

CLINICAL SCIENCES

CORRELATION BETWEEN SURGICAL LUNG BIOPSY AND AUTOPSY FINDINGS AND CLINICAL DATA IN PATIENTS WITH DIFFUSE PULMONARY INFILTRATES AND ACUTE RESPIRATORY FAILURE

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INTRODUCTION: Surgical lung biopsy is an invasive procedure performed when other procedures have failed to provide an urgent and specific diagnosis, but there may be reluctance to perform it in critically ill patients with diffuse pulmonary infiltrates.

PURPOSE: To evaluate the diagnostic accuracy, the changes in therapy and survival of patients with diffuse lung infiltrates, mostly presenting acute respiratory failure, who underwent surgical biopsy.

METHODS: We retrospectively examined medical records and surgical lung biopsies from 1982 to 2003 of 63 patients older than 18 years with diffuse infiltrates. Clinical diagnoses were compared to histopathological ones, from biopsies and autopsies. Laboratory and epidemiological data were evaluated, and their relationship to hospital survival was analyzed.

RESULTS: All histological specimens exhibited abnormalities, mostly presenting benign/inflammatory etiologies. Fifteen patients had an etiologic factor determined in biopsy, most commonly *Mycobacterium tuberculosis*. The preoperative diagnosis was rectified in 37 patients. Autopsies were obtained in 25 patients and confirmed biopsy results in 72% of cases. Therapy was changed for 65% of patients based on biopsy results. Forty-nine percent of patients survived to be discharged from the hospital. Characteristics that differed significantly between survivors and nonsurvivors included sex ($P = 0.05$), presence of comorbidity ($P = 0.05$), SpO_2 ($P = 0.05$), and presence of diffuse alveolar damage in the biopsy ($P = 0.004$).

CONCLUSION: Surgical lung biopsy provided a specific, accurate etiologic diagnosis in many patients with diffuse pulmonary infiltrates when clinical improvement did not occur after standard treatment. Surgical lung biopsy may reveal a specific diagnosis that requires distinct treatment, and it would probably have an impact in lowering the mortality of these patients.

KEYWORDS: Surgical lung biopsy. Diffuse pulmonary infiltrate. Acute respiratory failure. Diffuse alveolar damage. Autopsy.

INTRODUCTION

Surgical lung biopsy (SLB) is an invasive procedure performed to assist in treatment efforts when other

noninvasive or minimally invasive procedures have failed to provide an urgent and specific diagnosis.^{1,2} Surgical lung biopsy is performed through a limited thoracotomy, without costal arc resection, allowing tissue samples to be obtained from 1 or more involved lobe(s)¹ for adequate histological, histochemical, and molecular analysis. However, there may be reluctance to perform SLB in critically ill patients with diffuse pulmonary infiltrates, who are frequently suffering from acute respiratory failure, often undergoing mechanical ventilation, and hemodynamically unstable. The progression of the disease and the need of a specific

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diagnosis are decisive factors. The purpose of this study was to evaluate diagnostic accuracy, changes in therapy, and survival of patients with diffuse lung infiltrates, mostly presenting acute respiratory failure, who underwent SLB.

METHOD

Patients. We examined the medical records of all patients who underwent SLB at our institutions from January 1982 to December 2003. From these we selected patients older than 18 years of age with diffuse infiltrates on chest roentgenogram and respiratory failure. Respiratory failure was defined as a PaO_2 of less than 60 mmHg (hypoxemic ARF), or a SpO_2 less than 90% of arterial blood, or PaCO_2 more than 50 mm Hg (hypercapnic acute respiratory failure) while breathing room air. Acute was defined as onset of symptoms of respiratory distress less than 10 days prior to SLB, in order to exclude those patients with a steady deterioration of a chronic process. Surgical lung biopsy was performed at the discretion of the attending physician under general anesthesia; progressive hypoxemia was the usual indication. Through a limited thoracotomy, tissue samples were removed with a cutting stapler. A specific protocol for specimen processing was followed. A portion of the surgical specimen was sent to surgical pathology, and permanent sections were examined after appropriate staining. The remaining specimen was sent to the microbiology laboratory where smears were prepared and part of the tissue was homogenized. Cultures were prepared for aerobic and anaerobic bacteria, fungi, mycobacteria, *Legionella*, *Nocardia*, *Chlamydia*, and viruses. Stains were performed with Gram stain for bacteria, Grocott for fungi and *Pneumocystis carinii*, and Ziehl-Neelsen for mycobacteria and *Nocardia*. Results of stains were usually available within 3 hours after specimen arrival in the laboratory.

