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TBeat

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In This Issue...

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New DSHS Tuberculosis Hospital Opens in San Antonio

The nation's largest new construction project for the inpatient care and treatment of tuberculosis patients has been completed at the Texas Center for Infectious Disease, a DSHS facility in San Antonio. A ceremonial opening for the center's new 75-bed hospital building was held on Sept. 22; patients will be transferred from older quarters into the new 65,000-square-foot facility in mid-January.

Officials cut the ribbon, opening the new TCID hospital. Jim Elkins, director of TCID, is at center left in blue suit and red tie. On the left side of him are DSHS Commissioner David Lakey, with scissors, and Dr. Kirk Calhoun, president UTHSCT, while on the right is Alfaretta Reininger, in red; at age 88 and after 28 years of service, she's the eldest member of TCID's Volunteer Services Council

"TCID is one of only six tuberculosis centers in the United States," says Jim Elkins, TCID director. "With 75 private rooms, our new hospital building has the country's largest concentration of air-isolation patient rooms."

An air-isolation room is a safety feature. Because TB bacteria can be airborne, the air drawn into the patients' rooms is replaced with fresh air every five minutes and is never expelled into the hallways or other common areas. This feature prevents the spread of contaminants and pathogens from patients who are still contagious before their treatment has ended.

An inpatient stay at a TB hospital lasts much longer than in general hospitals; most TB patients stay for six months, but the length of stay can range from three months to two years. At TCID, patients stay until their TB is cured or until they're stabilized enough to be returned to their communities to continue outpatient TB treatment at home.

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Orange tiles on the hallway floor indicate where patient rooms have their own entry foyer, or anteroom," where the air is safe from TB bacteria.

The modern air-quality systems in the patients' new rooms will make a big difference in their daily lives at the hospital. "Patients had to wait for one of only 19 air-isolation rooms to become available, and, to more appropriately group them, we had to shuffle patients among rooms or units in different buildings," says Elkins. "But now, the systems in each room can be individually tailored to the conditions for which each patient is being treated. Each unit can be tailored, too, so groupings can be made based on similar patient needs. Female patients are already lobbying for their own unit, something the staff was considering anyway, and an early idea to try."

The old patient-care building at TCID was built 57 years ago. The new one features energy-efficient air-conditioning, heating, dayrooms with magazines, games, educational material, and large TVs. All rooms have electronic locks and large windows, and they're designed to be accessible for people with disabilities. Contagious patients, who normally must remain alone in their rooms, are able to enjoy socializing with each other outdoors on a walking trail and on the new patios; the TB germ cannot survive for long in open air.

"Patients are excited about the improvements," says Elkins. "They're mostly looking forward to having their own room, one that can be locked for the first time in this hospital's history. They love the new privacy of having their own toilet and shower.

They like the colors and cheeriness of the rooms, the attention to air quality and safety for their visitors and themselves, and more spaces for their activities and interests."

TCID treats about 120 patients each year. Their care is managed through the collaborative efforts of attending physicians, registered and vocational nurses, and certified nurse aides. They're joined by social workers, chaplains, a dietitian, a psychologist, a substance-abuse counselor, radiology and laboratory diagnosticians, respiratory therapists, a nurse case manager, and support staff.

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The VISION of Heartland is to provide *excellence*, *expertise*, *innovation* in training, medical consultation, and product development to reduce the impact of tuberculosis in our region.

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The new two-story TCID hospital building has six wings; each can be separately secured. A specialty hospital, TCID has no emergency services, operating rooms, or intensive-care units.

TCID treats about 120 patients each year. Their care is managed through the collaborative efforts of attending physicians, registered and vocational nurses, and certified nurse aides. They're joined by social workers, chaplains, a dietitian, a psychologist, a substance-abuse counselor, radiology and laboratory diagnosticians, respiratory therapists, a nurse case manager, and support staff.

For employees, the new facility features strategically placed nursing stations, locker rooms with showers and a changing area, quiet charting rooms, locked medication rooms, and break rooms.

