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Original Article

Pulmonary Complications in Kidney Transplant Recipients

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ABSTRACT

Background Kidney transplantation recipients are at an increased risk of lung complications due to infectious or non-infectious reasons. We aimed to determine the lung complications after transplantation and what we could do to prevent the complications during the follow-up, retrospectively.

Material and Methods The 296 patients who underwent kidney transplantation surgery in our centre between the years 1999 to 2006 were included in the study.

Results 75% of the patients were male (n: 222). 77% of the patients (n: 228) had a living-related donor. The mean hospitalisation duration in the post-transplantation period was 13.3±9.07 days. During the follow-up, 37.2% of the patients (n: 110) had rejection, and pulse steroid treatments were given to the 74.5% of these patients. In our study, the lung complication development ratio was 16.2%, and 84% of these complications were due to infections. A specific aetiology was not identified in 63.5% of patients. The patients with a living-related donor had more lung complications due to infection ($p<0.05$). We determine that the hospitalisation period following transplantation increases lung complication development ($p<0.05$). The patients with pulse steroid treatment had more lung complications ($p<0.05$).

Conclusions We showed that close follow-up of the patients prevents lung complications, and non-invasive diagnostic methods could be the first considered choice. In addition, our study showed the importance of a multidisciplinary approach to solid organ transplantation patients during the evaluation of complications.

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Introduction

Kidney transplantation affiliates significantly with a patient's life who has dialysis because of their non-functional kidneys with renal failure. In contrast, the immune system is chronically suppressed with immunosuppressive drugs given for life. Complications such as infections, tumours, lymphoproliferative diseases and chronic rejection occur with immune and non-immune mechanisms. Lungs are the organs under an increased risk of complications due to infectious or non-infectious reasons after renal transplantation.1 This study aimed to evaluate pulmonary complications in the post-transplant period in kidney transplant recipients and suggested preventive strategies against these complications in the future.

Material and Methods

This study, which was conducted after ethical approval of Baskent University medical school (E-51173401-900-123354), included 296 renal transplant recipients with pulmonary complications who were transplanted in our centre between January 1999 and July 2006. The patient's files were evaluated retrospectively. Demographic and clinical data were obtained from subjects with abnormal radiology suggestive of pulmonary complications and one or more respiratory signs such as cough, sputum production, hemoptysis, dyspnea, pleuritic chest pain, and decreased partial oxygen pressure, and other constitutional symptoms such as fever, fatigue etc. Patients were thoroughly evaluated for current respiratory disease history, immunosuppression regimens, and other risk factors. The results of general laboratory tests, radiological evaluation of the pulmonary lesion, and specific investigations to isolate causative agents, antibiotic therapy, and response to treatment were also recorded. Analysis was performed using SPSS 15.0 program. Mann-Whitney U Test, λ2 test and Student T Test were used for statistical analysis.

Results

Characteristics of the patients

All patients were transplanted due to chronic kidney disease (CKD). The aetiology of CKD was unknown in 124 (42%) patients. The most frequent etiologies of 172 patients were glomerulonephritis (n: 37, 12%), hypertension (n: 27, 9.1%) and vesicoureteral reflux (n: 20, 6.8%) (*Table 1*). Among 296 renal transplant recipients, 74 (25%) of 296 patients were female, and 222 (75%) were male. The mean transplantation age was 32.4±10.2 years. 228 (77%) of the patients had living-related donors, and 68 (23%) had deceased donors. Antiviral (aciclovir PO), antifungal (fluconazole PO) and antibacterial (TMP-SMX PO) prophylaxis treatments were given for six months to 293 (99%) of the patients. INH prophylaxis was given to 3 (1%) patients for nine months due to positive PPD tests before transplantation, and all patients were

Table 1. Primary disease etiologies of kidney transplant recipients.

