Uluslararası Katılımlı
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9-12 Nisan 2025
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İAH ve Akciğer Kanseri

Dr Gamze KIRKIL

Fırat Üniversitesi Tıp Fakültesi

Göğüs Hastalıkları AD, ELAZIĞ

Epidemiyoloji

İAH hastalarında genel popülasyona göre akciğer kanseri gelişme riski **5 kat** fazla

Naccache JM, et al. J Thorac Dis. 2018

KDH-İAH olanlarda akciğer kanseri gelişme riski KDH olmayan İAH'ye göre 1.7-2 kat daha fazla, insidans %5.5-9

Choi WI, et al. Respir Res. 2019

Watanabe, S., et al. J. Thoracic Dis. 2018

İPF hastasında akciğer kanseri gelişme riski genel popülasyona göre **5-17 kat** fazla

Le Jeune I, et al. Respir Med 2007

Jung HI, et al. Medicine 2018

Cumulative Incidences of Lung Cancer in Various Interstitial Lung Diseases

Takafumi Suzuki, Tsukasa Okamoto, Masako Akiyama, Takayuki Honda, Masaru Ejima, Masahiro Ishizuka, Hiroyuki Sakashita, Yasunari Miyazaki

development was assessed to identify predictors. Results: In all, 587 ILD patients, including 161 IPF, 160 chronic HP, 133 non-IPF idiopathic interstitial pneumonias (IIPs), 87 connective tissue disease-related ILDs (CTD-ILDs), and 46 other ILDs, were included. Twenty-seven patients developed LC. The cumulative incidences of LC at 1, 3, and 5 years were 1.9%, 5.7%, and 12.3% in IPF, respectively; 2.0%, 4.6%, and 11.0% in chronic HP; 0.8%, 0.8%, and 4.0% in non-IPF IIPs; and 1.1%, 1.1%, and 2.9% in CTD-ILDs. Chronic HP patients had a high incidence of LC as IPF patients. Pack-years was associated with LC development in chronic HP patients. Conclusions: The incidences of LC are equally high in patients with chronic HP and IPF.

Table 2. Prevalence, histology, and location of lung cancer in patients with ILDs, based on refs. [45,52,59,62,76,77].

ILD	Prevalence of LC	Most Common LC Histology	Location
IPF	Median 11.6% in European cohorts and 15.3% in Asian cohorts	Squamous cell carcinoma	Lower lobes, fibrotic areas
CTD-ILD	1.9–9%	Adenocarcinoma	Peripheral lung lesions, equal distribution in upper and lower lobes
Hypersensitivity pneumonitis	0–10.6%	Squamous cell carcinoma	Peripheral lung lesions, equal distribution in upper and lower lobes

Drakopanagiotakis F, et al. Cancers 2024

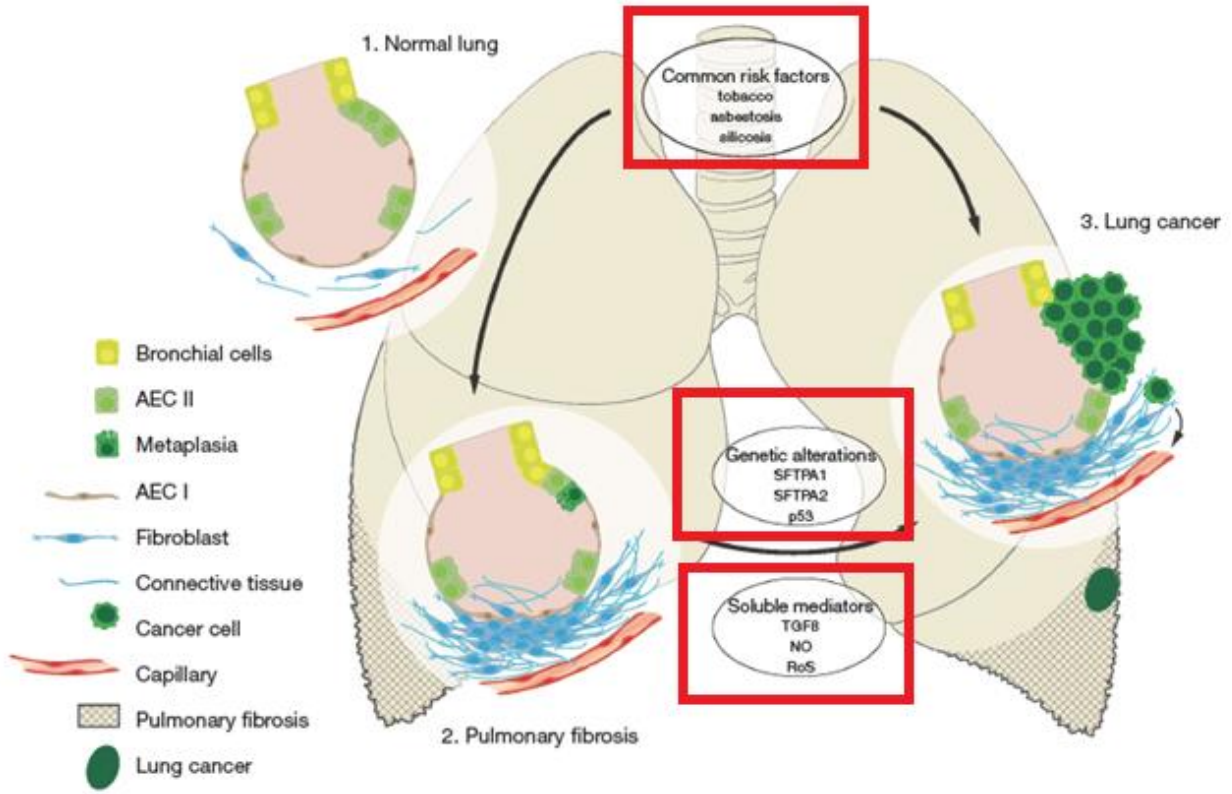
Ortak Patojenik Yolak

Proinflamatuar süreç

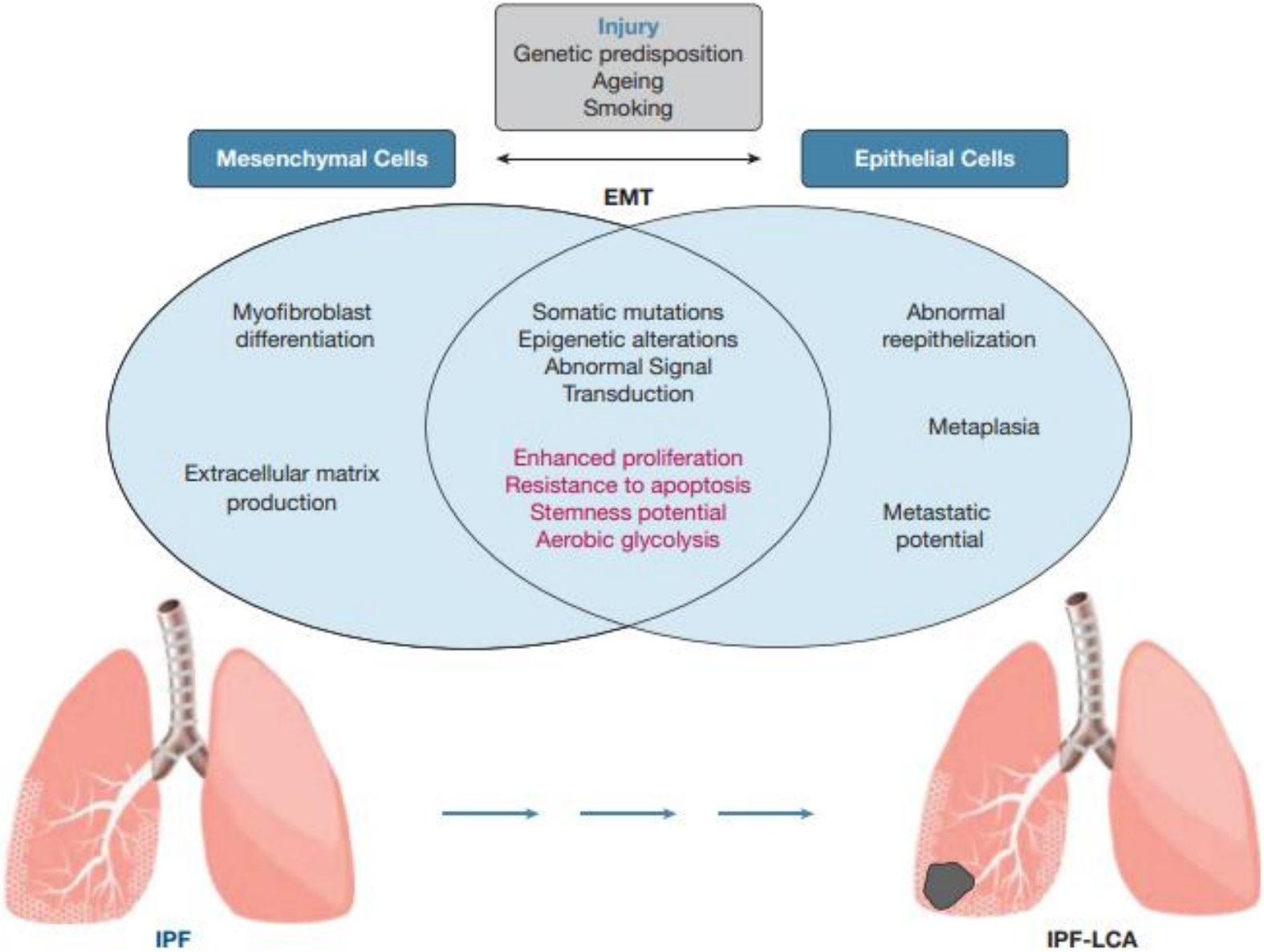
İnflamatuar hücrelerden salınan sitokinler epitel hücre proliferasyonuna neden olur

Bu proliferasyon genetik değişikliklere neden olur

Onkojenler aktive olur
Apoptotik genlere dönüşüm aktive olur
Tümör süpresör genler inhibe olur



Naccache JM, et al. J Thorac Dis 2018



Tzouvelekis A, et al. CHEST 2019

Risk kimlerde daha fazla?

Erkek
Aktif smoker
FVC'de >%10/yıl azalma
Kombine pulmoner fibrozis amfizem varlığı
Kollajen doku hastalığı (RA, SSc) varlığı
İmmünsüpresif tedavi almayan KDH

Yoo H, et al. BMC Pulm Med 2019
Tomassetti S, et al. Chest 2015
Watanabe, S., et al. J. Thoracic Dis. 2018

Table 2
Associations of clinical risk factors with comorbid lung cancer in adults with pulmonary fibrosis.