"The new TCID campus is planned to bolster peace of mind for staff and patients," says Elkins. "As staff get training on the use of each new feature – electronic health records, complex safety systems, an automated pharmacy, modern food-handling equipment – their nervous energy is really building up. They want to use the new tools in the shiny new spaces."

Renovations were also made to another campus building where TCID diagnostic and treatment services are offered. Offices for physicians and other professionals are located here, as well as referral TB clinics managed by each of the five physicians with hospital privileges. This separate building also houses the Hansen's Disease Clinic, one of only four in the state.

Other related improvements include new imaging equipment, electronic picture archiving, and information systems for Radiology. The patient's gym, additional physicians' offices, Cardiopulmonary Services, and the Women's Health Laboratory all have renovated spaces and new equipment.

"The lab's new Bio-Safety Level III section can be used to test for tuberculosis and a number of other infectious diseases in a much safer environment now," says Elkins.

TCID is located on a mixed-use campus that's shared by a dozen other government entities, including a psychiatric hospital. More than 300 DSHS employees work at the site; about 165 of them at TCID. Physicians across the campus consult with each other on internal medicine, infectious disease, and acute psychiatric conditions.

"Our buildings house the UT Health Science Center at Tyler staff, including our attending physicians and the Heartland National TB Center's medical director and her staff," says Elkins. "These workers, plus our own TCID medical director, are all UTHSC-Tyler employees."

Jim Elkins, left, thanks Alysia Thomas-Gibbons for her testimonial at the hospital's opening ceremony. Formerly homeless, drug-addicted, and weighing just 89 pounds, Thomas-Gibbons was admitted to TCID as a TB patient three years ago and was released – cured and rehabilitated – after nine months of treatment. She now works on the TCID campus as an administrative specialist for the Heartland National TB Center.

At the ribbon-cutting ceremonies for the new hospital building on Sept. 22, Elkins and his TCID colleagues were joined by construction managers and architects; the medical directors of TCID, UTHSC-Tyler, and the Heartland National TB Center; a county commissioner and a state senator's representative.

DSHS participants from Austin included our commissioner, Dr. David Lakey; Dee Porter, chief operating officer; Mike Maples, assistant commissioner for Mental Health and Substance Abuse Services; Peggy Perry, director of the State Hospital Section; and David McCormick, manager of the Hospital Construction Unit.

"I look at TCID as a sanctuary," says Elkins. "People with TB have a slow-growing, slow-dying disease. Our services are needed to support TB treatment and indigent care, and I'm glad that TCID is relevant."
POSTED OCT. 9, 2010—DSHS STAFF NEWS

The MISSION of the Heartland National TB Center is to build capacity with our partners. We will share expertise in the treatment and prevention of tuberculosis by: developing and implementing cutting-edge trainings, delivering expert medical consultation, providing technical assistance, and designing innovative educational and consultative products.

TUBERCULOSIS PLEURAL EFFUSIONS AND A CASE OF EMPYEMA NECESSITATIS (NECESSITANS)

David Griffith, MD

CASE HISTORY

A 15 year old pregnant female presented to the emergency department of a local hospital with respiratory distress at 32 weeks gestation. She had failed to gain weight appropriately during her pregnancy and throughout her third trimester of pregnancy had cough, shortness of breath and night sweats. She had an abnormal chest radiograph suggesting miliary tuberculosis and bilateral pleural effusions left side greater than the right. The patient was sent by air ambulance to a tertiary referral center where she was treated with broad spectrum antibiotic therapy in addition to multi-drug antituberculosis therapy including coverage for possible drug resistant TB with isoniazid, rifampin pyrazinamide, ethambutol, moxifloxacin and amikacin. Due to respiratory distress, labor was induced and a healthy baby was delivered vaginally. Initial evaluation of the child showed no evidence of tuberculosis.