Primary disease etiologies	n (%)
Unknown	124 (42)
Known	172 (58)
Glomerulonephritis	37 (12.5)
Hypertension	27 (9.1)
Vesicoureteral reflux	20 (6.8)
Nephrolithiasis	14 (4.8)
FMF	11 (3.8)
Alport syndrome	11 (3.8)
Vasculitis	9 (3.1)
ADPKD	6 (2.1)
SLE	6 (2.1)
Pyelonephritis	5 (1.7)
Nephrotic syndrome	5 (1.7)
Diabetes mellitus	4 (1.4)
Amyloidosis	4 (1.4)
Atrophic kidney	2 (0.7)
Ectopic kidney	2 (0.7)
Others*	9 (2.7)

FMF: familial Mediterranean fever; APKD: autosomal dominant polycystic kidney disease; SLE: systemic lupus erythematosus.

*Hypertension and diabetes (n: 1), Gilbert's disease (n: 1), methylmalonemic acidemia (n: 1), renal tuberculosis (n: 1), renal hypoplasia (n: 1), pregnancy (n: 1), hemorrhage (n: 1) and renal artery stenosis (n: 1), hyperreninemic hyperaldosteronism (n: 1).

vaccinated for influenza and pneumococcus. The mean hospitalisation time after transplantation was 13.3±9.07 days. Rejection which was diagnosed by the transplantation team by biopsy, was developed in 110 (37.2%) patients, and 82 (74.5%) of them were given pulse steroid treatment. The transplantation team determined the dose and duration of steroid treatment.

General demographics and features of the patients with pulmonary complications

Among 296 kidney transplant recipients, 48 (16.2%) patients had pulmonary complications. The male sex ratio was 71% (n: 34) in patients with pulmonary complications and 76% (n: 188) in patients without pulmonary complications (p>0.05). There was no statistically significant difference between the mean transplant ages of patients with and without pulmonary complications (33.5±9.5 vs 32.2±10.3, p>0.05, respectively). 15 of 48 patients had consecutive pulmonary complications (2 times in 10 cases, three times in 2 cases, four times in 1 case, five times in 1 case and six times in 1 case). And by the way, 75 pulmonary complications were detected in our transplant recipients. The mean hospitalisation time after transplantation were 17.9±11.5 days in the patients with pulmonary complication and 12.2±8.3 days in the patients with no pulmonary complications (p<0.05). Prolonged hospital stay was associated with the risk of developing pulmonary complications. Pulmonary complications developed at the earliest on the second day after transplantation and at the latest on the 2,289th day (mean 49.5±5.4 months). Thirty-seven (77%) of 48 patients with pulmonary complications and 191 (83.8%) of 248 patients without pulmonary complications were transplanted from a living donor, and there was no correlation between donor type and development of pulmonary complications (p>0.05). The history of rejection was significantly higher in patients with pulmonary complications than those without pulmonary complications (52% vs 34.3%, p<0.05, respectively).

Symptoms and radiologic findings of the pulmonary complication group

There was more than one symptom in the patients; cough in 51 (68%) patients, fever in 41 (54.7%) patients, sputum in 41 (54.7%) patients, extrapulmonary symptoms in 31 (41.3%) patients, dyspnea in 22 (29.3%) patients and hemoptysis in 2 (2.7%) patients.

Chest radiography was performed in all 75 pul-

Table 2. Radiological findings.

Chest X-ray	n (%)	Thoracic CT	n (%)
Normal	18 (24)	Normal	3 (5)
Pathologic	57 (76)	Pathologic	55 (95)
Heterogeneous density	37 (65)	Consolidation	30 (55)
Congestion	7 (12.3)	Patchy infiltration	8 (14.5)
Pleural effusion	5 (8.8)	Acinar infiltratation	8 (14.5)
Hiler pathology	4 (7)	Nodule	6 (11)
Atelectasis	3 (5.3)	Bronchiectasis	4 (7.2)
Patchy infiltration	3 (5.3)	Ground glass	3 (5.4)
Abscess	2 (3.5)	Pulmonary thromboembolism	3 (5.4)
Nodular lesion	1 (1.8)	Hiler or mediastinal lymphadenopathy	3 (5.4)
Pleural thickening	1 (1.8)	Cavity	2 (3.6)
		Abscess	2 (3.6)
		Alveolar filling	1 (1.8)
		Alveolar filling and ground glass	1 (1.8)
		Septic embolism	1 (1.8)
		Sequel changes	1 (1.8)