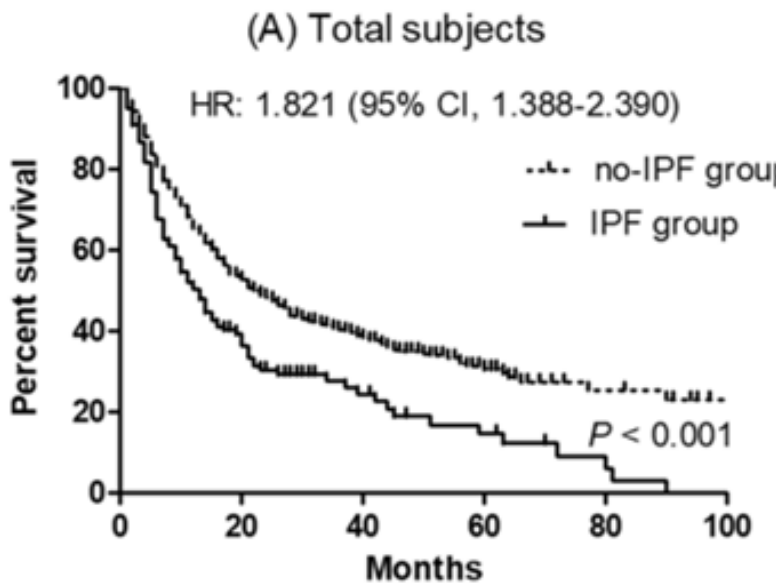
	Univariable Analysis		
	OR	95 % CI	p-value
Age*	1.46	1.14, 1.87	0.003
Male Sex	2.60	1.17, 3.59	0.01
Weight	1.00	0.98, 1.01	0.89
BMI	0.97	0.93, 1.02	0.27
Smoking Status†			
Ever Smoker	8.33	2.51, 27.65	<0.001
Smoking Pack-years‡	1.25	1.14, 1.36	<0.001
Comorbidities			
Hypertension	1.71	0.97, 2.99	0.06
Diabetes	0.99	0.53, 1.84	0.97
Immunosuppression	0.26	0.06, 1.10	0.07
Fibrosis Pattern§			
UIP	4.31	2.11, 8.81	<0.001
CPFE	4.39	2.35, 8.21	0.009
NSIP	1.26	0.45, 3.51	0.66

1366 İAH
Ortalama yaş 67.2 ± 12.4,
639 (%46.8) erkek,
227 (%16.6) hastada akciğer nodülü
55 (%24.3) hastada akciğer kanseri

Dobkin J, et al. Lung Cancer 2025

Fibrozis varlığı surveyi belirgin etkiliyor

122 İPF+AC Ca,
488 AC Ca hastası
5 yıllık survey İPF grubunda %14.5,
İPF olmayan grupta %30.1



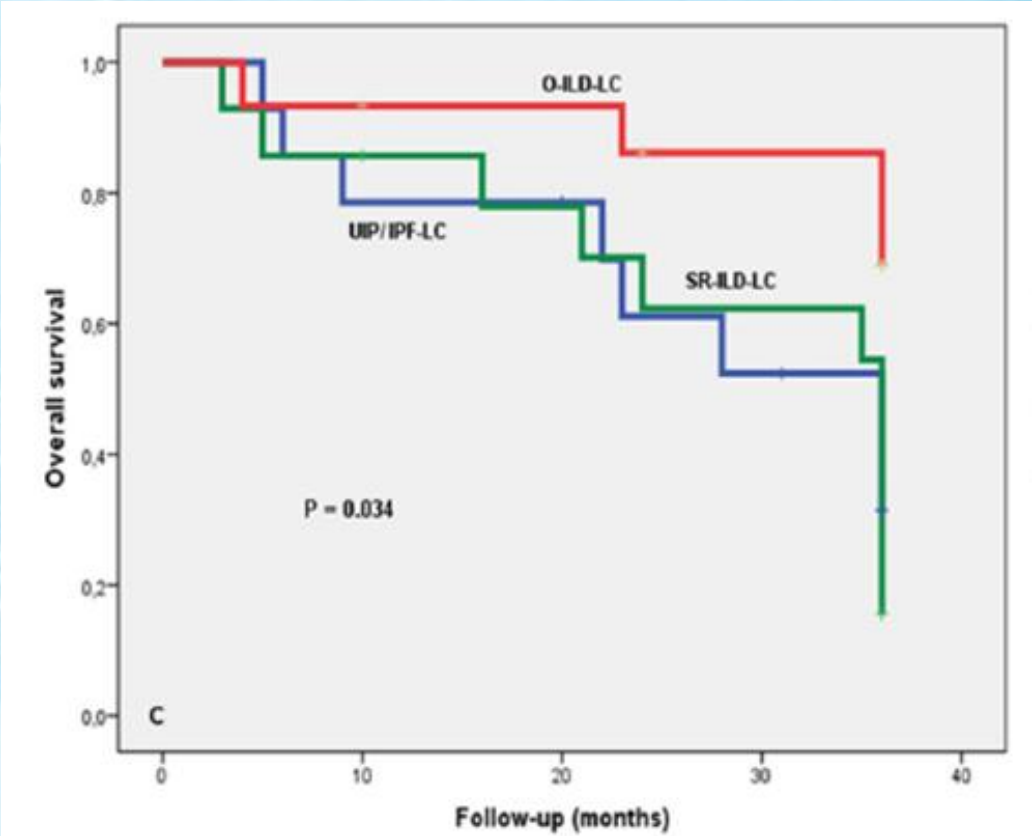
Number at risk

IPF	121	41	16	8	3	0
no-IPF	482	215	100	41	13	7

Kim HC, et al. Scientific Reports 2021

53 fibrozis+kanser;
17 UIP/IPF
17 sigara ilişkili İAH (RB-İAH ve CPFE)
19 diğer (kronik HP, NSİP, PPFE, sarkoidoz, pnömokonyoz, LİP, familyal fibrozis)

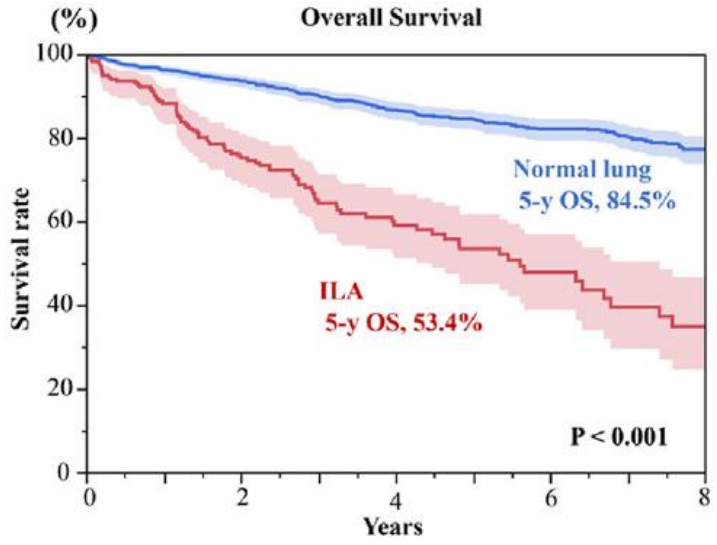
36.ayda survey diğer İAH+kanser hastalarında %70
UIP/IPF hastalarında %30
Sigara ilişkili olanlarda %10



Carobene L, et al. Int Emerg Med 2022

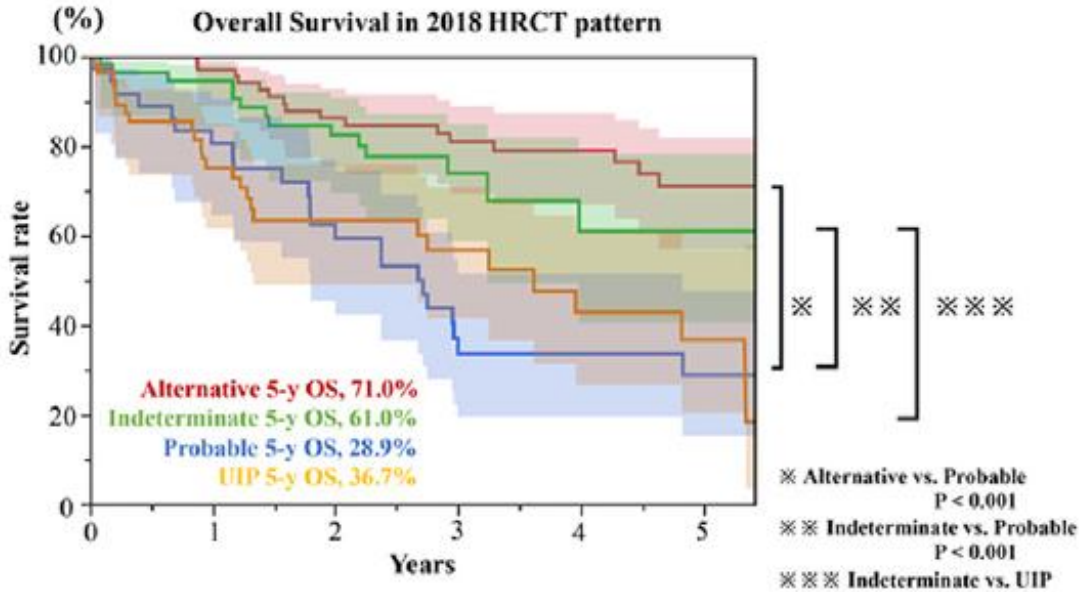
Evre I-III akciğer kanseri nedeni ile pulmoner rezeksiyon yapılan 1503 hasta
preop YRBT'de ILA saptanan 218 hasta
55 hasta %25.2 UIP, 36 hasta (%16.5) olası UIP, 56 hasta (%25.7) belirsiz UIP, 71 hasta (%32.6) alternatif tanı
UIP ve olası UIP paternleri postop survey için bağımsız risk faktörü (HR, 3.12; 95% CI, 1.70-5.73).

