

Black Pleural Effusion

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ABSTRACT

BACKGROUND: Black pleural effusions are extremely rare and have been reported in patients with infection, malignancy, and hemorrhage. However, no review articles appear to have focused on this rare clinical presentation.

PURPOSE: To classify and characterize diseases causing “black pleural effusion” based on the pathophysiological mechanisms involved.

METHODS: We searched the medical literature to find reports of “black pleural effusion” using the PubMed database.

RESULTS: We identified 8 cases and classified the underlying diseases into the following 4 entities based on pathophysiological conditions: 1) infection (*Aspergillus niger* and *Rhizopus oryzae*); 2) malignant melanoma, in which cells contain melanin pigment; 3) hemorrhage and hemolysis associated with non-small cell lung cancer or rupture of a pancreatic pseudocyst; and 4) other causes (charcoal-containing empyema). Discrimination between biliopleural fistula and pancreatico-pleural fistula, which also mimicking in color, was easily achieved by focusing on pleural amylase levels, elevation of pleural indirect bilirubin, presence of pleural glycolic acid, and the predominant site of pleural effusion.

CONCLUSION: Black pleural effusions can be divided into 4 major categories based on the underlying pathophysiological conditions.

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KEYWORDS: Biliopleural fistula; Black pleural effusion; Pancreaticopleural fistula; Pathophysiological classification

Serous (yellow) and blood-tinged (reddish) pleural effusions are the most common types of pleural fluid at thoracentesis.¹ Black pleural effusions are uncommon, and there have been no review articles on these effusions. Therefore, the previous reports of black pleural effusions were reviewed, and their characteristic findings are discussed based on pathophysiological assessment to identify clinical clues that would help a general physician or pulmonologist establish the etiology when dealing with a patient with a black pleural effusion.

EPIDEMIOLOGY

A black pleural effusion is extremely rare, and only 8 cases have been reported to date (Table 1).²⁻⁹ Therefore, the precise incidence of black pleural effusion has not yet been clarified.

Causes and Pathophysiological Classification of Black Pleural Effusion

Based on the pathophysiological conditions, the 8 cases were divided into 4 groups, as described in Figure 1.

Infection: Associated with *Aspergillus niger* and *Rhizopus oryzae*. The black color of the sputum and pleural fluid is due to the black-pigmented spores of *A. niger* (Figure 2).² Macroscopically, *A. niger* colonies on potato dextrose agar consist of a white or yellow basal felt covered by a dense layer of dark brown to black conidia (Figure 2A). The microscopic morphology of *A. niger* shows the conidial heads, which are large, globose, and dark brown to black in color, and contain the fungal spores, facilitating propagation of the organism (Figure 2B). Indeed, Kimmerling et al⁴ reported that black, friable, and gritty material was con-

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firmed by transbronchial lung biopsy in a patient with invasive aspergillosis, which showed necrotic tissue, calcium oxalate crystals, and *Aspergillus* hyphae. According to Raper,¹⁰ in 1891, Wehmer showed oxalic acid to be a fermentation product of *A. niger*. Oxalic acid was incriminated in the blood vessel destruction by identifying oxalate crystals by polarization (**Figure 2C, D**), and these crystals cause localized tissue damage or severe hemorrhage.¹¹ On partly crossed Polaroids, calcium oxalate is birefringent adjacent to black deposits (**Figure 2C**, arrow) of *A. niger* (**Figure 2C**, asterisk), whereas, on fully crossed Polaroids, calcium oxalate is more clearly identified (**Figure 2D**). The appearance of black sputum or acidic pH in pleural fluid (as low as 5.9 in previous reports), or sputum or cavitory washings containing *A. niger* should suggest the presence of *A. niger* infection or invasion.

Regarding *Rhizopus oryzae*, only one report has been published.⁵ This mold has a characteristic colonial appearance (whitish-to-yellowish cotton candy-like colonies grown on Sabouraud dextrose agar at 25°C for 3 days), and microscopic examination shows broad hyphae without septa and the presence of rhizoids where the stolons and long sporangiophores meet. The mechanism of generating a black pleural effusion associated with *R. oryzae* infection was thought to be due to necrotic debris caused by the *R. oryzae* infection⁵ or to liquefaction of old blood from a previous thoracentesis in the presence of underlying coagulopathy.