monary complications. Thoracic computed tomography (CT) was also performed in 58 cases of pulmonary complications for which chest X-ray images were not diagnostic. Thoracic CT findings were pathological in 12 (20.7%) cases of pulmonary complications with normal chest X-ray images. While chest X-ray was normal in 18 (24%) patients with pulmonary complications, it was pathological in 57 (76%) patients. Pathological findings of chest X-ray images were unilateral in 34 (59.6%), and bilateral in 23 (40.4%) pulmonary complications; 26 (49%) had pulmonary complications in one zone, and 27 (50%) had pulmonary complications in more than one zone. The most common findings were heterogeneous density, congestion, pleural effusion, hilar pathology, atelectasis, patchy infiltration, abscess, nodular lesion, and pleural thickening (*Table 2*).

The findings of thoracic CT were pathological in 55 (94.8%) of 58 pulmonary complications in which thoracic CT was performed. The pathological findings of thoracic CT were unilateral in 20 (36.4%) pulmonary complications, bilateral in 35 (63.6%) complications, at one lobe in 13 (23.6%) pulmonary complications and multilobar in 38 (69.1%) complications. The most frequent findings were consolidation, patchy infiltration, acinar infiltration, nodule, bronchiectasis, ground glass, pulmonary thromboembolism (PTE), hilar or mediastinal lymphadenopathy, cavitary lesion, abscess, alveolar filling pattern and ground glass and septic embolism (*Table 2*).

Infectious pulmonary complications

Sputum samples were taken from 28 of 41 pulmonary complications and evaluated with gram strain and cultures. 20 (71.4%) sputum samples were culture positive. 13 sputum samples were stained with Erlich Ziehl Nielsen (EZN), and the results were culture-negative. Fiberoptic bronchoscopy (FOB) was performed in 11 pulmonary complications who had no sputum and in 3 pulmonary complications whose sputum cultures were negative. For differential diagnosis, bronchial lavage was performed in 1 complication, bronchoalveolar lavage (BAL) in 7 complications, BAL and pro-BAL in 5 complications and BAL and biopsy in 1 complication. All samples were evaluated with gram strain and cultures. 12 FOB samples were stained with EZN, and the results were negative. 3 (21.4%) FOB samples were culture-positive. Thoracentesis (T/S) was performed in 4 complications with pleural effusion; exudative effusion was determined in 2 with negative cultures. FOB and deep tracheal aspiration (DTA) were performed in 4 complications who were intubated; 2 (50%) of DTA samples were cultures positive (*Table 3*).

It was applied to one of the three pulmonary complications (33.3%) in FOB and DTA. In the FOB sample culture, *M. tuberculosis* was positive, and *Enterobacter aerogenes* and *Candida spp.* were positive in DTA sample cultures. In one complication, cytomegalovirus (CMV) pneumonitis was shown in the FOB biopsy sample, and *C. albicans* were grown in culture. In sputum cultures, grown of *S. epidermidis* and grown of *Enterobacteria* in 1 patient with congestion were considered contamination. After all assessments, 12 (16%) of 75 pulmonary complications were diagnosed as non-infectious and 63 (84%) infectious complications. A specific agent was shown in only 23 (36.5%) of 63 infectious complications. The diagnosis was acute bronchitis in 9 (14.3%) complications, abscess in 2 (3.2%) complications, septic embolism in 1 (1.6%) complication and pneumonia in 51 (80.9%) complications.