A



Patients at risk					
Normal lung	1275	979	675	430	228
ILA	218	138	66	28	12

C



Patients at risk						
Alternative	71	65	54	43	37	25
Indeterminate	56	51	39	18	10	9
Probable	36	29	20	12	8	8
UIP	55	34	25	14	10	5

Fujiwara M, et al. Ann Thorac Surg 2023

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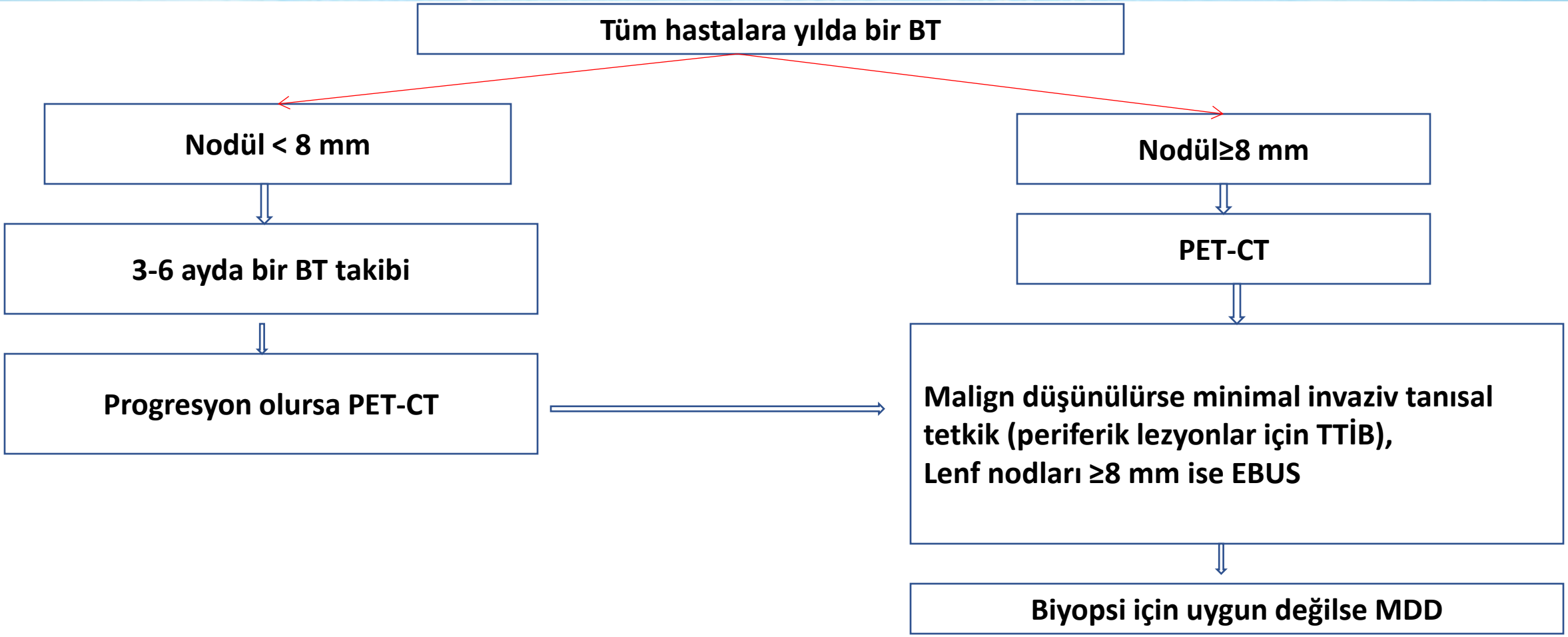
Tanısal Yaklaşım

Non-invasive and invasive strategies for diagnosing and staging lung cancer in the setting of interstitial lung disease.

Non-invasive	**CT** • Diagnosis is challenging due to background architectural distortion • Lung cancer in patients with IPF more often peripheral and in the lower lung zone • Nodules often occur at fibrotic zone of transition and demonstrate rapid growth • Reactive lymph node enlargement common in interstitial lung disease and may or may not represent metastases **FDG PET/CT** • Increased sensitivity for diagnosing local and distant metastases • Reactive lymph nodes often FDG avid
Invasive	**CT-guided PTNB** • Outpatient procedure performed with moderate sedation • Ideal for peripheral lesions Higher risk in IPF (particularly for pneumothorax) • Similar success rate for non-IPF and IPF patients **ENB and EBUS TBNA** • Outpatient procedure performed under general anesthesia • EBUS ideal for central lesions and mediastinal lymph nodes; ENB able to target more peripheral lesions • Lower risk in IPF relative to PTNB • Limited data comparing success in patients without and with ILD

Fisher DA, et al. Radiol Clin North Am. 2022



Tzouvelekis A, et al. Lancet Respir Med 2018

İAH + Akciğer kanseri hastasını tedavi edelim mi?

Tedavinin faydası?

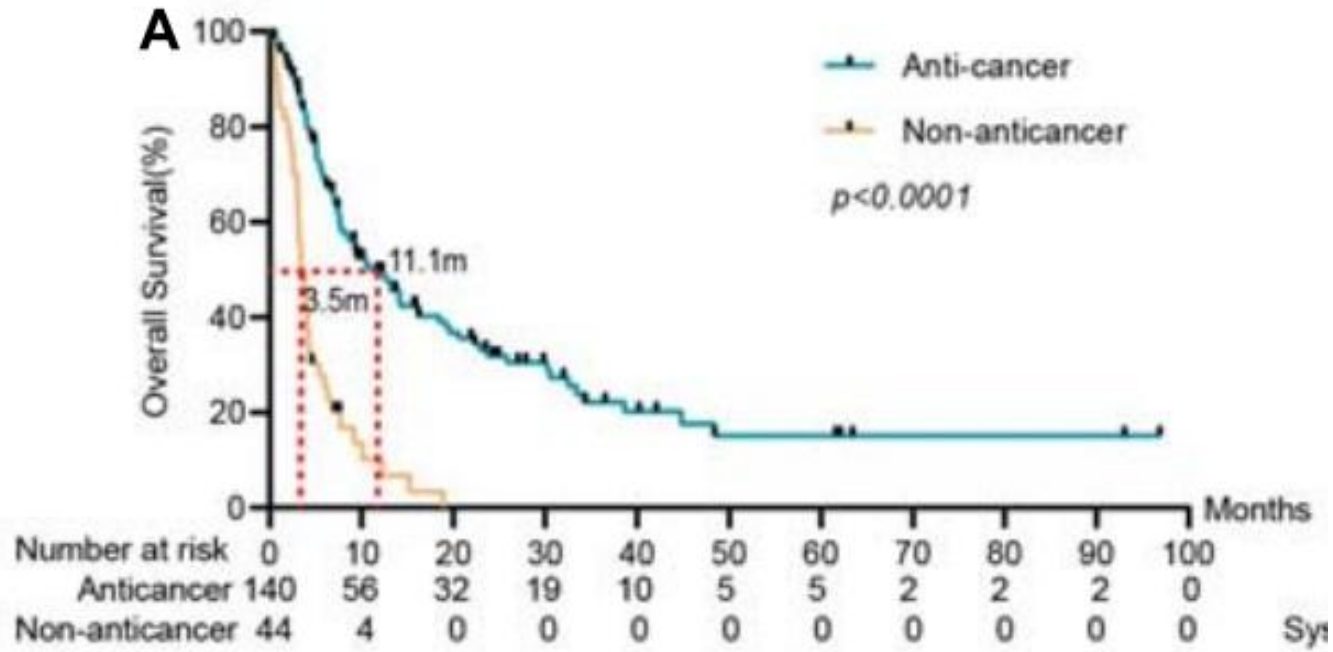
Tedavinin riskleri?

Kanser tedavisinin faydası?

184 İAH (%58.2'si İPF) + akciğer kanseri
%85.3 erkek, %75.5 sigara içici, %67.9 komorbidite, %65.2 ECOG-PS 1

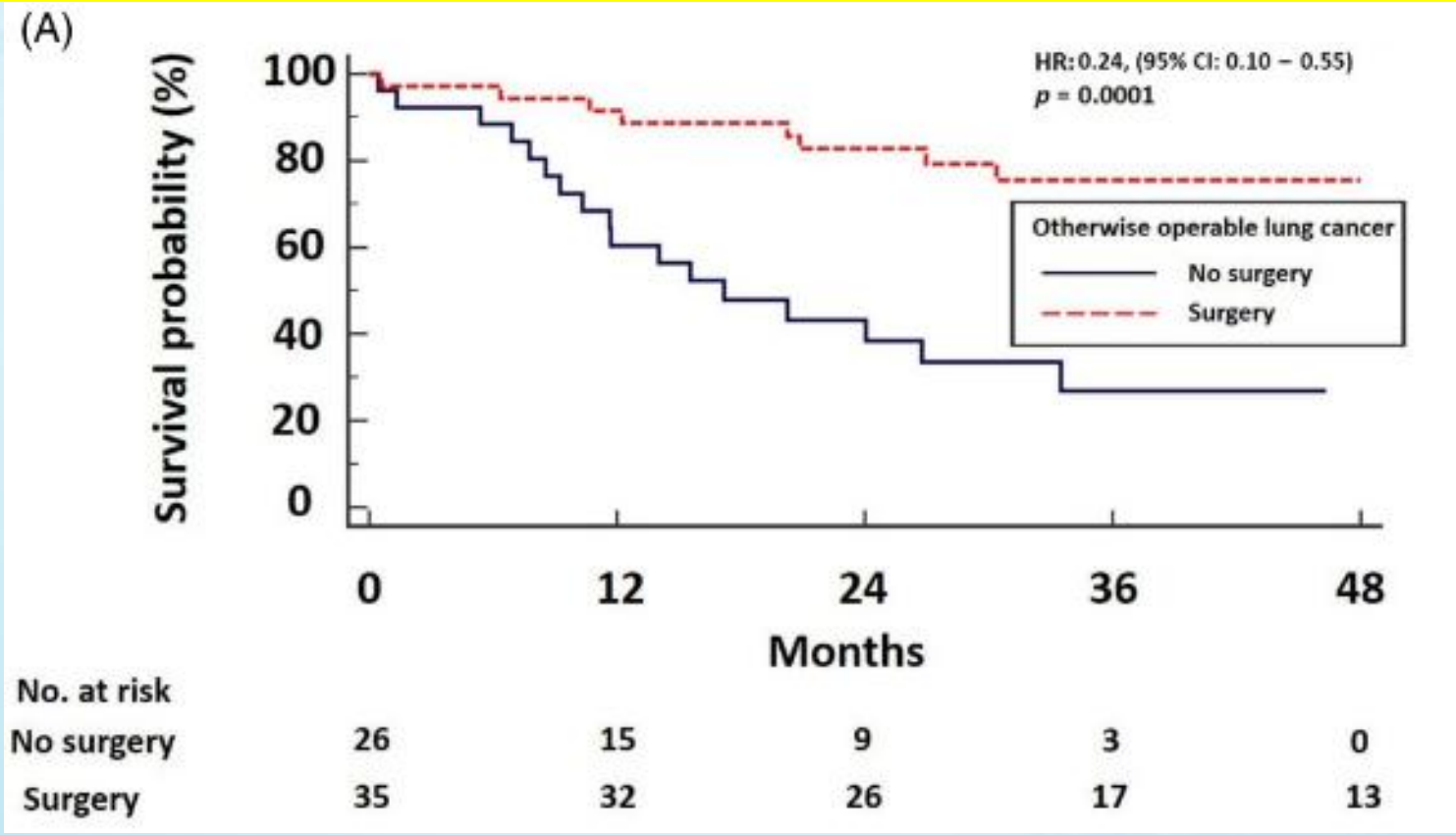
Kanser tedavisi almayanlarda OS 3.5 ay

Kanser tedavisi alanlarda OS 11.2 ay



Xiaohong X, et al. Frontiers in Molecular Biosciences 2021

Cerrahi uygulanan İPF+AC kanseri hastalarında cerrahi uygulanmayanlara göre yaşam süresi daha uzun



Karampitsakos T, et al. Respir 2023

Kanser tedavisinin riskleri?