The patient was noted on admission to have an anterior chest wall abscess. Thick, purulent fluid was obtained which was very difficult to aspirate. The abscess fluid was AFB smear positive and subsequently grew M TB sensitive to all first line antituberculous medications. Ultrasound and CT examination following delivery suggested that the pleural fluid was too thick to be aspirated or evacuated. Following delivery, the patient was able to produce sputum that was smear and subsequently culture positive for M TB. The patient responded well to the antituberculosis therapy in general, including, resolution of fever, decrease in cough, normalization of albumin and improvement in the bilateral pulmonary densities and effusions. The chest mass decreased in size following the initial aspiration and then enlarged to a 2 X 2 cm tender area of fluctuance without erythema in her right lower anterior chest. A CT scan of the chest revealed bilateral pleural effusions with extenstion of the pleural fluid on the right between her ribs into the soft tissues of her anterior chest wall (emypema necessitatis). After approximately 4 weeks of antituberculosis therapy she underwent a right video assisted thoracoscopy (VATS) with complete decortication of the anterior chest wall and pleural abscess. The surgeon noted a clear tract through the chest wall into the right pleural space. The patient had an uneventful postoperative recovery with no drainage from the surgical incision.

CASE DISCUSSION

PATHOPHYSIOLOGY

Mycobacterium tuberculosis affects the pleura at different stages of pulmonary and systemic disease as the pleura can be involved in either primary or post-primary (reactivation) TB disease. In primary disease, pleural TB occurs as a result of mycobacterial antigen entering the pleural space, perhaps due to the rupture of a subpleural caseous focus, causing a delayed hypersensitivity (DTH) immunogenic reaction mediated primarily by CD4 cells but also involving other inflammatory cells and a complex array of inflammatory mediators including interferon gamma and tumor necrosis factor alpha. The initial cellular reaction in the first 2-5 days is macrophage predominate but from then onward, lymphocytic cells generally predominate and PPD reactivity is usually found. Paradoxically, PPD reactivity may be delayed in some patients, possibly as a consequence of the sequestration of DTH mediating cells, such as CD4 cells, in the pleural space. This form of TB pleural effusion is pauci-bacillary and tubercle bacilli are typically difficult to isolate in this setting. In contrast, tuberculous emypema is a chronic active infection of the pleural space that contains a large number of tubercle bacilli, also likely introduced into the pleural space via rupture of a sublpleural caseous focus. Other factors predisposing to this type of effusion may be progression of a primary TB pleural effusion or direct extension of infection into the pleural space from a tubercle on the surface of the lung or from thoracic lymph nodes. Hematogenous spread of the organism to the pleural space is also possible, especially in immune compromised patients. The accumulation of fluid is multi-factorial, but the primary mechanism is likely intense inflammation that impedes lymphatic drainage of the pleural space.

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CASE PRESENTATION—CONTINUED FROM PAGE 5

EPIDEMIOLOGY

The frequency of tuberculous pleural effusions varies by location. In the United States they are relatively rare, occurring in perhaps 3-5% of all tuberculosis cases, and are reportedly the 10th most common cause of pleural effusion in the U.S. (see below). In contrast, in areas of the developing world where TB is more endemic than in the U.S., TB is the most common cause of pleural effusion, especially exudative pleural effusion. In the industrialized world, pleural effusion with TB is more likely due to reactivation of TB disease whereas in the developing world it is more likely a manifestation of progressive primary disease. In that context, HIV coinfection and multidrug resistant TB are altering the epidemiology of TB pleuritis. In one series, the incidence of TB pleural effusions in HIV/TB coinfected patients was as high as 90%.

CLINICAL PRESENTATION

For some patients with TB pleural effusions associated with primary tuberculosis, the appearance of pleural fluid may occur without symptoms and the pleural effusion is recognized only when a chest radiograph is done as part of a routine evaluation. This combination of minimal symptomotology and lack of sensitive culture techniques (see below) would suggest that tuberculous effusions, especially those associated with primary disease are under-diagnosed. More commonly, however, the illness is manifested by onset of fever, cough, pleuritic chest pain and dyspnea. 90% of patients have symptoms for less than one month. TB pleural effusions affect one hemithorax in 90-95% of cases. Massive effusions are unusual. Untreated pleural effusion as a manifestation of primary TB is usually a self-limited inflammatory process. In 90% of cases, there is complete resolution, even without treatment, spontaneously occurring within weeks to months. However, even this relatively benign and self-limited process is an important harbinger of the progression to active pleuro-pulmonary or extra-pulmonary disease which occurs in 40-60% of patients with untreated pleural effusions within 5 years. Residual pleural thickening is also common and may occur in 50% of cases. Patients with TB empyema are invariably symptomatic with fever, sweats, cough, dyspnea and pleuritic chest pain. This type of effusion will not spontaneously resolve and may progress to more serious complications such as empyema necessitatis.