Malignant Cells Containing Melanin Pigment Associated with Melanoma. Libshitz and North¹² reported that metastatic malignant melanoma constituted about 5% of all secondary malignancies of the lung, and Chen et al¹³ reported that only 2% of patients with malignant melanoma metastatic to the thorax had pleural effusions. Malignant melanoma with thoracic metastases can cause a black pleural effusion. Mohan and Gowrinath⁷ reported that, on cytological examination of pleural effusion, cytoplasmic pigment was stained black with Fontana Masson's stain, highlighting the presence of cytoplasmic melanin within the tumor cells. However, the degree of melanin pigment in their cytoplasm varied in individual cases, which might explain why melanin pigment was seen in only 25% of cytology specimens of fluids (pleural, ascitic, cerebrospinal, and other fluids) in another report.¹⁴

Hemorrhage and Hemolysis Associated with Non-small-cell Lung Cancer or Rupture of a Pancreatic Pseudocyst.

Rojas-Solano et al⁶ reported a black pleural effusion occurring in a patient with non-small-cell lung cancer, subtype adenocarcinoma, stage IIIB (T4, N1, M0). This case suggests that the black color of the pleural fluid was the result of hemolysis, which was confirmed by the presence of large quantities of hemosiderin-laden macrophages on Prussian blue staining following massive bleeding into the pleura.

Rupture of a pancreatic pseudocyst is one of the causes of a black pleural effusion.⁹ The development of a pseudocyst was preceded by pancreatitis (90%) or pancreatic trauma (10%). Sankaran and Walt¹⁵ reported that alcoholism was the most common cause of pancreatitis, and 3% of patients with a pancreatic pseudocyst developed a pancreaticopleural fistula, but the precise incidence of pancreaticopleural fistula is difficult to ascertain because of its rarity.¹⁶ A pancreatic pleural effusion develops due to a pancreaticopleural fistula expanding

mainly in 2 directions. Anterior disruption of a pancreatic pseudocyst into the peritoneal cavity results in pancreatic ascites,¹⁷ and posterior disruption causes a retroperitoneal fistula track into the pelvis (**Figure 3**, route 1) or superiorly through a natural hiatus in the diaphragm into the mediastinum (esophageal or aortic hiatus) (**Figure 3**, routes 2 and 3). The predominant left-sided pleural effusion would be noted as route 2 when a retroperitoneal fistula spreads via a natural hiatus owing to the anatomical proximity. Another mechanism is direct penetration through the dome of the diaphragm, ignoring normal fascial planes (**Figure 3**, route 4).^{18,19}

Uchiyama et al²⁰ reported that only 20% of patients complained of abdominal pain, and most complained of dyspnea and chest pain; because the pancreatic secretions drain into the pleural cavity, abdominal pain and peritonitis may not develop. We reported a massive left pleural effusion in a patient with a pancreaticopleural fistula with a pancreatic pseudocyst caused by chronic pancreatitis⁹ (**Figure 4**). The pleural fluid was black in color, and the fluid analysis was consistent with an exudative pleural effusion (lactate dehydrogenase 784 IU/L, total protein 4.4 g/dL, glucose 115 mg/dL). The mechanism of the black fluid color was due to hemolysis of the blood that entered the chest from thoracic bleeding. This etiology was confirmed by further fluid analysis, including marked elevations of amylase (5292 IU/L), total bilirubin (7.3 mg/dL; indirect bilirubin 6.5 mg/dL), and a pleural fluid-to-serum bilirubin ratio of 24:3, along with the presence of iron (223 mg/dL). Cytology

CLINICAL SIGNIFICANCE

- We divided black pleural effusions into 4 classifications based on their pathophysiology.
- Infection (*Aspergillus niger* and *Rhizopus oryzae*), malignant melanoma, hemorrhage and hemolysis, and other cause (charcoal-containing empyema) were the main etiologies for black pleural effusion.
- Discrimination of greenish dark or straw-colored bilious pleural effusion from a black or blood-tinged pleural effusion associated with pancreaticopleural fistula is a critical issue for general physicians.