The culture result was negative in one pulmonary complication, but with high serum LDH, hypoxemia and radiological findings, *Pneumocystis jirovecii* pneumonitis (PCP) was diagnosed. In six complications, the diagnosis was fungal pneumonia. In the first case of fungal pneumonia, S. epidermidis was grown in sputum culture, but this growth was considered contamination. Also, there was no growth in FOB samples, but *A. fumigatus* was shown in the skin lesion and was accepted as an etiological agent. In the second case, there were white plaques on the mucosa in FOB; yeast in the gram strain, but the culture results were negative. In the third case, there was yeast in the gram strain of the FOB sample, but the culture results were negative. The fourth case was diagnosed as invasive candidiasis; sputum cultures were positive for *P. aureginosa* and *C. albicans*, and hyphae were shown in the FOB biopsy sample. In the fifth case, CMV pneumonitis was demonstrated in the FOB biopsy sample, and the FOB culture was positive for *C. albicans*. The sixth case was intubated due to respiratory failure. BAL culture was positive for *M. tuberculosis*, and DTA culture was positive for

Table 3. Culture results in sputum, fiberoptic bronchoscopy and deep tracheal aspiration samples.

Sputum (n: 28)	n (%)
Culture (−)	8 (28.6)
Culture (+)	20 (71.4)
α-hemolytic streptococcus	5 (17.9)
Hemophilus influenza	3 (10.7)
Pseodomonas auregenosa	2 (7.1)
Enterobacteria	2 (7.1)
Pseodomonas auregenosa and *S. pneumonia*	1 (3.6)
Pseodomonas auregenosa and *C. albicans*	1 (3.6)
S. pneumonia	1 (3.6)
Enterococcus	1 (3.6)
α-hemolytic streptococcus and *Hemofilius influenza*	1 (3.6)
S. pneumonia and *Hemofilius influenza*	1 (3.6)
Enterobacteria and *α-hemolytic streptococcus*	1 (3.6)
S. epidermidis	1 (3.6)
Fiberoptic bronchoscopy (n: 13)	
Culture (−)	10 (76.9)
Culture (+)	3 (23.0)
Candida albicans	1 (7.7)
M. tuberculosis	1 (7.7)
Burkholderia cepacia	1 (7.7)
Deep tracheal aspiration (n: 4)	
Culture (−)	2 (50)
Culture (+)	2 (50)
Acinetobacter baumannii	1 (25)
Enterococcus ve *Candida spp.*	1 (25)

Enterococcus and *Candida spp.*; but before the culture results, the patient died. Cold agglutinins were positive in one of the complications diagnosed as pneumonia, and the case was diagnosed as atypical pneumonia according to the history, physical examination and radiological findings. In other pneumonia cases, an agent can not be defined or diagnosed with defined etiologies.

Non-infectious pulmonary complications were congestion in 7 (58.3%) complications, atelectasis in 3 (25%) complications and PTE in 2 (16.6%) complications.

Comparison of the patients with infectious and non-infectious pulmonary complications

Four (33.3%) of 12 non-infectious pulmonary complications were female, and 8 (66.7%) were male. 16 (25.4%) of 63 infectious pulmonary complications were female, and 47 (74.6%) were male. There was no statistically significant difference between the groups in gender ($p>0.05$). The mean transplantation ages of non-infectious and infectious pulmonary complication groups were 33.1±9.4 years and 33±9.8 years, respectively. There was no statistically significant difference between the groups in mean transplantation age ($p>0.05$). In the non-infectious complication group, 7 (11.5%) complications had living-related donors, and in the infectious complication group, 54 (8.5%) complications had living-related donors. Infectious pulmonary complications were