Statistical Analysis. Comparisons between groups were made using chi-square analysis and unpaired *t* test when appropriate; $P < 0.05$ was taken as significant. Every analysis was performed with the SPSS for Windows 10.0 statistical software.

RESULTS

Patient Characteristics. Over the period examined, 303 surgical lung biopsies were performed, and 63 cases (20.8%) met our study criteria. Thirty-one patients (49%) were women and 32 (51%) were men, with a median age of 51 years (range, 18 to 92 years). Arterial blood samples for gas analysis taken during inspiration of room air in the 24 hours preceding SLB were obtained from 56 patients

(89%). Forty-four patients were in acute respiratory failure as indicated by the following: 38 of them had a $\text{PaO}_2 < 60$ mm Hg; 4 had a $\text{PaCO}_2 > 50$ mm Hg; and in 2 patients, both PaO_2 and PaCO_2 indices were altered. Eleven cases had only SpO_2 values (under 90%) available. Tables 1 and 2 list respectively the gasometric data and preexisting medical conditions in the study population. Thirty-three patients (52%) were immunocompromised, and 48 (66%) had preexisting medical conditions. The mean time from admission to SLB was 18 days (range, 0 to 61 days), the mean interval from SLB to hospital discharge or death was 21 days (range, 1 to 143 days) and the mean total hospitalization time was 39 days (range, 4 to 184 days).

Diagnostic Results. The diagnostic results of SLB are shown in Table 3. All biopsy specimens exhibited abnormalities. Forty-eight patients (76%) had benign/inflammatory etiologies; 14% had infectious causes, and 10% had infiltrates directly or indirectly due to neoplastic process. Among the main benign/inflammatory etiologies, 26 patients (41.3%) had diffuse alveolar damage (DAD)—as the only histopathological diagnosis in 7 patients, or associated with the main process in 19 patients. Twenty one of these were in the exudative stage and 5 in the proliferative stage. Acute lung injury was observed in 6 cases. At least 15 patients received steroid treatment, but in only 3 cases was corticotherapy started after the histopathological result (Table 4). Only 1 patient was in acute respiratory distress clinically. Seven patients (11%) had usual interstitial pneumonia (UIP), and 7 patients had chronic bronchiolitis, both in acute exacerbation and discordant radiology vs histopathology. Seven patients (11%) had a granulomatous process (2 sarcoidosis and 5 tuberculosis); 5 patients (8%) had pulmonary thromboembolism, and 4 patients (6.3%) had passive congestion. For 15 patients (23%) an etiologic factor was determined through the biopsy as follows: acute and chronic broncho-aspiration in 4 patients (6.3%) and infectious etiologic factor in 11 patients (17%). The most common cause found was tuberculosis, occurring in 5 patients (8%), followed by cytomegalovirus in 2 patients (3%), *Pneumocystis carinii* in 2 patients (3%), *Strongyloides sp.* in 1 patient (1%), and *Aspergillus sp.* in 1 patient (1%). Clinical, biopsy, and necropsy diagnoses were compared (Table 3). In this situation, the preoperative diagnosis of the cause of respiratory failure or diffuse pulmonary infiltrate was changed in 37 patients (59%). Autopsies were obtained in 25 patients who died in hospital and confirmed SLB results in 18 cases (72%). Therapy was changed in 31 patients (65%) based on SLB results. Change in therapy was defined as the as the addition or subtraction of 1 or more drugs or the early discontinuation of aggressive life support. Drug changes usually involved anti-

Table 1 - Gasometric data of the 63 patients studied, previous to surgical lung biopsy