Table 1 Review of the Literature Reported as Black Pleural Effusion

Ref.	Author	Year	Age (Y)	Sex	Etiology	Findings on Black Pleural Effusion
2	Metzger et al	1984	63	M	Bronchopleural fistula and empyema caused by <i>Aspergillus niger</i> and oxalic acid	pH <5.9; glucose 70 mg/dL; Leukocytes counts were not counted (segmented neutrophils 75%, 10% lymphocytes, and 15% mononuclear cells); <i>Aspergillus niger</i> was cultured
3	Justiniani et al	1985	25	M	Charcoal treatment for alcohol and drug overdose complicated with esophageal rupture	Charcoal particles with MSSA clusters or PMN on Gram stain, suggesting empyema
4	Kimmerling et al	1992	64	M	Invasive aspergillosis caused by <i>Aspergillus niger</i>	pH <5.9, Lactophenol cotton blue mount showed septate hyphae, demonstrating <i>Aspergillus niger</i>
5	Lai et al	2006	46	M	<i>Rhizopus oryzae</i> empyema thoracis in an allogenic BMT patient	Positive culture for <i>Rhizopus oryzae</i> , WBC, 2500/mm ³ with a lymphocyte predominance (93%); LDH. 11,230 U/L; TP, 3.4 g/dL; Glu, 86 mg/dL
6	Rojas-Solano et al	2009	89	M	Massive bleeding into the pleura by lung cancer (adenocarcinoma), stage IIIB (T4, N1, M0)	RBC, 5000/mL; WBC, 34,000/mL with a monocyte predominance (80%); Glu, 5 mg/dL; pH, 7.0; pigment-laden-Mφ on Prussian blue stain, indicating the presence of hemosiderin
7	Mohan and Gowrinath	2010	35	M	Thoracic metastasis of cutaneous malignant melanoma	Cytoplasmic melanin pigment stained with black within the tumor cells on Papanicolaou or Fontana Masson's stain
8	Liao et al	2010	71	F	Thoracic metastasis of malignant melanoma	Cytoplasmic melanin pigment deposits within the tumor cells on Diff-Quick stain
9	Koide et al	2012	54	M	Pancreaticopleural fistula with a pancreatic pseudocyst caused by chronic pancreatitis, hemolysis of thoracic bleeding	LDH, 784 IU/L; TP, 4.4 g/dL; Glu, 115 mg/dL; hematocrit, 0.1%; amylase 5,292 IU/L; T-bil, 7.3 mg/dL (ID-bil, 6.5 mg/dL); iron, 223 mg/dL; no detectable glycocholic acid

BMT = bone marrow transplantation; Glu = glucose; ID-bil = indirect bilirubin; LDH = lactate dehydrogenase; Mφ = macrophage; MSSA = methicillin-sensitive *Staphylococcus aureus*; PMN = polymorphonuclear leukocyte; RBC = red blood cell; T-bil = total bilirubin; TP = total protein; WBC = white blood cell count.

of the centrifugation sediment of the pleural fluid demonstrated red blood cells in the background and a small number of neutrophils. The fluid contained a large amount of amylase, which resulted in hemolysis of the blood in the thoracic cavity. Of note, Kaye¹⁸ reported that a blood-

stained pleural effusion was seen in 24 of the 73 cases with pancreatic pseudocysts, and no correlation was noted between the presence of blood and the amylase level. In our case, no detectable glycocholic acid was found in the fluid analysis, which is the main bile com-

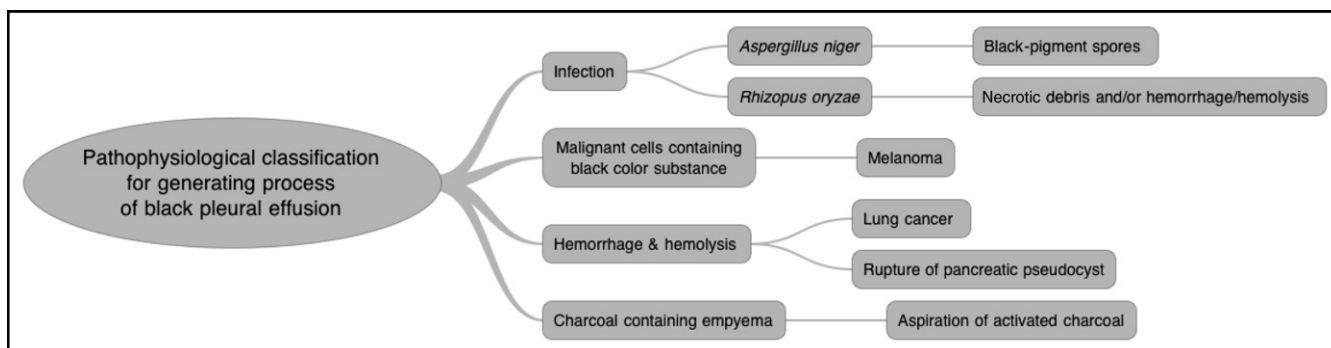


Figure 1 Schema of the suggested classification based on the pathophysiological mechanism.