Akut atak oranları

Birinci basamak KT alanlarda oran %8-13

Wang Y, et al. Front Oncol 2020

Minegishi Y, et al. ERJ Open Res 2020

Docetaxel verilen hastalarda oran %28, Gemcitabine verilenlerde %43

Kenmotsu H, et al. J Thorac Oncol 2011

ICI (nivolumab, pembrolizumab, atezolizumab) kullananlarda oran %14.5-61.5

Nishiyama N, et al. Int J Clin Oncol 2020

Takahara Y, et al. Thorac Cancer 2021

Akut atak oranları

Kemoradyoterapi alanlarda oran %6-83

Saha A, et al. Clin Oncol R Coll Radiol 2022

Cerrahi uygulananlarda %9-23

Zanini U, et al. J Clin Med 2024

Pulmoner rezeksiyon uygulanan İPF hastalarında oran %14-23

Sato S, et al. BMC Pulm Med 2018

Patel AJ, et al. BMJ Open Respir Res 2023

Atak riski öngörülebilir mi?

JACS risk score

Table 1 Risk scoring system for predicting acute exacerbation of interstitial pneumonia after pulmonary resection in lung cancer patients

History of acute exacerbation	5
CT findings: UIP pattern	4
Surgical procedure > wedge resection	4
Preoperative steroid use	3
Male gender	3
KL-6 > 1,000 U/mL	2
% vital capacity	1

Grade of risk	Risk score
Low risk < 10%	< 11
Intermediate risk < 10–25%	11–14
High risk < 25%	> 14

Sato T, et al. General Thoracic and Cardiovascular Surgery 2015

Postoperatif dönemde akut atak gelişiminde risk faktörleri

Yaş>75
İPF varlığı
BT'de bal peteği, >%5 buzlu cam, konsolidasyon varlığı, ana pulmoner arter >28 mm
>4 saat operasyon süresi
Rezeke edilen dokunun genişliği
İleri evre kanser
Postop dönemde steroid kullanılmaması
Karşı AC'de fibrozis skorunun yüksek olması

Naccache JM, J Thorac Dis 2018
Ozawa Y, Am J Roentgenol 2021
Iyoda A, et al. Exp Ther Med 2011
Suzuki H, et al. Surg Today 2011

Kemoterapi alanlarda akut atak gelişimini öngören skorlama

Risk skoru=

(1xantikanser ajan skoru)+(3xsigara öyküsü (>70p/yıl)+(4xsteroid öyküsü)+(3xDLCO<%50)

Skor 0-5 ise atak oranı %12, 6-10 ise %47, >11 ise %66.7

Skorlama sisteminin sensitivitesi %78.6, spesifitesi %67.8

Table 1
Anticancer Agent Score.

Risk Group	Regimen	Score
High (AE frequency ≥30%)	EGFR-TKI, amrubicin, gemcitabine, irinotecan	3
Moderate (AE frequency 11-29%)	pemetrexed, docetaxel, vinorelbin, topotecan, carboplatin/docetaxel	2
Low (AE frequency ≤10%)	carboplatin/etoposide, carboplatin/paclitaxel, cisplatin/vinorelbine, S-1	1
History of second-line chemotherapy	Any regimen	1

Isobe K, et al. Lung Cancer 2018

Kemoterapi alan hastalarda akut atak gelişiminde risk faktörleri

Sigara öyküsü
CRP yüksekliği
Bazal % pred FVC düşüklüğü
Tedavi öncesi Toraks BT'de UIP paterni varlığı
Karşı akciğerde FDG tutulumu
Küçük hücreli akciğer kanseri varlığı

Niwa H, et al. Mol Clin Oncol 2017
Isobe K, et al. Respirology 2010
Minegishi Y, et al. Lung Cancer 2011
Enomoto Y, et al. Lung Cancer 2016
Akaike K, Int J Clin Oncol 2020
Kenmotsu H, J Thorac Oncol 2011

Radyasyon Pnömonitis gelişiminde risk faktörleri

- FVC < %70,
- Normal AC'in > %10 radyasyon uygulaması
- ECOG 2-4
- Skuamöz hücreli karsinom varlığı
- Klinik evre T2
- SBRT öncesi düzenli steroid kullanımı
- Ortalama akciğer dozu ≥ 12 Gy
- Akciğer volümünün ≥ %25'inde İAH varlığı
- Tedavi öncesi PET/CT'de tüm akciğer parankiminde SUV95>1.5 olması

Li F, et al. Radiat Oncol Lond Engl 2021

Onishi H, et al. Cancers 2018

Castillo R, Radiat Oncol Lond Engl. 2014

Akut atak gelişimi önlenebilir mi?

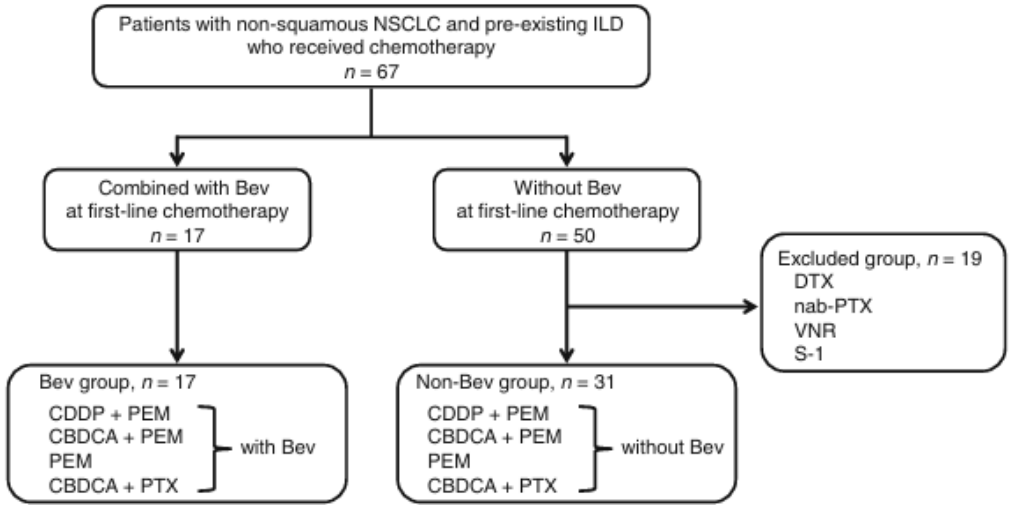
Hamada *et al. BMC Pulmonary Medicine* (2019) 19:72
https://doi.org/10.1186/s12890-019-0838-2

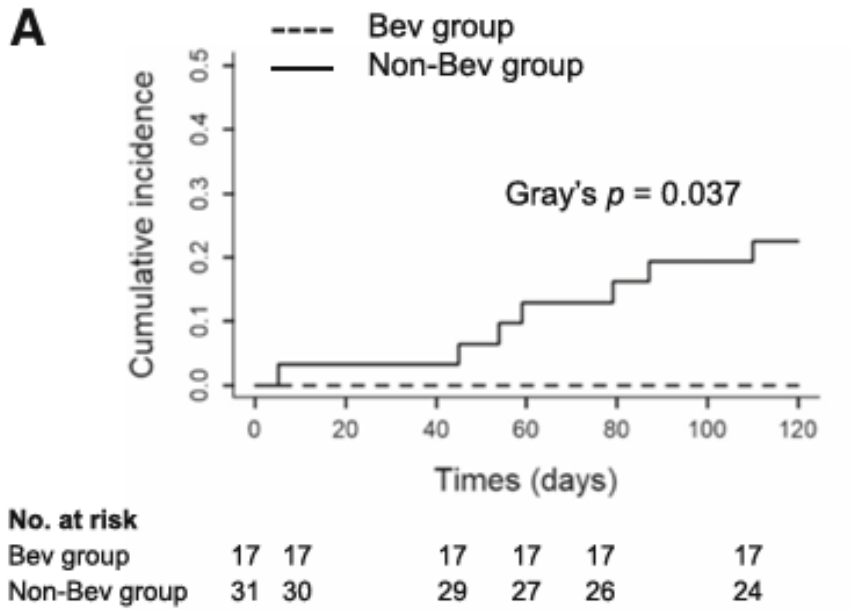
BMC Pulmonary Medicine

RESEARCH ARTICLE Open Access

Protective effect of bevacizumab on chemotherapy-related acute exacerbation of interstitial lung disease in patients with advanced non-squamous non-small cell lung cancer

Shohei Hamada[1†], Hidenori Ichiyasu[1*†], Tokunori Ikeda[2], Megumi Inaba[3], Kosuke Kashiwabara[4], Tomoki Sadamatsu[5], Nahoko Sato[1], Kimitaka Akaike[1], Hiroko Okabayashi[1], Koichi Saruwatari[1], Yusuke Tomita[1], Sho Saeki[1], Naomi Hirata[3], Takeshi Yoshinaga[3] and Kazuhiko Fujii[1]





Birinci basamak KT'den 120 gün sonra
atak insidansı Bev grubunda %0, non-Bev grubunda %22.6, p = 0.037
PFS Bev grubunda 8 ay, non-Bev grubunda 4.3 ay, p=0.026

ORIGINAL ARTICLE

Safety and effectiveness of pirfenidone combined with carboplatin-based chemotherapy in patients with idiopathic pulmonary fibrosis and non-small cell lung cancer: A retrospective cohort study

Yuji Yamamoto, Yukihiro Yano, Tomoki Kuge, Fukuko Okabe, Mikako Ishijima, Takeshi Uenami, Masaki Kanazu, Yuki Akazawa, Toshihiko Yamaguchi & Masahide Mori

Department of Thoracic Oncology, National Hospital Organization Osaka Toneyama Medical Center, Toyonaka, Japan

Thoracic Cancer 11 (2020) 3317–3325

14 İPF+NSCLC hastası
Birinci basamak kemoterapi;
Pirfenidone+Carboplatin+Paclitaxel
Hastalık progrese olunca;
Nivolumab, pembrolizumab

Table 4 Occurrence of acute exacerbations of idiopathic pulmonary fibrosis (AE-IPF) (*n* = 14)

Variable	Events
Until the initiation of second-line chemotherapy, *n* (%)	0 (0.0)
Within 30 days from the last first-line chemotherapy administration, *n* (%)	0 (0.0)
Entire observation period, *n* (%)	4 (28.6)