Case	pH	PaO ₂	PaCO ₂	SpO ₂	Fi O ₂	Case	pH	PaO ₂	PaCO ₂	SpO ₂	Fi O ₂
1	7,36	100	30	97	60	33	7,48	57	36	92	NA
2	7,08	73	70	90	95	34	7,43	44,5	44,8	82,4	NA
3	7,45	33	35	59,9	NA	35	NA	NA	NA	87	NA
4	7,31	56	31	85,6	60	36	NA	NA	NA	88	NA
5	7,39	66	44,9	92,9	NA	37	NA	NA	NA	NA	NA
6	NA	NA	NA	80	NA	38	7,42	30,9	28,4	59,6	NA
7	7,38	51,8	42	85	NA	39	7,28	48,1	29	76,2	NA
8	NA	NA	NA	88	NA	40	NA	NA	NA	96	60
9	7,36	120	36	96,3	60	41	7,42	50,6	34,7	86,2	50
10	7,43	NA	50,1	86,9	100	42	NA	NA	NA	NA	NA
11	7,46	51	28,6	86	NA	43	7,42	52	25	88,3	60
12	7,22	57,9	62,4	88,2	45	44	7,43	51,1	36,8	84	NA
13	7,39	58,2	40,3	89,6	NA	45	7,46	59,5	37	92	NA
14	7,43	125,9	19,5	98,8	40	46	7,33	47	46,5	79,5	NA
15	7,44	45,3	27,5	82,5	NA	47	7,03	49	87	63,1	40
16	7,5	46,3	29	86,7	100	48	NA	NA	NA	90	NA
17	7,35	29	40	49,1	NA	49	7,13	65	63	85,8	100
18	NA	NA	NA	NA	NA	50	NA	NA	NA	96	NA
19	NA	NA	NA	87	NA	51	NA	NA	NA	NA	NA
20	7,51	48,3	26,3	88,4	60	52	7,37	42,8	23,5	75,7	NA
21	7,56	67	25	95,6	NA	53	7,47	59,8	29,6	92,4	NA
22	NA	NA	NA	83	NA	54	7,36	40	46	72	NA
23	7,37	54	40,7	86,6	NA	55	NA	NA	NA	NA	NA
24	NA	NA	NA	68	100	56	7,44	38,9	33,8	75,8	50
25	7,42	80	27	96,2	NA	57	NA	NA	NA	80	NA
26	7,48	43	29	80	60	58	NA	NA	NA	NA	NA
27	NA	NA	NA	NA	NA	59	7,49	87,1	23,9	97,3	NA
28	7,4	27,2	37,2	49,9	80	60	7,49	45,2	37,2	85	NA
29	7,43	71,7	32,1	94,6	NA	61	7,1	79	54	88	40
30	7,41	36,5	42,5	70,1	NA	62	7,23	105,9	20,4	96,3	40
31	NA	41,5	33	77,5	NA	63	7,43	52,2	38	87,8	NA
32	7,44	35,5	38	70,8	40						

NA: data non-available

Note: Data concerning PaO₂ and PaCO₂ expressed in mmHg; data concerning SpO₂ and FiO₂ expressed in percentage.

biotics and/or corticosteroids, but also included heparinization and the initiation or discontinuation of antineoplastic agents for malignancy or drug-induced toxicity, respectively. Surgical complication data was obtained in 32 patients; surgical complications were present in 5 patients (1 infection of the surgical wound, 1 pneumothorax, and 3 subcutaneous emphysema, Table 4).

Survival. Thirty-one patients (49%) survived to be discharged from the hospital.. For the 34 patients who died in hospital, survival time after SLB was 20 ± 29 days. Although not statistically significant, alterations in therapy resulting from SLB modified the survival rate in 7 patients (22%) out of 32 patients who had the information available ($X^2 = 0.14$, $P = 0.4$). A marginal significance was found between mortality and the presence of surgical complications ($X^2 = 3.80$, $P = 0.05$). In addition, neither the availability of a specific diagnosis nor changes in diagnosis, as well as infectious agent or immunocompromised status affected survival rates ($X^2 = 0.44$, $P = 0.24$; $X^2 = 0.51$, $P = 0.27$; and $X^2 = 0.13$, $P = 0.08$, respectively).