DIAGNOSIS

Tuberculin skin testing or interferon gamma release assay (IGRA). A positive PPD or IGRA, alone, is not adequate for diagnosing TB pleural effusion but can be important supportive evidence in the appropriate context. Conversely, a negative PPD or IGRA does not rule out the diagnsosis. As noted previously, sequestration of DTH cells in the pleural space may delay the onset of the positive PPD, but the majority of patients with TB pleural effusion (excluding patients with advanced immune suppression) will at some point in the course of the illness have a positive PPD or IGRA.

Thoracentesis to obtain pleural fluid for cellular, biochemical and microbiological analysis is an absolutely essential element for diagnosing tuberculous pleural effusions. There is no substitute for obtaining pleural fluid for analysis. TB effusions are exudative, sometimes with protein concentrations > 5 g/dL. The differential cell count is almost invariably lymphocyte predominant (>50% lymphocytes), although for some effusions, especially TB emypema, in the first few weeks there may be a polymorphonuclear (PMN) cellular predominance that evolves into the classic lymphocyte predominant effusion. Cellular findings in the fluid that do not suggest TB effusion include a differential cell count with > 5% mesothelial cells or > 10% eosinophils (unless there is concomitant pneumothorax or intrapleural bleeding which can be associated with a high pleural fluid eosinophil count).

Pleural fluid should be sent for AFB staining and culture, even though microbiological analysis is not a sensitive diagnositic tool. However, positive cultures remain the gold standard for diagnosis. AFB smears are positive in only approximately 20-25 % of TB effusions, although that percentage would be higher with TB emypema or in HIV coinfected patients. Cultures are positive in a many as 40% of TB

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CASE PRESENTATION—CONTINUED FROM PAGE 6

effusions, again higher with TB empyema and HIV coinfection. These figures vary with populations studied and the type of TB effusion present, but they demonstrate the importance of ancillary testing for confirming the diagnosis of TB effusion in a majority of patients.

Some patients, perhaps 20%, will also have evidence of parenchymal lung involvement associatedwith TB effusion. The parenchymal involvement is usually on the same side of the chest as the effusion. This percentage would likely be higher with the use of chest CT scanning. The yield from sputum analysis is sometimes confirmatory with sputum smears positive in approximately 20% of patients. Sputum AFB cultures may be positive in as many as 50% of patients with aggressive efforts to collect sputum , such as sputum induction. As with pleural fluid specimens, the yield of sputum AFB analysis varies with the population studied and the type of TB effusion.

Adenosine deaminase (ADA) is the enzyme that catalyzes the conversion of adenosine to inosine and is found in high concentrations in TB pleural effusions. Values of ADA > 70 U/L are virtually diagnostic of TB effusions and values < 40 U/L virtually exclude the diagnosis of TB effusion. The higher the ADA value, the more likely TB is the diagnosis. The differential diagnosis of elevated ADA in a pleural effusion includes empyema (bacterial) and rheumatoid pleuritis which can usually be eliminated as possibilities after taking into account the other factors included in the pleural fluid analysis such as cell type predominance in the pleural fluid. Overall, an ADA level > 40 U should raise suspicion of TB pleural effusion. In one recent meta analysis the sensitivity and specificity of ADA for diagnosing TB effusions were 92% respectively. ADA levels are widely available and relatively inexpensive . This test is almost universally recommended for suspected TB pleural effusions, but clinically underutilized. Some authors propose measurement of interferon gamma (IFG) in pleural effusions to differentiate TB effusions from other etiologies. An IFG cutoff of 3.7 IU/ml in one study had a sensitivity of 98% and specificity of 98% for diagnosing TB effusions. This test is not yet widely available or approved for use in this setting.