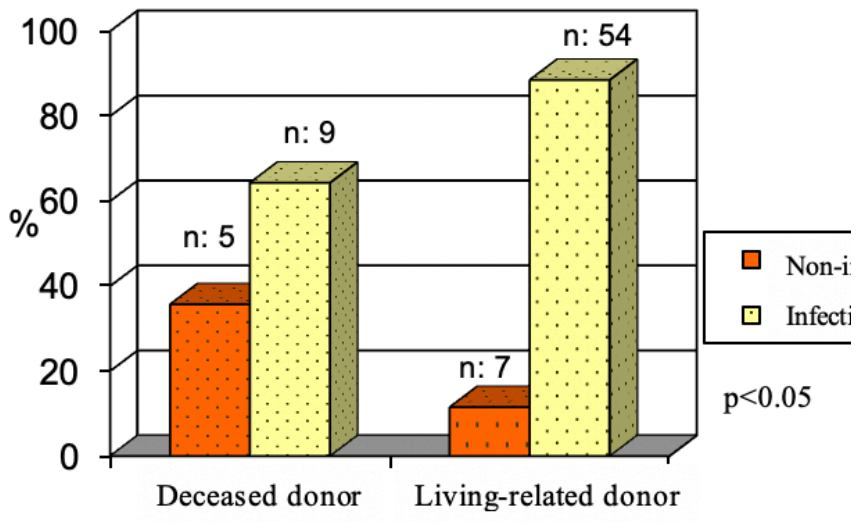

Figure 1. Frequency of infectious lung complications by donor types.

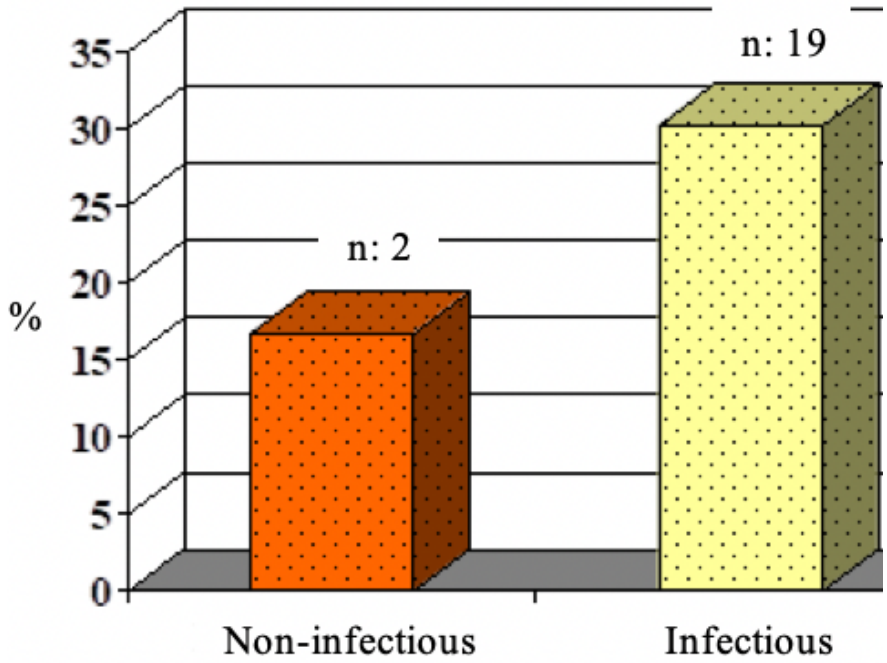

Figure 2. Pulmonary complication rates were higher in transplant recipients who were given pulse steroid treatment for rejection ($p<0.05$).

frequent in the patients with living-related donors (p<0.05) (*Figure 1*). Time of complication occurrence was 588.8±576.9 (range 0-2,289) days in the infectious complication group and 571.8±487.5 (range: 7-1,353) days in the non-infectious complication group (p>0.05) (the mean following time was 4.5 years). The mean hospitalisation time after transplantation was 17.1±11.9 days in the infectious complication group and 17.5±7.8 days in the non-infectious complication group (p>0.05). Rejection was developed in 6 (50%) complications in non-infectious complications and 34 complications in infectious complications (p>0.05).