Preoperative characteristics that did not differ between

survivors and nonsurvivors included age ($X^2 = 2.42$; $P = 0.9$), PaO₂, ($X^2 = 5.07$; $P = 0.1$) PCO₂, ($X^2 = 4.02$; $P = 0.1$) and breathing frequency at the time of SLB ($X^2 = 1.37$; $P = 0.6$). There was no difference between the preoperative primary diagnosis and the diagnosis found at SLB in survivors versus nonsurvivors. Characteristics that differed significantly between survivors and nonsurvivors included sex ($X^2 = 3.56$; $P = 0.05$), comorbidity ($X^2 = 3.56$; $P = 0.05$), SpO₂ ($X^2 = 3.98$; $P = 0.05$), and presence of DAD in the SLB ($X^2 = 8.38$; $P = 0.004$). Eight out of 26 patients (31%) with DAD and only 3 out of 11 patients (27 %) with infectious etiology survived, in contrast to the case with acute exacerbation of UIP, in which 4 of 7 patients (57%) survived. A marginal significance was obtained for the clinical and gasometric pattern of acute respiratory failure ($X^2 = 3.1$; $P = 0.07$)

DISCUSSION

Although SLB did provide a specific diagnosis in many patients with diffuse pulmonary infiltrates and acute res-

Table 2 - Epidemiological data of the 63 patients studied

Case	Sex	Age	Total Hospitalization Time (days)	Underlying disease	Presence of immunosuppression factor	Outcome
1	m	56	20	No	No	Dead
2	m	48	32	No	No	Dead
3	m	76	60	Renal and heart failure	No	Survived
4	f	19	47	BMT (aplastic anemia)	Yes	Dead
5	m	67	44	No	No	Survived
6	m	55	17	Rheumatologic disease NOS	No	Dead
7	f	52	27	Heart failure	No	Survived
8	m	48	55	Crohn's disease+ renal failure	No	Dead
9	m	66	42	Rheumatoid arthritis	No	Dead
10	f	37	44	SLE	No	Dead
11	m	34	16	AIDS	Yes	Dead
12	m	45	18	CML	Yes	Dead
13	m	52	20	No	No	Survived
14	f	21	38	Non-Hodgkin's lymphoma	Yes	Survived
15	f	30	37	No	No	Survived
16	f	63	9	Carcinomatosis	Yes	Dead
17	m	37	48	No	No	Dead
18	f	51	22	Gastroesophageal reflux	No	Survived
19	m	52	38	Liver cirrhosis	No	Survived
20	f	46	26	Adenocarcinoma NOS	No	Dead
21	f	34	4	CML	Yes	Dead
22	f	49	19	Carcinomatosis	Yes	Survived
23	m	23	21	Burns	No	Dead
24	m	58	13	Adenocarcinoma NOS	Yes	Dead
25	m	77	45	COPD	Yes	Dead
26	m	36	20	BMT	Yes	Dead
27	m	78	17	No	Yes	Survived
28	f	54	9	SLE	No	Dead
29	f	51	184	Wegener's granumatosi	No	Dead
30	m	57	33	Ischemic myocardiopathy	No	Dead
31	f	21	42	SLE	No	Survived
32	f	74	16	Congestive myocardiopathy	No	Dead
33	m	55	91	Congestive myocardiopathy	No	Dead
34	m	65	69	Sarcoidosis	No	Dead
35	f	46	15	Wegener's granumatosi	No	Survived
36	f	52	76	No	Yes	Survived
37	f	92	24	Ischemic myocardiopathy	No	Survived
38	f	26	27	BMT (CML)	Yes	Dead
39	f	53	98	Rheumatoid arthritis	No	Dead
40	m	70	47	Non-Hodgkin's lymphoma	Yes	Dead
41	f	30	65	Colitis NOS	No	Survived
42	f	82	22	Neoplasia NOS	Yes	Survived
43	f	50	91	SLE	Yes	Survived
44	m	57	38	Gastroesophageal reflux+DM 2	No	Survived
45	m	65	37	Ischemic myocardiopathy+DM 2	No	Dead
46	m	47	37	ALL	Yes	Dead
47	m	32	16	ALL	Yes	Dead
48	f	68	37	Hypertension	No	Survived
49	m	53	26	Heart transplantation	Yes	Dead
50	m	22	89	ALL	Yes	Survived
51	m	18	7	BMT (ALL)	Yes	Survived
52	f	76	36	Ischemic myocardiopathy	No	Dead
53	f	55	17	Carcinomatosis	Yes	Dead
54	f	25	47	No	No	Survived
55	f	38	42	No	No	Dead
56	m	37	84	CML	Yes	Dead
57	f	36	10	Ischemic myocardiopathy	No	Survived
58	f	36	14	Carcinomatosis	Yes	Survived
59	m	73	34	No	No	Survived
60	f	52	36	No	No	Survived
61	m	31	49	SLE	No	Dead
62	m	28	48	Aplastic anemia	Yes	Dead
63	f	73	37	No	No	Survived