Surgery Today (2022) 52:736–744
https://doi.org/10.1007/s00595-021-02343-0

REVIEW ARTICLE

Surgical treatment for patients with idiopathic pulmonary fibrosis and lung cancer: postoperative acute exacerbation of idiopathic pulmonary fibrosis and outcomes

Akira Iyoda[1] · Yoko Azuma[1] ·

Perioperatif antifibrotik kullanımı İPF akut atak oranını azaltır

Table 1 Reports on postoperative acute exacerbation of IPF in non-small-cell lung cancer patients with IPF

Author	Published year	Case number	Stage	Perioperative pirfenidone treatment	Rate of acute postoperative exacerbations	Mortality rate
Watanabe A	2008 [33]	56	p- I–IV	–	4 cases (7.1%)	4 cases (7.1%)
Saito Y	2011 [34]	28	p-IA	–	3 cases (10.7%)	1 case (3.6%)
Suzuki H	2011 [35]	28	p-I–IV	–	9 cases (32.1%)	0
Mizuno Y	2012 [36]	52	p-IA-IIIB	–	7 cases (13.5%)	6 cases (11.5%)
Sato S[a]	2018 [32]	13	p-IA, IB	–	3 cases (23.1%)	3 cases (23.1%)
Iwata T	2015 [37]	12	p-I, II or more	Pirfenidone (retro)	0	0
Iwata T	2016 [38]	31	p-IA-IV	Pirfenidone (retro)	1 case (3.2%)	–
Iwata T	2016 [39]	32	p-IA-IV	Pirfenidone (phase II)	1 case (3.1%)	1 case (3.1%)
Sekihara K	2020 [40]	36	p-I–III	Pirfenidone (retro)	3 cases (8.3%)	1 case (2.8%)
Kanayama M	2020 [41]	28	p-IA-IIIA	Pirfenidone (retro)	1 case (3.6%)	–

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İAH + Akciğer Kanseri Tedavisi

Comparison of available lung cancer treatments in the setting of interstitial lung disease.

Treatment	Definitive therapy?	Benefits	Risk of acute exacerbation	Unique risks
Surgery	Yes	Gold standard	0-20%, increases with extent of resection and perioperative factors	Most invasive; permanent reduction in vital capacity
Stereotactic body radiotherapy	Yes	Non-invasive	18-20.5%	Radiation pneumonitis; can reduce lung function
Percutaneous thermal ablation	Yes	Minimally invasive, no permanent decrease in lung function (in absence of acute exacerbation)	18%	Shares some risks with surgery (e.g., pneumothorax, bronchopleural fistula, prolonged air leak)
Systemic therapy	No	Non-invasive	13-50%	Immunosuppression from certain agents/ regimens

Fisher DA, et al. Radiol Clin North Am. 2022

Stage of NSCLC	Treatment Options for NSCLC with ILD
Stage I	- Surgical resection (lobectomy or segmentectomy): for patients with stable and well-controlled ILD, with mild to moderately impaired lung function - Stereotactic body radiation therapy (SBRT): alternative to surgery for patients who are not surgical candidates due to their ILD, due to poor lung function or comorbidities. Option in a minority of very carefully selected patients due to the possible detrimental risk of pneumonitis - Percutaneous image-guided ablation to be considered in patients with poor lung function and small tumors
Stage II	- Surgical resection: for patients with stable and well-controlled ILD, with mild to medium impaired lung function - SBRT: For patients unsuitable for surgery due to ILD, SBRT may be considered. Option in a minority of very carefully selected patients due to the possible detrimental risk of pneumonitis
Stage III	- Radio chemotherapy: standard of treatment for locally advanced NSCLC. Decision to proceed in patients with ILD to be taken in oncology board, considering the high potential risk of pulmonary toxicity. Sequential, instead of concomitant therapy may be associated with reduced risk of pulmonary toxicity. Radiation therapy may be an option in a small minority of very carefully selected patients due to the possible detrimental risk of pneumonitis - Immunotherapy: may be considered in patients with stable ILD and mild to moderately impaired lung function, as part of the treatment regimen
Stage IV	- Systemic therapy: chemotherapy, targeted therapy, immunotherapy, or a combination of the above depending on the patient's performance status, ILD status and comorbidities - Palliative care: in advanced stages of NSCLC or end-stage ILD

Drakopanagiotakis F, et al. Cancers 2024

Gerçek yaşam verisi

140 İAH+Akciğer kanseri

TABLE 3 | Treatment options for the anti-cancer cohort of ILD-LC stratified according to histological type, clinical stage, subtype of ILD, and ECOG-PS score.

	N	Surgery[&]	Chemotherapy	Interventional therapy	Radiotherapy	Anti-angiogenic therapy	Targeted therapy	Immunotherapy	Others
Histology									
Squamous cell carcinoma[#]	42	10 (23.8)	31 (73.8)	3 (7.1)	16 (38.1)	6 (14.3)	2 (4.7)	4 (9.5)	5 (11.9)
Adenocarcinoma	50	8 (16.0)	36 (72.0)	0 (0)	5 (10.0)	14 (28.0)	15 (30.0)	1 (2.0)	1 (2.0)
Other NSCLC	15	4 (26.7)	11 (73.3)	1 (6.7)	2 (13.3)	3 (20.0)	3 (20.0)	1 (6.7)	1 (6.7)
Small cell carcinoma	33	3 (9.1)	31 (93.9)	2 (6.1)	12 (36.4)	6 (18.2)	0 (0)	2 (6.1)	0 (0)
Clinical stage									
I	17	14 (82.4)	10 (58.8)	0 (0)	5 (29.4)	3 (17.6)	1 (5.9)	1 (5.9)	2 (11.8)
II	9	5 (55.6)	8 (88.9)	0 (0)	2 (22.2)	1 (11.1)	0 (0)	0 (0)	0 (0)
III	38	6 (15.8)	30 (78.9)	1 (2.6)	13 (34.2)	8 (21.1)	4 (10.5)	2 (5.3)	3 (7.9)
IV	76	0 (0)	61 (80.3)	5 (6.6)	15 (19.7)	17 (22.4)	15 (19.7)	5 (6.6)	2 (2.6)
ILD classification									
IPF	78	12 (15.4)	60 (76.9)	3 (3.8)	15 (19.2)	17 (21.8)	13 (16.7)	4 (5.1)	6 (7.7)
non-IPF	48	11 (22.9)	40 (83.3)	3 (6.3)	16 (33.3)	6 (12.5)	6 (12.5)	4 (8.3)	1 (2.1)
CTD-ILD	14	2 (14.3)	9 (64.3)	0 (0)	4 (28.6)	6 (42.9)	1 (7.1)	0 (0)	0 (0)
ECOG-PS									
1	99	23 (23.2)	81 (81.8)	2 (2.0)	35 (35.3)	21 (21.2)	8 (8.1)	7 (7.1)	6 (6.1)
2	39	2 (5.1)	27 (69.2)	3 (7.7)	0 (0)	7 (17.9)	12 (30.8)	1 (2.6)	1 (2.6)
3	2	0 (0)	0 (0)	1 (50)	0 (0)	1 (50)	0 (0)	0 (0)	0 (0)

Xiaohong X, et al. Front Mol Biosci 2021

Kemoterapi ne kadar etkili?

Table S5 Efficacy of chemotherapy in various interstitial lung diseases

Citations	n	Histology, n			Stage	Chemotherapy	ILD	Response, %	Control, %	PFS, months	Survival, months	1-year survival, %	AE, n (1st line)	Hematological complications
		AD	SCC	Other										
Shukuya, *Anticancer Res* 2010	15	10	5		IIIA/IIIB/IV/ POR: 1/5/7/2	Carbo (AUC5)-Pacli, n=7; Carbo (AUC2)-Pacli, n=8	IPF, n=4; NSIP, n=9; DIP, n=1; CTD, n=1	33	53	2.5	7	29	4	ND
Minegishi, *Lung Cancer* 2011	18	6	7		IIIA/IIIB/IV/ POR: 2/3/13	Carbo-Pacli	IPF, n=6; Non-IPF, n=12	61	83	5.3	10.6	22	4 [1]	33%
Okuda, *Anticancer Res* 2012	19	10	7	2	IIIA/IIIB/IV/ POR: 4/6/9/5	Carbo-Vino, n=9; CDDP-Vino, n=10	IPF, n=16; Other, n=3	42.1	73.7	4.4	7.4	36.8	15.8 [15.8]	63.2
Kinoshita, *Oncol Lett* 2012	22	11	7	4	IIIA/IIIB/IV/ POR: 1/6/15	Carbo-Pacli, n=19; CDDP-Vino, n=2 CDDP-Doce, n=1	IPF and NSIPi	36.4	77.3	3.2	5.4	ND	3 [3]	ND
Choi, *Cancer Chemoth Pharm* 2014	52	32	13	8	ND	Carbo, n=33; CDDP, n=19; GEM, n=39; PEM, n=13	IILD, n=42; Pneumoconiosis, n=5; CTD, n=5	42.3	78.8	5.4	7.9		7 [3]	ND
Shimizu, *Cancer Chemoth Pharm* 2014¶	21	21			IIIA/IIIB/IV: 3/2/16	Carbo-Pacli, n=11; Carbo-Pacli-Beva, n=10	CT-scan: UIP, n=5; Non-UIP, n=16	27 *vs.* 40 (ns)	90 *vs.* 82	4.4 *vs.* 5.5	9.7 *vs.* 16.1		1 (placebo group)	ND
Enomoto, *Anticancer Res* 2015	25	21	0	4	IIIA/IIIB/ IV/POR: 1/3/16/5	Carbo-Pacli-Beva	IPF, n=13; Non-IPF, n=12	72		7.2	8.5	40	3 including 1 during maintenance Beva	72%
Watanabee, *Anticancer Res* 2015	67	26	21	20	IIIB/IV/POR: 20/42/5	CDDP-Vino	ND	34.3	73.1	3.7	7.4	22.4	7 [7]	60%
Kenmotsu, *Cancer Chemoth Pharm* 2015	104	50	47	7	III A and B/IV/ POR: 41/55/5	Carbo-, n=85; (Pacli, n=63; Pacli-Beva, n=5; S1, n=7; GEM, n=6; other, n=4); CDDP-, n=19 (Vino, n=6; Doce, n=4; S1, n=3; Eto, n=3; other, n=3)	CT-scan: confirmed UIP, n=70; poss. or incompat. UIP, n=34	38	80	4.8	9.9		26 (9 including 2 deaths)	
Kashiwabara, *anticancer*	14/109	ND	ND	ND	?	ND	ND	ND	ND	ND	10.6 *vs.* 27.9			ND

Naccache JM, et al. J Thorac Dis 2018

Table S6 Small-cell lung cancers treated with chemotherapy

Citations	n	Localized	Disseminated	Chemotherapy	IPF/other	Response, %	Control, %	PFS, months	Survival, months	AE, n (1st line)	Hematological complications
Minegishi, *JTO* 2011	17	8	9	Carbo/Eto, n=17	8/9	88.2	94	5.5	8.7	3 [1]	88.2
Watanabee, *Int J Clin Oncol* 2013	11		11	Carbo/Eto, n= 8; CDDP/Eto, n=3	11/0	62.5	75	4.7	7	4 [3]	72.7
Yoshida, *Anticancer Res* 2013	52	29	23	Carbo/Eto, n=22; CDDP/Eto, n=30	ND	69	ND	4.5	9.4	6 [1]	ND

Naccache JM, et al. J Thorac Dis 2018

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RESPIRATORY INVESTIGATION 57 (2019) 512–533

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Respiratory Investigation

journal homepage: www.elsevier.com/locate/resinv

Statement

Summary of the Japanese Respiratory Society statement for the treatment of lung cancer with comorbid interstitial pneumonia

Molecular-targeted drugs and ICI are poorly suited for lung cancer patients with comorbid IP [6–7]. At present, clinicians must decide the therapeutic indications and the regimen based on their own experience. The establishment of a guideline is needed.