Pulse steroid treatment

Pulse steroid treatment was given to the patients who were diagnosed with rejection. The transplantation team defined the dose and duration of the treatment according to the patient's situation. Pulse steroid treatment was applied to 14 (53.8%) recipients who developed rejection, while 12 (46.2%) did not receive treatment. Pulmonary complications were more frequent in the patients who were given pulse steroid treatment for rejection (p<0.05) (*Figure 2*). Pulse steroid treatments were given to 19 (30.2%) infectious pulmonary complications and 2 (16.7%) non-infectious pulmonary complications. There was no statistically significant difference between pulse steroid therapy and the occurrence of infectious or non-infectious pulmonary complications (p>0.05). The mean pulse steroid dose in infectious pulmonary complications was 3026.3±1989.3 mg.

Specific laboratory findings in pulmonary complications

Urine legionella antigen was studied in 10 patients with pulmonary complications; serum galactomannan in 14 patients; serum CMV pp65 antigen in 9 patients, and cold agglutinins in 8 urine legionella antigen was negative in 10 patients. Serum galactomannan was positive in one patient, serum CMV pp65 antigen was positive in two patients, and cold antigens were positive in two patients. □*-hemolytic streptococcus* was grown in the patient's sputum culture whose cold agglutinins were positive, and there was no growth in the sputum culture of the second patient with positive cold agglutinins. The patient with positive galactomannan was diagnosed with septic embolism. One of the patients with positive serum CMV pp65 antigen was diagnosed with CMV pneumonitis, and the preemptive treatment was given to the other patient in whom any microbiological agent could show. PPD test was performed in 6 patients with pulmonary complications and was positive (>5 mm) in 3 of them, but none were diagnosed with tuberculosis. One of the patients with negative PPD was diagnosed with tuberculosis due to the growth of *M. tuberculosis* in the FOB sample culture.

Treatment

The patients with pulmonary complications were treated through their diagnosis. Chest physiotherapy and mucolytic agent N-acetyl cysteine for atelectasis; diuretic agents, hemodialysis for congestion and transudative effusions; albumin replacement for hypoalbuminemia and anticoagulant treatment for PTE were given. Antibiotics were given to infectious pulmonary complications through the possible aetiology or defined microbiological agents. The rate of development of pulmonary complications after kidney transplantation in our centre was 25.3%. In the follow-up, 4 of these 48 patients died because of pulmonary complications. The cause of the death was sepsis due to pneumonia.

Discussion

Close follow-up is essential in these patients, as immunosuppressive treatment continues life-long and the immune system is suppressed chronically, so infections, lymphoproliferative diseases, and chronic rejection with or without immune mechanisms might occur, and dialysis could be required again. Transplant success rates are increasing with new immunosuppressive regimens.[1]

Solid organ transplant recipients are at an increased risk of infectious complications due to chronic immunosuppression. Pulmonary infections are one of the frequent causes of mortality in immunosuppressive patients.[2,3] Immunosuppressive drugs prevent rejection but increase the susceptibility to community-acquired and opportunistic infections. Also, the risk of non-infectious pulmonary complications is higher in these patients.[4-7] The risk of pneumonia is lower in kidney

transplant recipients than other solid organ transplant recipients due to the method of surgery and lower dose of immunosuppressive drugs.[8,9] Tveit et al.[10] described the risks of pneumonia in kidney transplant recipients who had no pneumonia history before as age (>65 years) and male gender. The pulmonary defence mechanism gets worse with age.[11] In our study, 48 (16.2%) of 296 patients had pulmonary complications, and none had lung disease before the transplantation. Our results showed no relationship between the mean transplantation age and posttransplant infectious or non-infectious pulmonary complications. In our group, the highest transplantation age (57 years) was lower than the literature, the patients had no history of lung diseases, and all our patients had received low-dose conventional immunosuppressive regimens and T-cell antibodies combination regimens as low-dose immunosuppressive drugs. By the way, our rate of pulmonary complications (25.3%) was lower. In the pulmonary complication group, 34 (71%) patients were male, and 14 (29%) patients were female, similar to the literature, whereas there was no correlation between age and developing pulmonary complications in our group.