Closed pleural biopsies, while potentially quite helpful when performed by experienced clinicians is a procedure that is done with progressively less frequency due to declining numbers of clinicians who are comfortable and experienced with this procedure. Currently, pleural biopsies are more likely done via video assisted thoracoscopy (VATS) either by a pulmonary physician or thoracic surgeon. Pleural biopsies have the advantage of demonstrating pathologic changes such as granulomatous inflammation in the parietal pleura with AFB smears positive in 26% and cultures positive in 56 % in patient with TB effusions. The differential diagnosis of granulomatous pleuritis includes fungal disease, sarcoidosis, and rheumatoid disease. Pleural biopsies are generally reserved for patients who prove to be diagnostic delimnas after other less invasive evaluation is inconclusive. Additionally, biopsies may be necessary for patients in whom malignancy is a concern as malignant pleural effusions also present as a lymyphocyte predominate exudates.

The nucleic acid amplification tests (NAAT) offer extremely high diagnostic specificity (> 95%) but variable diagnositic sensitivity for non-respiratory specimens such as pleural fluid . Currently, neither of the commercially available NAAT are FDA approved for use in the diagnosis of extra-pulmonary TB. At some point, these test may prove to be a very valuable addition to the diagnostic evaluation of pleural TB.

DIAGNOSIS SUMMARY

The diagnosis of TB pleural effusion includes clinical suspicion, radiographic confirmation of pleural fluid, +/- radiographic evidence of parenchymal tuberculosis disease, thoracentesis with protein/LDH measurement, cell count and differential, ADA level, AFB smear and culture. Sputum AFB smear and culture (induced if necessary). Pleural biopsy would then be considered under some circumstances.

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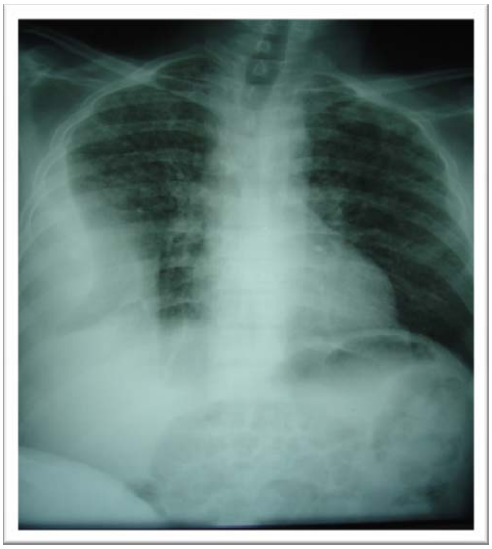
CASE PRESENTATION—CONTINUED FROM PAGE 7

TREATMENT

TB effusions associated with primary TB usually resolve spontaneously. For large effusions that cause respiratory compromise, an attempt to externally drain the effusion by thoracentesis or by pigtail catheter would be indicated. Indwelling tube thoracostomy is rarely necessary in this circumstance. Appropriate antituberculosis therapy should also be instituted. The role of steroids is controversial in this context with the exception of effusions associated with immune reconstitution syndrome in which case, steroids may be of substantial benefit, as with other manifestations of the IRIS syndrome.



(Above) Chest radiograph of a 50 year old man with pleuropulmonary TB with loculated right pleural effusion (TB emypema). Patient did not have significant improvement with medication alone and had significant lung restriction at the end of therapy. He required thoracoscopic pleural decortication in the right chest.

TB emypemas should also be approached initially in a conservative fashion with institution of adequate multidrug antituberculosis therapy and an attempt to drain the effusion by thoracentesis or pigtail catheter. These effusions a) tend to recur and b) tend to septate and loculate, so it may be difficult to completely remove the fluid by external drainage. Sometimes serial thoracenteses are necessary to minimize fluid reaccumulation. Even complete removal of pleural fluid, however, may not decrease the amount of residual pleural thickening. Tube thoracostomy should only be considered under specific circumstances such as bronchopleural fistula, which is likely not to heal spontaneously. Chest tubes tend to be associated with prolonged fluid drainage and may also be associated with pleural-cutaneous fistulas when removed. Pleural decortication is generally not considered until the patient has had at least 6 months of therapy and then only if there is significant residual pleural fibrosis with pulmonary function restriction. The majority of TB emypemas will improve sufficiently (minimal residual pleural thickening/fibrosis) with medication alone so that surgical intervention is not necessary. Surgery may be necessary with a thick residual pleural rind and significant lung restriction or in the rare case of emypema necessitatIs.