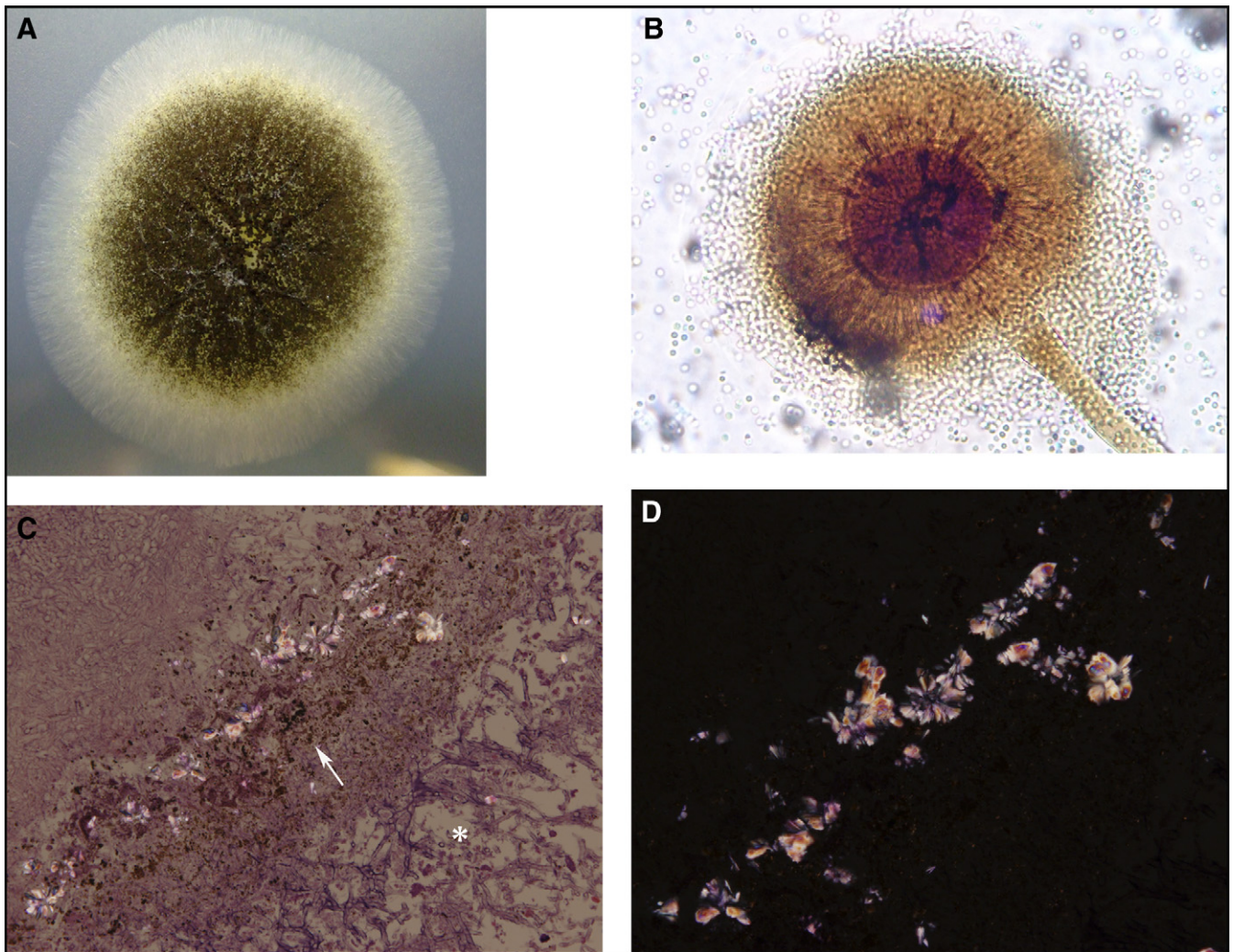


Figure 2 Macro and microscopic morphology of *Aspergillus niger*. Macroscopically, *A. niger* on potato dextrose agar colonies appears white or yellow. Basal felt covered by black-colored spore heads that spread out on almost the entire colony with radial furrows is present (A). The microscopic morphology of *A. niger* shows very rough, conidial heads that are large, globose, and dark brown to black and contain the fungal spores (B). On partly crossed Polaroids, calcium oxalate is birefringent adjacent to black deposits of (C, arrow) *A. niger* (C, asterisk), whereas on fully crossed Polaroids, calcium oxalate is more clearly identified (D).