6.1.4.1. Selection of chemotherapy for NSCLC with comorbid IP

Table 4 – Study of combination chemotherapy including platinum preparation for NSCLC with comorbid IP

Author	Regimen	N	Study design	AE (%)	Median OS
Minegishi [5]	CBDCA + weekly PTX	18	Prospective	1 (5.6%)	10.6 m
Sekine [6]	CBDCA + S1	21	Prospective	2 (10%)	9.7 m
Shukuya [20]	CBDCA (weekly or monthly) + weekly PTX	15	Retrospective	4 (27%)	7.0 m
Kinoshita [48]	Any	22	Retrospective	3 (14%)	5.4 m
Okuda [27]	Platinum + VNR	19	Retrospective	3 (16%)	7.4 m
Watanabe [26]	CDDP + VNR	67	Retrospective	7 (10.4%)	7.4 m
Shimizu [19]	CBDCA + PTX	11	Retrospective	0 (0%)	9.7 m
Shimizu [19]	CBDCA + PTX + Bv	10	Retrospective	1 (10%)	16.1 m
Enomoto [49]	CBDCA + PTX + Bv	25	Retrospective	3 (12%)	8.5 m
Kenmotsu [18]	Any	104	Retrospective	9 (9%)	9.9 m

AE, acute exacerbation of IP; OS, overall survival; CBDCA, carboplatin; PTX, paclitaxel; VNR, vinorelbine; Bv, bevacizumab.

6.1.4.2. *Selection of chemotherapy for Small-cell lung cancer (SCLC) with comorbid IP*

- The options for first-line chemotherapy in SCLC are limited compared to NSCLC, but a consensus has been reached to make the standard treatment, a platinum preparation with concomitant etoposide therapy, and the standard treatment for cases with comorbid IP [9–12].
- It is expected that second-line chemotherapy also improves prognosis of recurrent SCLC with comorbid IP [13–14]. However, the risk of AE may be greater than in first-line chemotherapy. It is hoped that future research

- Chemoradiation therapy is not recommended for limited-disease (LD)-SCLC with comorbid IP at present, because thoracic radiotherapy carries a high risk of AE of IP [16,17]. It is unknown whether radiotherapy could be possible if patients are selected appropriately.

ERJ OPEN RESEARCH
REVIEW
N. KEWALRAMANI ET AL.

ERJ Open Res 2022; 8: 00115-2022

Lung cancer in patients with fibrosing interstitial lung diseases: an overview of current knowledge and challenges

Namrata Kewalramani[1], Carlos Machahua[1,2], Venerino Poletti[3], Jacques Cadranel[4], Athol U. Wells[5] and Manuela Funke-Chambour[1,2]

TABLE 1 Suggestions for treatment of lung cancer in fibrosing interstitial lung disease (fILD)

Suggestions based on the available studies:
The choice of treatment with chemotherapy should be carefully made by analysis of the risk/benefit balance. Patient selection and careful counselling are crucial.
Carboplatin plus weekly (nab-)paclitaxel for four (or six) cycles remains the first-line standard therapy in fit NSCLC fILD patients without maintenance therapy.
Addition of bevacizumab should be considered in fit nonsquamous NSCLC patients.
Vinorelbine (squamous) and pemetrexed (adenocarcinoma) monotherapy should be administered in a second-line setting.
Carboplatin etoposide for four cycles (or six cycles) remain the standards of care for SCLC.
Drugs that are not recommended: gemcitabine and docetaxel.

fİAH + NSCLC Hastalarında Tedavi

Treatment	Stage I–IB	Stage IIA–IIIA	Stage IIIB/C	Stage IV		
Standard	Lobectomy VATS No adjuvant chemotherapy		Treat as stage IV?	Carboplatin weekly (nab-)paclitaxel (± bevacizumab)	Carboplatin weekly (nab-)paclitaxel (± bevacizumab) Nivolumab/pembrolizumab second line?	Carboplatin weekly (nab-)paclitaxel (± bevacizumab)
Option/not fit	SBRT Sublobar resection	Treat as stage IV?	Treat as stage IV?	Kinase inhibitors?	Mono-chemotherapy Paclitaxel, vinorelbine, pemetrexed	
Frail/oldest patients/PS2	SBRT	Palliative care	Palliative care	Kinase inhibitor	Palliative care	Palliative care

Kanser gelişimini engelleyebilir miyiz?

RESPIRATORY INVESTIGATION 56 (2018) 72–79

Contents lists available at ScienceDirect

Respiratory Investigation

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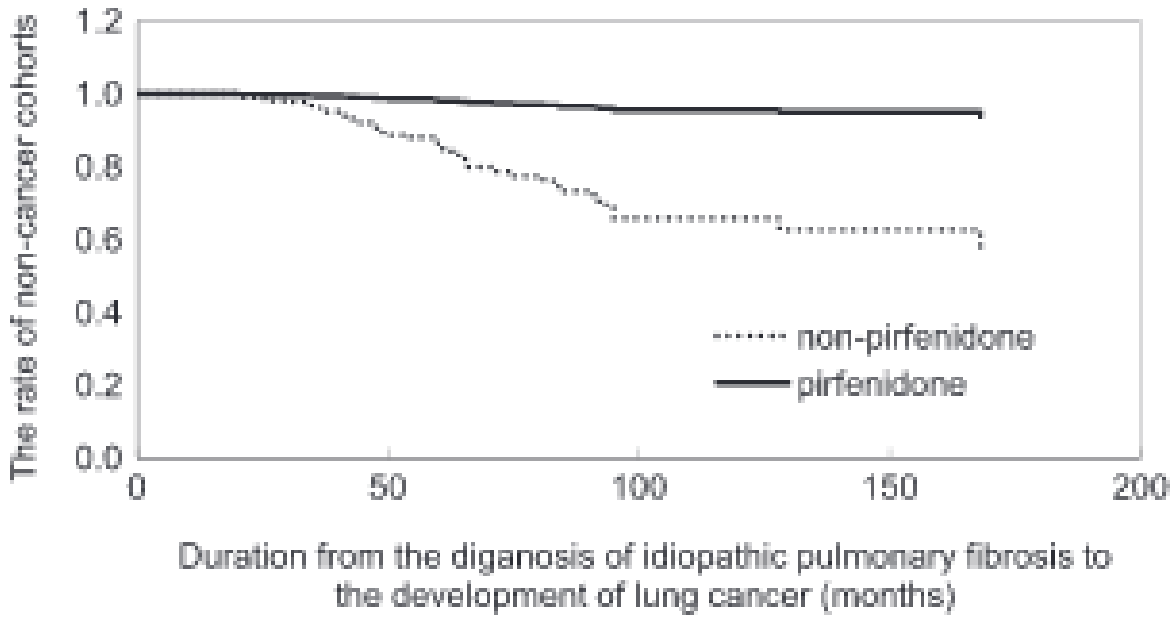
Original article

Reduced incidence of lung cancer in patients with idiopathic pulmonary fibrosis treated with pirfenidone

Yukiko Miura[a,b,*], Takefumi Saito[a], Toru Tanaka[a], Hiroyuki Takoi[a], Yohei Yatagai[a], Minoru Inomata[b], Takahito Nei[c], Yoshinobu Saito[b], Akihiko Gemma[b]. Arata Azuma[b]

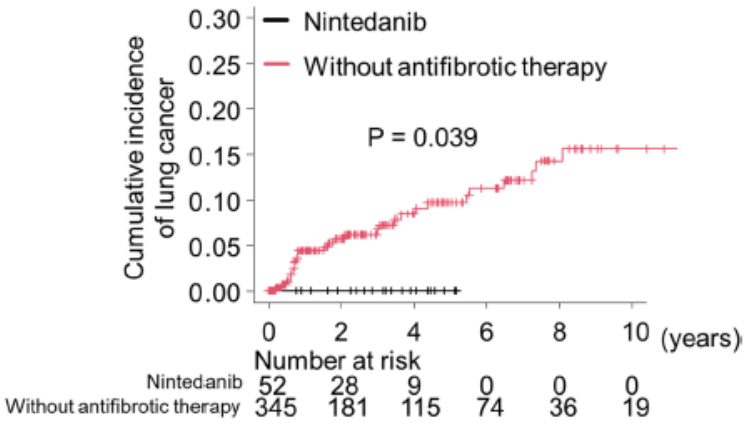
PFD grubunda kanser insidansı %2.4 (2/83), non-PFD grubunda %22.0 (39/178), p<0.0001



Impact of antifibrotic therapy on lung cancer development in idiopathic pulmonary fibrosis

Hyogo Naoi,[1] Yuzo Suzuki ,[1] Kazutaka Mori,[2] Yuya Aono,[1] Masato Kono,[3] Hirotsugu Hasegawa,[4] Koshi Yokomura,[4] Yusuke Inoue,[1] Hironao Hozumi ,[1] Masato Karayama,[1] Kazuki Furuhashi,[1] Noriyuki Enomoto,[1] Tomoyuki Fujisawa , Yutaro Nakamura,[1] Naoki Inui,[1] Hidenori Nakamura,[3] Takafumi Suda[1]

