveolar hemorrhage in systemic lupus erythematosus: a single center retrospective study in Taiwan. *Ren Fail* 2002; **24**(6): 791-802.

[5] **Miszalski-Jamka T, Szczeklik W, Sokołowska B, Miszalski-Jamka K, Karwat K, Grządziel G, Mazur W, Kereiakes DJ, Musiał J.** Cardiac involvement in Wegener's granulomatosis resistant to induction therapy. *Eur Radiol* 2011; **21**(11): 2297-2304.

[6] **Rankin JA, Matthay RA.** Pulmonary renal syndromes. II. Etiology and pathogenesis. *Yale J Biol Med* 1982; **55**(1): 11-26.

[7] **Jara LJ, Vera-Lastra O, Calleja MC.** Pulmonary-renal vasculitic disorders: differential diagnosis and management. *Curr Rheumatol Rep* 2003; **5**(2): 107-15.

[8] **Han TS, Mahon RT.** Wegener's granulomatosis presenting with diffuse alveolar hemorrhage and negative antineutrophil cytoplasmic antibody test. *Military Med* 2006; **171**(8): 781-783.

[9] **Ioachimescu OC, Stoller JK.** Diffuse alveolar hemorrhage: diagnosing it and finding the cause. *Cleveland Clinic J Med* 2008; **75**(4): 258-280.

[10] **Primack SL, Miller RR, Müller NL.** Diffuse pulmonary hemorrhage: clinical, pathologic, and imaging features. *AJR Am J Roentgenol* 1995; **164**(2): 295-300.

[11] **Schwarz MI, Brown KK.** Small vessel vasculitis of the lung. *Thorax* 2000; **55**(6): 502-510.

[12] **Santos-Ocampo AS, Mandell BF, Fessler BJ.** Alveolar hemorrhage in systemic lupus erythematosus: presentation and management. *Chest* 2000; **118**(4): 1083-1090.

[13] **Bowly NB, Steiner RE, Chin WS.** The chest X-ray in antiglomerular basement membrane antibody disease (Goodpasture's syndrome). *Clin Radiol* 1979; **30**(4): 419-429.

النزيف الرئوي المصاحب لحالات التهاب الأوعية الدموية الصغيرة: سلسلة حالات من المملكة العربية السعودية

فاطمة البلادي، وسوزان عطار، وأفنان صمان، وحنين إسماعيل، وعالية فلفلان، ومريم الشيخة

قسم الطب الباطني، كلية الطب، جامعة الملك عبدالعزيز.

جدة – المملكة العربية السعودية

المستخلص.

المقدمة والأهداف.

يعتبر النزيف الرئوي من الأعراض النادرة المصاحبة لعدة أمراض منها التهاب الأوعية الصغيرة. تهدف الدراسة لتحديد نسب حدوث النزيف الرئوي في التهاب الأوعية الدموية الصغيرة ودراسة العوامل المؤدية لأقل نسبة وفاة ممكنة.

طريقة البحث.

أجريت دراسة على ٣٦ حالة شخصت بالتهابات الأوعية الدموية الصغيرة في مستشفى جامعة الملك عبدالعزيز بجدة في الفترة (٢٠٠٠-٢٠١٠). وتم استخلاص الآتي: المعلومات الشخصية، التشخيص المسبب لحدوث النزيف الرئوي، بداية النزيف الرئوي ومدته، الأعضاء المتضررة، الأشعات التشخيصية، التحاليل المخبرية، العلاجات المستخدمة وعدد الوفيات. وتم تحليل العوامل المؤثرة على تطور المرض عبر البرنامج الاحصائي (SPSS).

النتائج.

تم من خلال الدراسة تشخيص ستة مرضى (١٦,٧٪) بالنزيف الرئوي المصاحب لالتهاب الأوعية الدموية الصغيرة حسب المتطلبات

المعينة للتشخيص. الأمراض التي تم دراستها هي: الذئبة الحمراء (٣,٣٣٪)، متلازمة Goodpasture (١٦,٧٪)، ومتلازمة Wegner Granulomatosis (١٦,٧٪)، مرض Microscopic polyangitis (١٦,٧٪). نسبة الوفيات بين المرضى المصابين بنزيف الأوعية الدموية كأحد مضاعفات لالتهاب الأوعية الدموية الصغيرة كانت (٣,٣٣٪).

الاستنتاج والتوصيات.

أوضحت الدراسة نسبة حدوث النزيف الرئوي لدى المرضى المصابين بالتهاب الأوعية الدموية الصغيرة (١٦,٧٪) ونسبة وفاة كالتالي (٣,٣٣٪). نقترح المزيد من الأبحاث لدراسة تأثير العلاجات الحديثة المتوفرة على أمراض التهابات الأوعية الدموية الصغيرة ونسبة الوفاة والنتيجة النهائية للمريض.