ponent in bile acid, suggesting that a bilious pleural effusion was unlikely.⁹

Other Causes: Charcoal-containing Empyema. Justiniani et al³ reported a case of alcohol and drug overdose with a charcoal-containing empyema after treatment of a tension pneumothorax. It was assumed that aspiration of gastric contents plus activated charcoal-associated empyema was caused by esophageal perforation via vomiting or gastroesophageal reflux. This case clearly showed the presence of black, purulent pleural fluid with charcoal particles in the pleural fluid.

Differential Diagnosis, with Special Reference to Biliopleural Fistula and Pancreaticopleural Fistula

There are a small number of effusions, transudates, and exudates that develop from an extravascular origin.²¹ Many

such pleural effusions are secondary to diseases of the gastrointestinal tract including pancreatic disease, intra-abdominal abscesses, esophageal perforation, abdominal operations, diaphragmatic hernia, variceal sclerotherapy, hepatic transplantation, and diseases of the biliary tract. Of these diseases, black pleural effusions were only seen in pancreaticopleural effusion,⁹ mimicking in color that of bilious pleural effusion, which presents as a greenish dark or straw color together with a pleural fluid-to-serum total bilirubin ratio >1.0.²¹⁻²⁴ Discriminating between these 2 diseases is important.

Bilious pleural effusion is a rare complication of biliary tract disorders. Causes of a biliopleural fistula include thoracoabdominal trauma,^{22,23} parasitic liver disease,^{24,25} suppurative complications of biliary tract obstruction, postoperative strictures of bile ducts,²⁶ liver biopsy,²⁷ internal stent,²⁸ and percutaneous biliary drain-

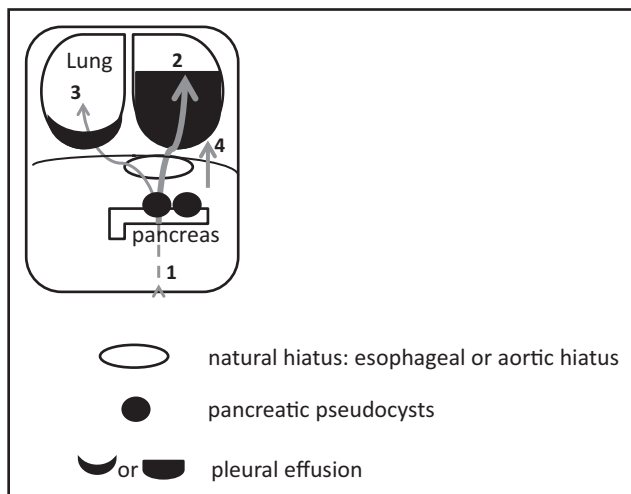


Figure 3 The pathogenesis of pancreaticopleural fistulas and the direction of pancreatic pleural effusions via retroperitoneal spread. Retroperitoneal spread of a pancreatic pleural effusion could take 4 migratory pathways. **Route 1** is a rupture of the pancreatic pseudocyst posteriorly, whereas **routes 2 and 3** are pathways going superiorly through a natural hiatus in the diaphragm into the mediastinum. The predominant left-sided pleural effusion is noted as **route 2**, expanding superiorly because of the anatomical proximity. Another mechanism is direct penetration through the dome of the diaphragm, ignoring normal fascial planes (**route 4**).

age for an obstructed biliary system.^{29,30} Nichols et al³¹ reported that pleural and diaphragmatic penetration is usually unavoidable during intercostal puncture because of the great depth of the pleural sinus beneath the lung margin. Indeed, Pisani and Zeller²⁷ reported that the risk of fistula increases with:

- inadvertent removal of drainage tubes;
- prolonged drainage;

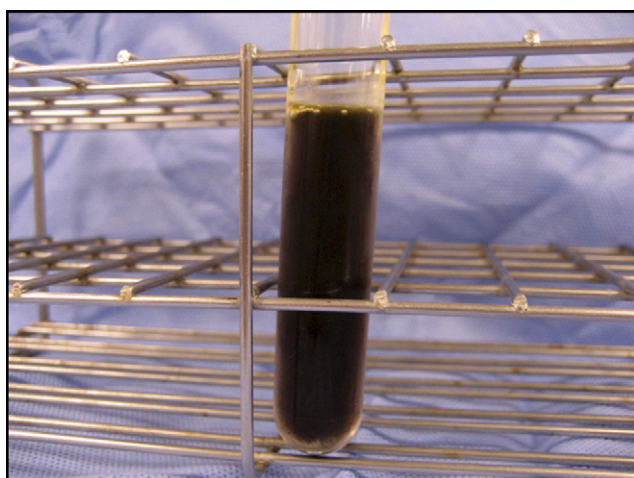


Figure 4 Black pleural effusion associated with a case of pancreaticopleural fistula.