Thorax 2022;**77**:727–730. doi:10.1136/thoraxjnl-2021-218281



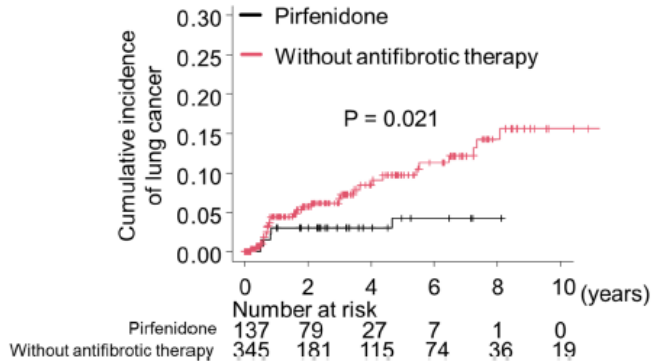


Table 2 Incidence and prevalence of LC and rate of LC-related mortality in patients with IPF treated with or without antifibrotic therapy

	All patients (N=345)	IPF-antifibrotic therapy (+) patients (n=189)	IPF-antifibrotic therapy (–) patients (n=156)	Risk ratio*
LC cases, n	35	5	30	
Incidence, per 100 person-years (95% CI)	2.14 (1.50 to 2.96)	1.07 (0.35 to 2.47)	4.53 (3.08 to 6.41)	0.235 (0.092 to 0.602)
Prevalence, % (95% CI)	10.1 (7.2 to 13.8)	2.65 (0.86 to 6.07)	19.2 (13.4 to 26.3)	0.138 (0.055 to 0.346)
LC-related mortality, % (95% CI)	7.9 (4.7 to 12.1)	1.61 (0.20 to 5.70)	15.2 (9.0 to 23.6)	0.106 (0.025 to 0.450)

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Early View

Original Research Article

Pirfenidone and risk of lung cancer development in IPF: a nationwide population-based study

Hee-Young Yoon, Hoseob Kim, Yoonjong Bae, Jin Woo Song

Please cite this article as: Yoon H-Y, Kim H, Bae Y, *et al*. Pirfenidone and risk of lung cancer development in IPF: a nationwide population-based study. *Eur Respir J* 2024; in press (https://doi.org/10.1183/13993003.01484-2024).

Table 2. Incidence of lung cancer in patients with IPF according to pirfenidone use and treatment duration

	Number of Patients	Lung cancer, Number (%)	Incidence (95% CI), cases per1,000 person-year
Total population	19,614	1,355 (6.9)	17.8 (16.9–18.8)
Pirfenidone usage			
No	9,952	925 (9.1)	27.9 (26.1–29.7)
Yes	9,662	431 (4.7)	10.4 (9.5–11.4)
Duration			
<1 month	11,109	925 (8.3)	24.8 (23.2–26.4)
≥1 month	8,505	431 (5.1)	11.1 (10.1–12.2)
<6 month	13,594	1,083 (8.0)	22.4 (21.1–23.8)
≥6 months	6,020	273 (4.5)	9.8 (8.7–11)
<1 year	15,303	1,189 (7.8)	21.2 (20–22.5)
≥1 year	4,311	167 (3.9)	8.3 (7.1–9.6)
<2 year	17,553	1,282 (7.3)	19.6 (18.6–20.7)
≥2 years	2,060	73 (3.5)	6.8 (5.4–8.5)

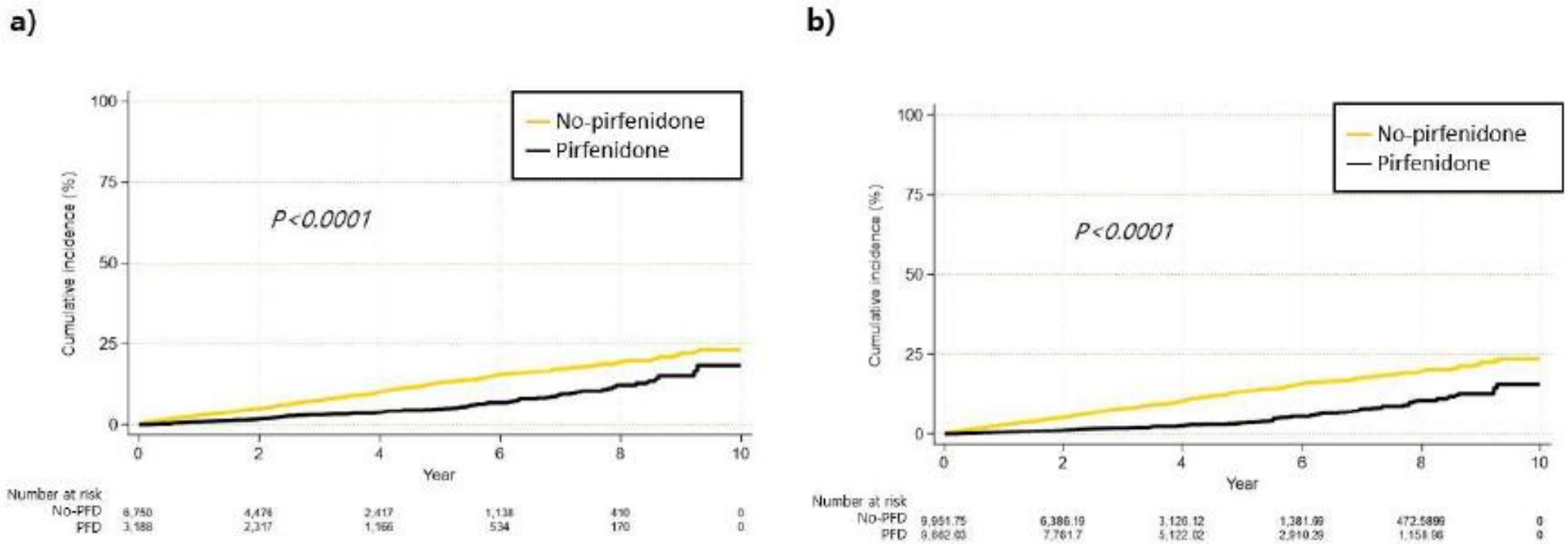

Figure 2. Comparison of cumulative lung cancer incidence between pirfenidone and no-pirfenidone groups in patients with IPF

Kaplan–Meier survival curve analysis with log-rank test was used to compare patients with and without pirfenidone treatment.

(A) Original cohort, and (B) IPTW cohort

IPF, Idiopathic pulmonary fibrosis; IPTW, inverted probability of treatment weighting; PFD,

Table 4. Age- and sex-stratified subgroup analysis for association between pirfenidone and risk of lung cancer in patients with IPF

Variables	Weighted HR		Weighted adjusted HR*	
	HR (95% CI)	*p-value*	HR (95% CI)	*p-value*
Age†		0.941#		0.549#
<65 years	0.351 (0.288–0.429)	<0.001	0.336 (0.274–0.412)	<0.001
≥65 years	0.356 (0.310–0.409)	<0.001	0.356 (0.310–0.410)	<0.001
Sex†		0.119#		0.096#
Male	0.338 (0.300–0.380)	<0.001	0.337 (0.299–0.379)	<0.001
Female	0.503 (0.318–0.796)	0.003	0.519 (0.327–0.823)	0.005

Conclusion: Pirfenidone use may be associated with a reduced lung cancer risk in patients with IPF.

RESEARCH ARTICLE

The preventative effects of statin on lung cancer development in patients with idiopathic pulmonary fibrosis using the National Health Insurance Service Database in Korea

PLOS ONE | https://doi.org/10.1371/journal.pone.0299484 March 13, 2024

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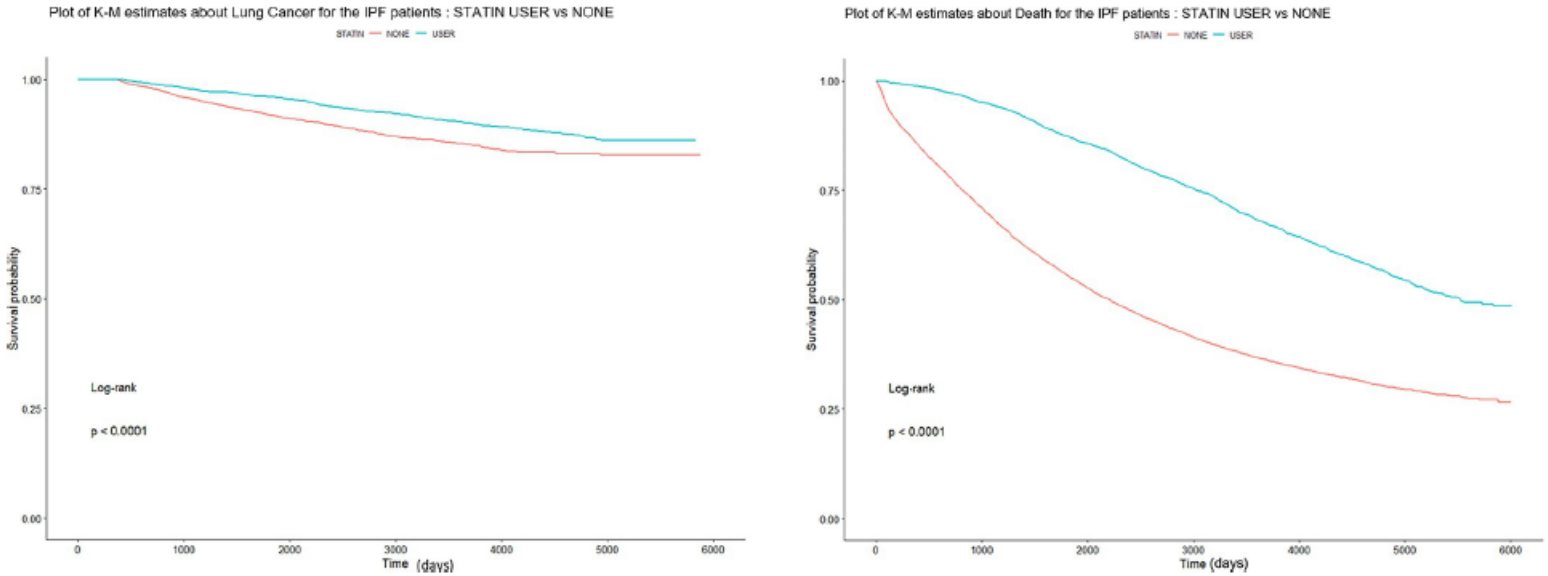

Fig 1. Kaplan-Meier plot displaying cumulative incidence of lung cancer development (A) and overall survival (B) in IPF patients with and without statin use. IPF = idiopathic pulmonary fibrosis, K-M = Kaplan-Meier.

Table 2. Comparison of clinical outcomes in IPF patients with and without statin use.