Transplant recipients with deceased donor organs are supposed to receive higher doses of immunosuppressive drugs, and more severe and frequent infectious complications are thought to arise.[10,12,13] In one study, there was no statistically significant difference between having a deceased or living-related donor and developing an infection.[14] But another study showed that bacterial infections were frequent in transplant recipients with living-related donors.[15] Our results showed no relationship between donor type and developing pulmonary complications, but infectious pulmonary complications were frequent in patients with living-related donors. In our group, the number of patients with living-related donors was higher in the rejection group. This could be the reason for this result. Intensive immunosuppressive treatments are given for rejection, increasing the risk of pulmonary infections. The prolonged hospitalisation after transplantation, immobilisation and extended exposure to nosocomial flora increase the risk of developing pulmonary complications. Also, we showed that the increased mean hospitalisation time after transplantation increases the rate of pulmonary complications.

In our study, 12 (20.7%) of 75 pulmonary complication chest X-rays were normal, whereas thoracic CT findings were pathological. In the literature, if there is a clinical suspect, thoracic CT is recommended to define the pulmonary pathology in renal transplant recipients for early diagnosis and treatment.[16] Studies showed that consolidation was the frequent pathology in chest X-rays and thorax CTs.[17-19] Heterogeneous density in chest X-rays and consolidation in thoracic CTs were the frequent pathologies in our study. Also, we showed that every pathological finding was not specific to an infectious complication or a microbiological agent. Radiology also defines other than infectious complications.

Despite all research, a specific microbiological agent could not be defined in immunosuppressive patients.[20] In the study of Rano et al.[21], a particular aetiology was determined in 46% of sputum cultures, 64% of DTA samples and 36.1% of FOB samples. They diagnosed 66% of the patients by FOB and could not define a specific aetiology in 12.5% of them. Chang et al.[17] could not determine a particular aetiology in 22.8% of the patients (10/27). BAL's diagnostic value was 80.5% in Xaubet et al.'s[22] study and 75.7% in Kalra et al.'s[23] study. Eyuboglu et al.[24] showed a specific aetiology in 66% of sputum samples and 11% of BAL samples. In their results, BAL was not superior to sputum cultures.[24] In one study that included 33,479 kidney transplant recipients, 4.7% of the patients were hospitalized due to pneumonia.[10] FOB was performed in 9.9% of the patients, and open lung biopsy in 4.8% of the patients. A specific aetiology could not be defined in 72.5% of the patients, and the authors indicated that invasive diagnostic methods were not superior to non-invasive methods. Our study's results were similar; we could not define a specific aetiology in 63.5% of our patients. In our research, the best method to determine specific aetiology was sputum cultures. A particular aetiology was shown in 20 (71.4%) sputum samples, 4 (28.4%) FOB samples and 2 (50%) DTA samples. In case of suspicion of infectious pulmonary complications, empiric antibiotic therapy was given to transplant recipients, and FOB was performed under this treatment. This

should be the reason for our negative culture. Invasive diagnostic methods like FOB in immunosuppressive patients can cause new colonizations and complications, whereas they do not contribute to the diagnosis. So non-invasive diagnostic methods should be the first choice for diagnosing the complications, and invasive methods should be considered in the presence of requirements.