Abbreviations: AIDS: Acquired Immunodeficiency Syndrome; ALL: Acute Lymphocytic Leukemia; BMT: Bone Marrow Transplantation; CML: Chronic Myelocytic Leukemia; COPD: Chronic Obstructive Lung Disease; DM: Diabetes Mellitus; NOS: no other specification; SLE: Systemic Lupus Erythematosus

Table 3 - Distribution of clinical, biopsy and necropsy diagnoses

Diagnoses	Clinical		Biopsy		Necropsy	
Infectious lung disease	22	(35)	9	(14)	9	(38)
Granulomatous lung disease	7	(11)	7	(11)	1	(4)
Airway diseases	4	(6)	10	(16)	1	(4)
Interstitial pneumonia	11	(17)	10	(16)	1	(4)
Circulatory disturbance	6	(10)	7	(11)	2	(8)
Carcinoma	6	(10)	5	(8)	3	(13)
Hypersensitivity pneumonia			2	(3)		
Diffuse alveolar damage			7	(11)	6	(25)
Capillaritis/ vasculitis	5	(8)	4	(6)	1	(4)
Amyloidosis			1	(2)		
Histiocytosis X	1	(2)				
Lymphoma	1	(2)	1	(2)		
Total	63	(100)	63	(100)	24	(100)

piratory failure, the hospital mortality rate of 51% was high in this hypoxic patient population. Hospital mortality rates from similar but smaller series of hypoxic patients undergoing SLB are consistent with our experience, ranging from 50% to 70%.³⁻⁷ In contrast, reported mortality rates for all patients (hypoxic and nonhypoxic) undergoing SLB tend to be lower.^{4, 8-10} Greenman and coworkers¹¹ found a 73% mortality rate in hypoxic ($\text{PaO}_2 < 55$ mm Hg) immunosuppressed patients and a 39% mortality rate in nonhypoxic patients. In immunosuppressed patients, Jaffe and Maki¹² found a significantly higher PaO_2 in survivors of SLB, as compared to nonsurvivors. While our study was not designed to evaluate hypoxia as a predictor of mortality in patients undergoing SLB, probably our hypoxic patients ultimately did not fare well.

Given the high mortality rate, it would be helpful to be able to predict outcome. Poe and coworkers¹³ in a retrospective review attempted to predict mortality in immunocompromised patients with pulmonary infiltrates undergoing either transbronchial biopsy or SLB. They found that early mechanical ventilation (within the first 72 hours of admission, either preoperatively or for more than 24 hours postoperatively), the initial room-air PaO_2 , and corticosteroid therapy at the onset of symptoms were significant predictors of mortality. None of the 51 patients who simultaneously had a PaO_2 of < 50 mm Hg on room air, was receiving corticosteroids, and required early mechanical ventilation survived.

Most investigators feel that mortality in patients undergoing SLB is due to the severity of the underlying disease and not to complications of the surgical procedure.^{3,4,14-16} With the exception of a mortality percentage of 21.4% re-

ported by Ferson et al,¹⁷ more recent series refer indices lower than 8% directly related to the biopsy procedure.¹⁸⁻

²¹ While this assertion is difficult to prove, the complications in our series did not appear to directly cause death. However, the mortality rate in the patients suffering complications, while marginally significant, suggests that such complications requires more complex patient care. Studies evaluating the impact of SLB complications on outcome should include a nonoperative control group. In a prospective, randomized study comparing SLB and empiric antibiotic therapy for acute pneumonitis in 22 non-neutropenic cancer patients, Potter and coworkers²² did not find a significant difference in mortality rates between the 2 groups. The investigators did feel that 3 patients (21% of those undergoing SLB) experienced complications of SLB that contributed directly to their deaths. Although our patients did not suffer this high rate of catastrophic complications, any complication of a procedure of uncertain benefit is of concern.