Table 2 Comparison of the Characteristic Findings between Bilious Pleural Effusion and Rupture of the Pancreatic Pseudocyst

	Bilious Pleural Effusion	Rupture of the Pancreatic Pseudocyst
Color	Greenish dark or straw	Black or blood tinged
Pleural protein (g/dL)	+	+
Pleural LDH (U/L)	+	+
Pleural amylase (U/L)	-/+	+++ (>1000)
Pleural T-bil (mg/dL)	+++	+++
Pleural ID-bil (mg/dL)	+	++/+++
Pleural T-bil/serum T-bil	>1.0	>1.0
Pleural glycocholic acid	+	-
Predominant site	Right	Left

ID-bil = indirect bilirubin; LDH = lactate dehydrogenase; T-bil = total bilirubin.

- tubes placed intercostally between the 9th and 10th ribs in the midaxillary line;
- persistent biliary tract obstruction; and
- biliary tube dysfunction.

The possibility of a bilious pleural effusion should be considered, especially in patients having a right pleural effusion following such procedures.

The characteristic findings of bilious pleural effusion and rupture of a pancreatic pseudocyst are compared in **Table 2**. At a glance, one might confuse a greenish dark-colored bilious pleural effusion with a black or blood-tinged one. However, no or mild elevation of amylase in a pleural effusion suggests a bilious pleural effusion, whereas a pleural effusion associated with rupture of a pancreatic pseudocyst shows marked elevation of pleural amylase (>1000 IU/L). Thus, the pleural fluid amylase level is the most crucial element in the diagnosis of a pancreaticopleural fistula. Importantly, pleural effusions associated with acute pancreatitis are small and self-limited and have pleural fluid amylase levels of no greater than 4000 IU/L.¹⁶ Although the ratio of pleural fluid to serum bilirubin is >1.0 in both diseases, pleural effusion associated with rupture of a pancreatic pseudocyst shows a significantly higher indirect bilirubin level than a bilious pleural effusion that is derived from the presence of an amylase-rich pleural effusion with thoracic bleeding and hemolysis. Although iron in the pleural effusion was detected in widely scattered results in both benign (congestive heart failure, tuberculosis, pulmonary embolism, collagen disease, pneumonitis, nonspecific pleuritis, hypoalbuminemia, and constrictive pericarditis) and malignant diseases (lymphoma, breast carcinoma, and lung carcinoma), the median values were equally low in both groups.³² Therefore, the existence of iron in a pleural effusion also might be a clinical clue to the diagnosis of rupture of a pancreatic pseudocyst with hemorrhage or hemolysis.⁹ Alternatively, the presence of glycocholic acid in a pleural

effusion would suggest a bilious pleural effusion⁹ (Table 2). Glycocholic acid is a main component of bile acids, normally synthesized by the liver and secreted by the gall bladder, and the presence in a pleural effusion suggests a fistula from the biliary tree to the pleural space. The fact that the predominant site of bilious pleural effusion is the right side, whereas rupture of a pancreatic pseudocyst is more common on the left side, also is a clue in the differential diagnosis.

TREATMENT AND PROGNOSIS

Regarding the management of pancreatic pseudocysts, currently there is no clear consensus on the absolute indications for intervention.³³ In cases with progression, such as pleural effusion associated with rupture of a pancreatic pseudocyst, if conservative therapy for 2 weeks is not effective, surgical correction, including ductal decompression and drainage or resection of the pseudocyst, is indicated.²¹ The treatment and prognosis of black pleural effusions differ in individual cases. Of note, treatment of a bilious pleural effusion is aimed at securing adequate biliary decompression.²¹

CONCLUSION

The previous 8 reports on black pleural effusion were reviewed, summarized, and categorized into 4 clinical entities based on the pathophysiological findings. How to discriminate between pleural effusions associated with biliopleural fistula and those caused by pancreaticopleural fistula also was discussed.

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