	Statin non-user (n = 5810)	Statin user (n = 3372)	*P*-value
Lung cancer development in IPF patients (n = 850)	534 (9.2%)	316 (9.4%)	0.803
Duration (days)			
Lung cancer development	2194.6 ± 1601.4	3361.0 ± 1331.2	< 0.0001
IPF diagnosis to death	2413.9 ±1778.7	3741.8 ± 1443.1	< 0.0001
No. of deaths	3887 (66.9%)	1404 (41.6%)	< 0.0001

ORIGINAL ARTICLE

SARCOIDOSIS VASCULITIS AND DIFFUSE LUNG DISEASES 2024; 41 (4): e2024050 DOI: 10.36141/svdld. v41i4.15675

LUNG CANCER IN PATIENTS WITH PULMONARY FIBROSIS: CHARACTERISTICS FEATURES AND PROGNOSIS

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Table 1. IPF-LC and non-IPF-LC Patients' General Demographic Characteristics.

	IPF-LC	Non-IPF PF-LC	
	n:164 (%)	n:87 (%)	p-value
Age	69.7±7.7	67.9±8.2	0.090
Sex			
Male	153 (93.3)	72 (82.8)	**0.015**
Female	11 (6.7)	15 (17.2)	
BMI	26.6±3.8	26.3±3.3	0.561
Smoking history			
Never	14 (8.8)	8 (10.4)	0.232
Former	122 (76.3)	51 (66.2)	
Current	24 (15)	18 (23.4)	0.222
Malignancy history in family	26 (16.3)	18 (22.8)	0.450
Environmental exposure	43 (26.9)	25 (31.6)	
Non-IPF Pulmonary Fibrosis			
Sarcoidosis		4 (4.6)	
Chronic HP		8 (9.2)	
CTD-Associated Interstitial Lung Disease (CTD-ILD)		16 (18.4)	
Rheumatoid arthritis		8 (9.2)	
Systemic sclerosis		3 (3.4)	
Polymyositis/Dermatomyositis		1 (1.1)	
SLE		1 (1.1)	
Mixed-type CTD		3 (3.4)	
CPFE		14 (16.1)	
Unclassified Pulmonary Fibrosis		45 (51.7)	

Table 2. Radiological and PFT results of all cases.

	IPF-LC	Non-IPF PF-LC	
	n:164 (%)	n:87 (%)	p-value
HRCT Patterns			
Definite UIP	116 (70.7)	16 (18.4)	<0.001
Probable UIP	32 (19.6)	6 (6.9)	<0.009
Indeterminate UIP	6 (3.7)	7 (8.0)	0.146
Alternative diagnosis	2 (1.2)	16 (18.4)	<0.001
HRCT Results			
Traction bronchiectasis	115 (70.1)	53 (60.9)	0.159
Honeycomb pattern	103 (62.8)	33 (37.9)	<0.001
Ground glass	55 (33.5)	52 (59.8)	<0.001
Mosaic attenuation	19 (11.6)	11 (12.6)	0.839
Emphysema	62 (37.8)	52 (59.8)	0.001
Mediastinal LAP	79 (48.2)	51 (58.6)	0.144
PFT			
FEV1 (%)	78.69±18.76	78.75±17.69	0.837
FVC (%)	74.06±16.69	78.67±19.52	0.298
FEV/FVC (%)	82.73±9.21	81.34±10.18	0.504
DLCO	48.20±16.63	46.14±17.20	0.930
TLC	64.24±16.57	63.43±22.97	0.139
Annual >10% decline in FVC			
No change	123 (75.0)	74 (85.1)	0.076
Decline	41 (25.0)	13 (14.9)	
Annual >15% decline in DLCO			
No change	128 (78.0)	15 (17.2)	
Decline	36 (22.0)	72 (82.8)	0.238
PHT	45 (27.4)	19 (21.8)	0.559

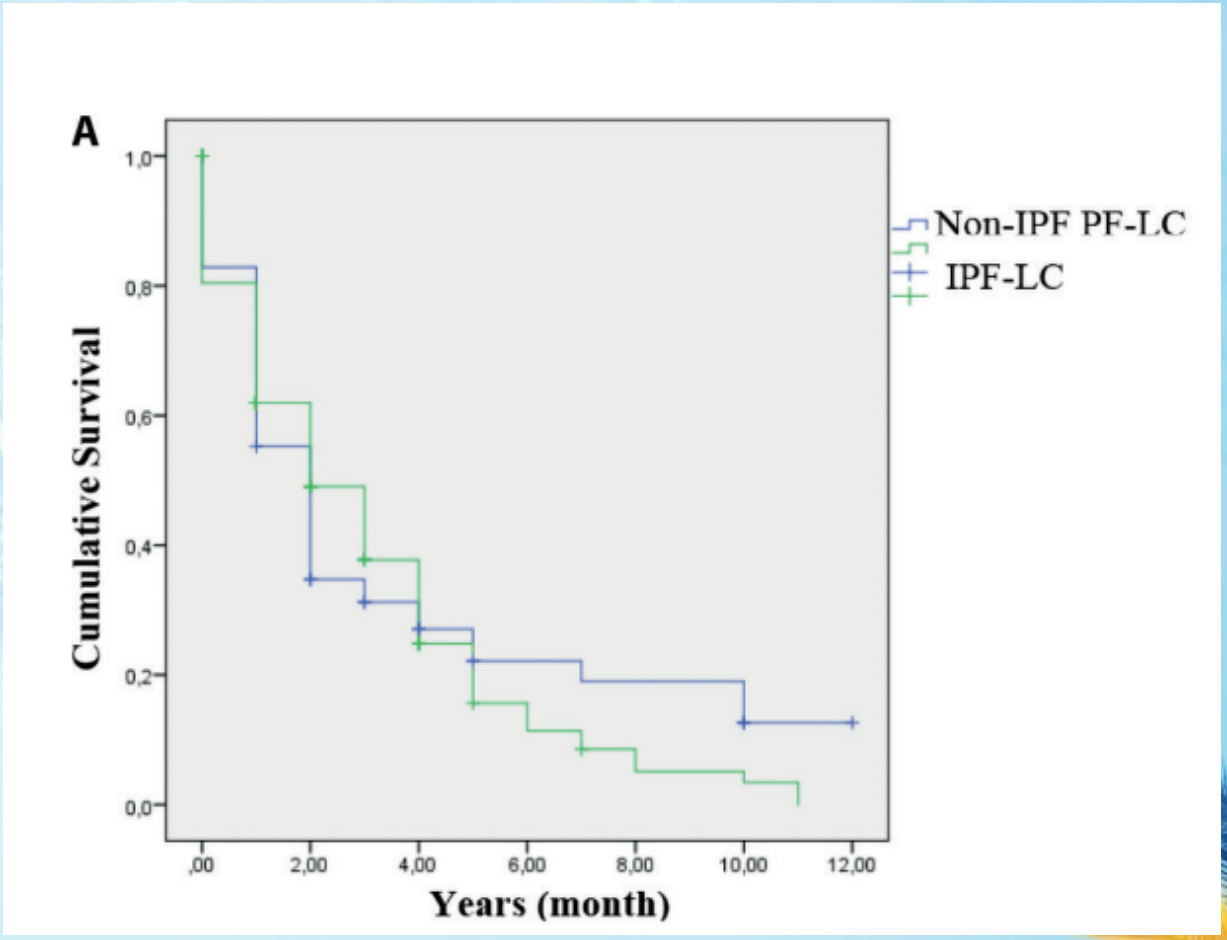
Table 3. Cancer characteristics of all cases.

	IPF-LC	Non-IPF PF-LC	
	n:164 (%)	n:87 (%)	p-value
Localization of LC			
Left lung	60 (36.6)	36 (41.4)	0.496
Right lung	99 (60.4)	50 (57.5)	0.687
Bilateral	3 (1.8)	1 (1.1)	
Upper Lobe	75 (46.0)	34 (39.5)	0.894
Middle / Lingula	16 (9.8)	15 (17.2)	0.107
Lower Lobe	80 (48.8)	41 (47.1)	0.349
Periphery	108 (65.9)	52 (59.8)	0.408
Central	37 (22.6)	32 (36.8)	0.118
Histology			
Squamous-cell carcinoma	70 (42.7)	33 (37.9)	0.807
Adenocarcinoma	47 (28.7)	28 (32.2)	
Small-cell carcinoma	31 (18.5)	19 (21.8)	
Others	16 (9.8)	7 (8.0)	
Staging			
I	27 (16.4)	15 (17.3)	0.644
II	14 (8.6)	11 (12.6)	
III	47 (28.6)	20 (22.9)	
IV	76 (46.4)	41 (47.2)	
Metastasis			
Bone	24 (15.3)	15 (17.6)	0.714
Contralateral lung	30 (19.1)	14 (16.3)	0.842
Liver	15 (9.6)	9 (10.7)	0.912
Adrenal gland	8 (5.1)	3 (3.6)	0.235
Brain	5 (3.2)	2 (2.4)	0.717
Others (Lymph node, pleura, chest wall)	17 (10.9)	15 (17.6)	0.140

Table 4. Diagnostic and treatment characteristics and mortality of all cases.

	IPF-LC n:164 (%)	Non-IPF PF-LC n:87 (%)	p-value
Modality of diagnosis			
FOB/EBUS	51 (31.3)	33 (37.9)	0.313
TTFNAB	54 (33.1)	27 (31.0)	
Mediastinoscopy	2 (1.2)	2 (2.3)	
Surgery	46 (28.2)	16 (18.4)	
Others	11 (6.7)	9 (10.3)	
Treatment of underlying diagnosis			
Steroid	21 (12.8)	13 (15.0)	-*
Nintedanib	42 (25.6)	4 (4.6)	
Pirfenidone	34 (20.8)	6 (6.9)	
Steroid + Nintedanib	-	2 (2.2)	
Steroid+ Pirfenidone	1 (0.6)	1 (1.2)	
Untreated	66 (40.2)	61 (70.1)	
Cancer treatment			
Untreated	29 (17.7)	17 (19.6)	0.528
Chemotherapy (CT)	52 (31.7)	29 (33.3)	
Radiotherapy (RT)	9 (5.5)	7 (8.0)	
CT/RT	37 (22.5)	12 (13.8)	
Surgery	22 (13.4)	16 (18.4)	
Palliative Treatment	15 (9.1)	6 (6.9)	
Death during follow-up			
Deceased	106 (64.6)	55 (63.2)	0.287
Living	37 (22.6)	15 (17.2)	
Unknown	21 (12.8)	17(19.5)	
Cause of mortality			
Respiratory failure + Lung cancer	43 (40.6)	26 (47.3)	
Primary respiratory failure	29 (27.4)	12 (21.8)	
Lung cancer	13 (12.3)	2 (3.6)	
Cardiac MI	4 (3.8)	1 (1.8)	
Pulmonary Thromboembolus	2 (1.9)	1 (1.8)	
Other	2 (1.9)	2 (3.6)	
Unknown	13 (12.3)	11 (20.0)	

Median survey IPF-LC grubunda 2±0.289 yıl, nonIPF-LC grubunda 2±0.364 yıl, **p=0.665**



Teşekkür ederim...