Intrathoracic surgery, especially in the hypoxic patient, carries with it the risk of prolonged postoperative mechanical ventilation. Although it became possible to extubate half of our patients postoperatively, 6 patients (50%) were mechanically ventilated until death. It is not known how many of these patients would have eventually required ventilatory support had SLB not been performed, although only 1 of the patients presented a serious complication (air leak) related to the biopsy.

In the last decades, advances in technology such as computerized tomography, acquisition of knowledge in lung pathology produced by indications of surgical lung biopsy, the new ATS/ERS Classification of the Interstitial Lung Diseases may be an explanation for our 100% of specificity in diagnosis, in contrast to those found in previous series of SLB in immunosuppressed patients, ranging from 40% to 83%.^{11,12,23-27} Autopsy results in 18 (72%) patients verified the accuracy of SLB in all but 7 cases; the additional diagnosis (4 infectious pneumonias, 5 diffuse alveolar damages, and 1 bronchiolitis) may represent infections or complications resulting from them, which occurred between SLB and death. In many patients, the preoperative diagnosis was either erroneous or incomplete. The impact of SLB on changes in diagnoses, the presence of a specific diagnosis, and subsequent alterations in therapy on survival were very encouraging, and mirrored the experience of many others investigators.^{7,14,16,28,29} Delays in diagnosis can account for the high mortality rate reported in the literature,^{25,28,30} because the study population included many patients undergoing SLB relatively late in the course of their acute respiratory failure. Mortality might have been even greater in the diagnostic group, had a diagnosis not been

Table 4 - Data concerning complications related to biopsy procedure, change in therapy after histopathological results and corticotherapy before and after histopathological results

Case	Complications	Diffuse alveolar damage	Change in therapy	Corticotherapy pre-biopsy	Corticotherapy post-biopsy
1	no	yes (exs)	yes	yes	yes
2	no	yes (exs)	yes	no	no
3	no	yes (pro)	yes	no	no
4	NA	no	NA	NA	NA
5	no	no	NA	yes	NA
6	no	yes (exs)	no#	no	no
7	no	no	yes	no	yes
8	no	yes (exs)	yes	no	no
9	yes*	no	yes	yes	yes
10	no	yes (pro)	yes	yes	yes
11	no	yes (exs)	yes	yes	yes
12	no	yes (exs)	yes	no	no
13	NA	no	NA	NA	NA
14	yes**	no	yes	yes	yes
15	no	no	yes	no	no
16	NA	no	NA	NA	NA
17	no	no	yes	no	no
18	NA	yes (exs)	NA	NA	NA
19	no	yes (exs)	yes	no	yes
20	NA	yes (exs)	NA	NA	NA
21	no	no	no#	no	NA
22	no	yes (pro)	yes	no	no
23	no	no	no	no	no
24	NA	yes (exs)	NA	NA	NA
25	no	no	yes	no	no
26	NA	yes (exs)	NA	NA	NA
27	NA	no	NA	NA	NA
28	no	yes (exs)	no#	yes	yes
29	no	no	yes	no	yes
30	no	no	No	no	no
31	NA	no	NA	NA	NA
32	no	yes (pro)	no#	yes	yes
33	no	no	No	no	no
34	no	no	yes	no	no
35	NA	no	NA	NA	NA
36	NA	no	NA	NA	NA
37	NA	no	NA	NA	NA
38	NA	yes (pro)	NA	NA	NA
39	no	no	yes	yes	no
40	yes***	yes (exs)	No	yes	yes
41	NA	no	NA	NA	NA
42	no	no	No	yes	yes
43	NA	no	NA	NA	NA
44	NA	no	NA	NA	NA
45	NA	yes (exs)	NA	NA	NA
46	NA	no	NA	NA	NA
47	NA	no	NA	NA	NA
48	yes***	yes (exs)	No	yes	yes
49	no	yes (exs)	Yes	yes	yes
50	yes***	no	No	no	no
51	NA	no	NA	NA	NA
52	NA	no	NA	NA	NA
53	NA	no	NA	NA	NA
54	no	yes (exs)	No	no	no
55	NA	yes (exs)	NA	NA	NA
56	NA	yes (exs)	NA	NA	NA
57	NA	no	NA	NA	NA
58	NA	no	NA	NA	NA
59	NA	yes (exs)	NA	NA	NA
60	NA	no	NA	NA	NA
61	NA	no	NA	NA	NA
62	NA	no	NA	NA	NA
63	NA	yes (exs)	NA	NA	NA