EMYPEMA NECESSITATIS

Empyema Necessitatis is a rare complication of tuberculous empyema whereby the infected pleural fluid penetrates through the pleural space and chest cavity into the chest wall and then, without intervention, may spontaneously drain through the skin of the chest wall, creating a pleural-cutaneous fistula (and sometimes a bronch-pleuro-cutaneous fistula). Extension of the purulent fluid occurs along the path of least resistance and along tissue planes. Increased pressure within the pleural loculation, chonic inflammation, and necrosis with erosion and fluid extension all contribute to this process. TB is reported to be the most common cause of empyema necessitates in the U.S. followed by actinomycosis. Prior to the advent of effective antituberculosis drugs, empyema necessitatis was associated with a high mortalitity. In the current era of effective anti-tuberculosis medication, the occurrence and mortality of this process have dramatically declined. In fact, it has become so unusual, that few current TB clinicians have experience with emypema necessitatis and few reference texts or review manuscripts offer specific instruction or advice on handling this potentially dangerous and disfiguring TB complication. The consensus appears to be that aggressive measures are necessary including appropriate aggressive antituberculosis therapy, early surgical intervention, preferably by VATS, with elimination of the abscess penetrating the chest wall and decortication of the adjacent infected pleural space. Early recognition is the key to successful therapy. Consultation with clinicians, including thoracic surgeons, experienced with empyema necessitatis is strongly advised.

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CASE PRESENTATION—CONTINUED FROM PAGE 8

TEACHING POINTS and KEY CONCEPTS

1. Pleural effusions are a relatively uncommon complication of tuberculosis but can occur with primary tuberculosis, reactivation tuberculosis and as part of an immune reconstitution response.

2. Pleural effusions associated with primary tuberculosis are due to a delayed type hypersensitivity response to tuberculosis antigens in the pleural space. These effusions are generally self limited and resolve spontaneously.

3. Tuberculous empyemas are due to persistence of *M. tuberculosis* organisms in the pleural space that results in a purulent effusion that will not resolve spontaneously.

4. The evaluation of pleural effusions in patients with suspected or diagnosed tuberculosis requires a thoracentesis. Most primary care clinicians should be able to perform this procedure. Ultrasound guidance is a useful adjunct for safely guiding the placement of the thoracentesis needle/catheter.

5. Pleural fluid from the thoracentesis should be sent for

    a. LDH and protein concentrations
    b. Cell count and differential
    c. AFB smear and culture
    d. Adenosine deaminase (ADA) level

6. The characteristic profile of a tuberculous pleural effusion is an exudate with a lymphocyte predominance associated with an ADA level > 40 U/L. AFB cultures are positive in less than 50% of tuberculous effusions, but are highly specific when positive.

7. Patients with pleural effusion or empyema who are suspected to have active TB should have smears and cultures of sputum in addition to those done on the pleural fluid. Nucleic acid amplification (NAA) should be considered if sputum is AFB smear negative as up to 80% of smear negative active TB will be positive by NAA. Sputum smears are negative in 50% of persons with active pulmonary TB. Even in the absence of obvious pulmonary densities, sputum cultures will be positive 40-50% of patients with TB involving the pleural space. Induced sputum should be attempted if the patient is not able to spontaneously provide natural sputum. Having the patient try to obtain a specimen upon arising after a warm shower may also be helpful if sputum induction is not available.