Corticosteroids have catabolic effects, especially in lymphoid and connective tissue, muscles, adipose tissue and skin. They also cause weakness in respiratory muscles; predisposition to infections by affecting T-cells and macrophages in cellular immunity. Also, corticosteroids have anti-inflammatory effects.[25] In our study, there was a relationship between developing pulmonary complications and pulse steroid treatment. Still, there was no statistically significant difference between pulse steroid treatment and developing infectious or non-infectious complications. Recent studies showed that steroid treatment in maintenance therapy was not a risk factor for infectious complications.[15] But corticosteroids in transplant recipients are accepted as risk factors for bacterial infections and sepsis due to intravenous pulse steroid treatment should be emerged.[26] In one study, the cumulative dose of pulse steroid treatment for rejection in patients with pulmonary complications was higher (mean 7,160±1,590 mg), but there was no statistically significant difference.[27] In our study mean pulse steroid dose was 3026.3±1989.3 mg in patients with infectious pulmonary complications.

The potential and defined microbiological agents were compatible with the infection schedule in our patients. With the developing surgery techniques and efficient prophylaxis treatment, the infection agents in solid organ transplant recipients are changing.[28] After kidney transplantation, all recipients were given prophylaxis treatment (acyclovir, fluconazole and TMP-SMX PO). Thus no pulmonary infections due to HSV developed.[29] One of our patients was diagnosed with *P. jirovecii* pneumonitis with his clinic and radiological findings, whereas he was given TMP-SMX prophylaxis treatment. Infection rates of CMV in solid organ transplant recipients approach 70%.[30] In our centre survey for CMV infection is regularly done, and in the occurrence of infection, pre-emptive treatment is given. Gancylovir should be added to the prophylaxis protocol according to the donor's and recipient's CMV serology. There were no recipients with negative CMV serology in our patients, so gancyclovir prophylaxis was not given. Serum CMV PCR and pp65 antigen levels in solid organs could guide the clinician for preemptive or prophylaxis treatment in solid organ transplant recipients. By the way frequency of CMV infection should be decreased, as in our results. On the other hand, CMV causes opportunistic bacterial and fungal infections by suppressing cellular immunity.[31] The growth of *C. albicans* in the BAL sample of one of the recipients diagnosed with CMV pneumonitis was accepted as an opportunistic infection secondary to CMV infection.

In our study, six of the patients died in the follow-up. Four (1.3%) of the deaths were due to sepsis by pneumonia. Two of the deaths due to pneumonia were in the first month after transplantation, and the other two were after six months after transplantation. A specific aetiology was defined in the recipients who died; *A. baumani* in the DTA sample and *M. tuberculosis* in the BAL sample, and *Enterococcus* and *Candida spp.* in the DTA sample. In our centre, by the close follow-up of kidney transplant recipients, we aimed to diagnose mortal infections early and give efficient treatments.

Conclusions

Kidney transplant recipients are at high risk for infectious and non-infectious pulmonary complications due to surgery and immunosuppressive drugs. After the transplantation, efficient prophylaxis treatment should prevent potential infections. Non-invasive diagnostic methods should be the first considered choice in the presence of complications. In transplant recipients, symptoms and radiological findings are not specific. The aetiology of infections might be multifactorial in these patients. Immunosupressive drugs given after kidney transplantation increase infection risk, but close follow-up can prevent infections. Thus, low complication rates should be achieved in both the lungs and other systems. Also, non-infectious reasons should be considered in the presence of complications. The follow-up of transplant recipients must be multidisciplinary as other solid organ transplant recipients. The standard follow-up

transplantation team consisting of surgery and infectious disease specialists and pulmonologists should reach low rates of pulmonary complications.

Conflict of interest

The authors have no conflicts of interest to declare.

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Authors' Contribution

Study Conception: NTS, FOE; Study Design: NTS, FOE; Supervision: FOE, MH; Literature Review: NTS, NGA; Critical Review: FOE; Data Collection and/or Processing: NTS, NGA; Statistical Analysis and/or Data Interpretation: NTS, NGA; Manuscript preparing: NTS.

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