NA: data non-available; * Infection of surgical wound; ** Atelectasia and pneumothorax; *** Subcutaneous emphysema; # Death before histopathological conclusion; "exs": exsudative phase; "pro": proliferative phase

made, this being a speculation that can be evaluated using our data. Also, patients without a specific diagnosis may have had less severe disease and may have thus been preselected for lower mortality. Nevertheless, while SLB is undoubtedly of value in securing or confirming diagnoses, our results suggest that SLB may influence patient survival rates through the direction of treatment strategy.

If patient benefit is defined by survival, the 6 patients (50% of total) who both survived and in whom SLB re-

sults were judged to be useful in guiding therapy can definitely be described as having benefited from SLB.

In conclusion, SLB provided a specific, accurate etiologic diagnosis in many patients with diffuse pulmonary infiltrates and acute respiratory failure when clinical improvement did not occur after standard treatment. Surgical lung biopsy may produce a specific diagnosis that indicates a distinct treatment, lowering patient mortality.

RESUMO

Canzian M, Soeiro A de M, Taga MF de L, Barbas CSV, Capelozzi VL. Correlação entre achados de biópsia pulmonar cirúrgica e de autópsia e dados clínicos em pacientes com infiltrados pulmonares difusos e insuficiência respiratória aguda. CLINICS. 2006(5):425-32.

INTRODUÇÃO: A biópsia pulmonar cirúrgica é um procedimento invasivo executado quando outros procedimentos não forneceram um diagnóstico urgente e específico; no entanto, pode haver relutância em sua execução em pacientes críticos, que apresentam infiltrados pulmonares difusos.

OBJETIVO: Avaliar a acurácia diagnóstica, mudanças na terapêutica e a sobrevida de pacientes com infiltrado pulmonar difuso, em sua maior parte apresentando a insuficiência respiratória aguda, submetidos a biópsia cirúrgica.

MÉTODO: Foram examinados retrospectivamente registros médicos e biópsias pulmonares cirúrgicas de 63 pacientes maiores de 18 anos de idade, com infiltrados difusos, entre 1982 a 2003. Os diagnósticos clínicos foram comparados aos histopatológicos, de biópsias e de autópsias. Dados laboratoriais e epidemiológicos foram avaliados e sua correlação com a sobrevida hospitalar analisada.

RESULTADOS: Todos os espécimes histológicos exibiram alterações, em sua maior parte de natureza benigna/infla-

matória. Em quinze casos o fator etiológico pôde ser determinado na biópsia, sendo o *Mycobacterium tuberculosis* o mais freqüente. O diagnóstico pré-operatório foi mudado em 37 pacientes. Autópsias foram realizadas em 25 pacientes e confirmaram resultados da biópsia em 72%. A terapêutica foi alterada em 65% dos pacientes com base nos resultados da biópsia. Quarenta e nove por cento dos pacientes sobreviveram ao final do período de hospitalização. Características que diferiram significativamente entre sobreviventes versus não sobreviventes incluíram sexo ($p=0.05$), a presença de comorbidade ($p=0.05$), a SatO_2 ($p=0.05$), e a presença de dano alveolar difuso na biópsia ($p=0.004$).

CONCLUSÃO: A biópsia pulmonar cirúrgica forneceu um diagnóstico etiológico específico e exato em muitos pacientes com infiltrados pulmonares difusos quando a melhora clínica não ocorreu após o tratamento padrão. A biópsia pulmonar cirúrgica pode fornecer diagnósticos que requeiram tratamentos específicos, com provável impacto na redução do índice de mortalidade destes pacientes.

UNITERMOS: Biópsia pulmonar cirúrgica. Infiltrado pulmonar difuso. Insuficiência respiratória aguda. Dano alveolar difuso. Autópsia.

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