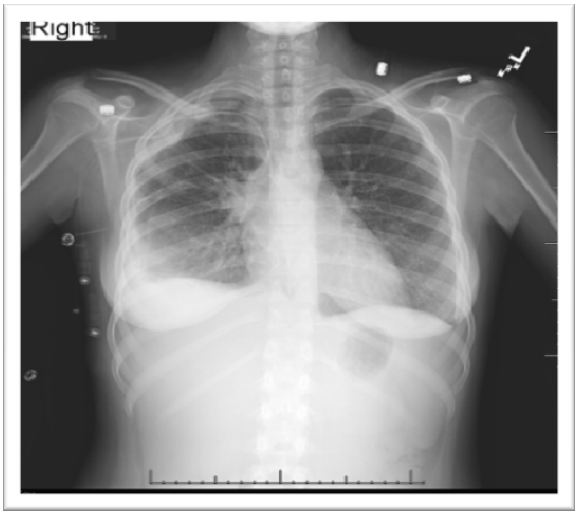
8. In general, effusions associated with primary TB require intervention only if there is respiratory compromise. TB empyemas should be drained by thoracentesis or pigtail catheter if possible. Repeated procedures may be necessary for recurrent effusions. Tube thoracostomy should be reserved for special circumstances such as TB empyema associated with bronchopleural fistula.

9. Surgical intervention (pleural decortication) for patients with TB empyemas is usually reserved for patients who have been treated with adequate antituberculosis therapy at least 6 months and have large residual pleural fluid/fibrosis with significant lung restriction.

10. Treatment of empyema necessitatis involves both adequate antituberculosis therapy and surgical drainage of the chest wall abscess as well as the pleural space abscess with local pleural decortication.

11. For questions about management of TB pleural effusions, expert consultation should be requested.

CASE PRESENTATION—CONTINUED FROM PAGE 9



(Above Left) 15 year old female with pleuropulmonary tuberculosis. Bilateral primarily nodular densities in the lung parenchyma with loss of the right costophrenic angle due to a pleural effusion.

(Above Right) Chest radiograph of the same 15 year old female after thoracoscopic drainage of the chest wall abscess and tuberculous empyema in the right pleural space with pleural decortication in the lower right pleural space.

(Left)) Chest CT cut from the same 15 year old female demonstrating extension of the pleural fluid into the chest wall causing a bulge in the skin over the abscess created in the chest wall.

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RELEASED OCTOBER 2010

CDC Surveillance Report: Reported Tuberculosis in the United States, 2009

http://www.cdc.gov/tb/statistics/reports/2009/default.htm

At this link you can find the following:

Full Report (PDF – 5MB)

Preface (PDF – 452k)

Executive Commentary (PDF – 270k)

Technical Notes (PDF – 351k)

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Morbidity Tables Reporting Areas, 2009

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Morbidity Tables, Cities and Metropolitan Statistical Areas, 2009

Appendices

Surveillance Slide Set, 2009

CDC. Reported Tuberculosis in the United States, 2009. Atlanta, GA: U.S. Department of Health and Human Services, CDC, October 2010.

T-Bit

NEW: Self-Study Modules on Tuberculosis, 1—5

The Centers for Disease Control and Prevention (CDC), Division of Tuberculosis Elimination (DTBE), is pleased to announce the release of the *Self-Study Modules on Tuberculosis, 1-5 Slide Sets*. These slide sets were developed as an accompaniment to the print-based *Self-Study Modules on Tuberculosis, 1-5* to aid in the presentation of module content for a facilitator-led training. These educational modules are designed to provide basic information about tuberculosis for health care workers, including outreach workers, nurses, physicians, and health educators. The slide set training package consists of a facilitator guide, presentation slides for each module, and participant slide handouts for each module. The topics included are as follows:

- **Module 1:** Transmission and Pathogenesis of Tuberculosis
- **Module 2:** Epidemiology of Tuberculosis
- **Module 3:** Targeted Testing and the Diagnosis of LTBI and Tuberculosis Disease
- **Module 4:** Treatment of Latent Tuberculosis Infection and Tuberculosis Disease
- **Module 5:** Infectiousness and Infection Control

Slide sets: www.cdc.gov/tb/publications/slidesets/selfstudymodules/default.htm.
Modules: www.cdc.gov/tb/education/ssmodules/default.htm.