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Bilateral pneumonia: Lessons learned from an unusual presentation

A GP's experience with a case of mercury poisoning illustrates the importance of eradicating the word "assume" from the physician's vocabulary.

ABSTRACT: An extremely ill man was initially diagnosed with bilateral pneumonia, despite some findings that did not fully support this diagnosis. These findings included mild anemia, normal white blood count, mild elevation of liver enzymes, profound nausea and vomiting, and marked gingivitis. The patient was hospitalized for 1 week and treated with antibiotics. When he visited his GP for follow-up the patient admitted to self-induced mercury poisoning. His destructive gambling addiction had led him to heat liquid mercury and inhale the vapors in an attempt to shorten his life and provide his family with financial assistance. The patient underwent chelation therapy as recommended by a consultant from the BC Poison Control Centre, and eventually completed two 19-day courses of meso 2,3-dimercaptosuccinic acid. Five years later the patient remains well and the GP uses his experience with this case to help medical students appreciate the importance of a differential diagnosis and the value of resources available through organizations such as the Poison Control Centre.

The roller-coaster pace and variety of cases handled by a GP give general practice an addictive quality. After three decades, I am frequently reminded of the unpredictability of our profession. With the exceptions of birth and death, there are few situations that I have not encountered in my office. Recently I was convinced that my list of exceptions would be reduced to one.

Initial presentation

Upon entering the examining room I was stopped in my tracks. The patient, a 43-year-old pipe fitter, was slumped in a chair *in extremis*. His chief complaint was straightforward: "I feel terrible!" He was pale, dyspneic, coughing, and had persistent rigors. There was questionable cyanosis of his lips and hands. He had a tachycardia of 120, hypotension with blood pressure 100/60, and a temperature of 39°C.

My initial concern was not diagnosis or treatment, but rather how to ensure urgent transfer of the man to the Royal Columbian Hospital emergency department before he suffered cardiopulmonary arrest in my office. He informed me that he had driven his vehicle to the office but had needed to pull over on two occasions because he was on the verge of losing consciousness. After asking my MOA to place an urgent call for an ambulance, I returned to continue my triage.

History

I soon learned that the patient had been well until 6 days earlier, when he experienced onset of pyrexia, pharyngitis, nausea, epigastric pain, and back pain. In addition, he had polydipsia and oliguria. During that time he had been assessed in the Burnaby General Hospital emergency department on two occasions. Initial treatment was with azithromycin (Zithromax). This was discontinued on the second visit because of recurrent emesis. Levofloxacin (Levaquin) was started with an initial intravenous dose and continued orally. The patient stated that he had undergone a chest X-ray that was "positive."

NOTE: This case was previously described in a classical case history published by Drs Glezos, Albrecht, and Gair in the *Canadian Respirology Journal* in 2006.[1]

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His past medical history included myocardial infarction at age 32, COPD at age 40, and hyperlipidemia at age 43. His surgical history included tonsillectomy, vasectomy, and right wrist ganglion excision. He had smoked one-half to one package of cigarettes per day since age 14. Three months previously he had been assessed in the Royal Columbian Hospital emergency department for suicidal ideation and gambling addiction.

An abbreviated physical examination revealed marked purulent gingivitis and a 1-cm tender left axillary lymph node. Chest auscultation was limited by the patient's agitated state. Heart sounds were distant and basal air entry was diminished. No adventitious sounds were noted.

He was transferred urgently by ambulance to hospital for assessment and treatment. On admission his temperature was 39.8°C, blood pressure 114/78, heart rate 116, and oxygen saturation 98% on room air. Diagnosis was bilateral pneumonia, which was confirmed radiologically with images showing patchy consolidations (**Figure 1**). The radiologist's report questioned the possibility of immunosuppression or aspiration. For this reason, the patient was seen in consultation by a respirologist and infectious disease consultant. The consensus was atypical community-acquired pneumonia, such as mycoplasma pneumonia.

Additional investigation results included hemoglobin 114 with normal white blood count and differential with reactive lymphocytes. Initial platelet count was normal at 267 but rose to 450. Vitamin B_{12} and ferritin levels were normal. Results from a Monospot test were negative. Random glucose was 7.0 mmol/L with follow-up fasting at 5.5 mmol/L. Assessment of electrolytes showed slightly depressed sodium of 134 and 132 mmol/L but a normal value of 136 on follow-up. Urea and creatinine levels were normal. Total protein was initially low at 54 g/L, with a normal result of 64 g/L upon follow-up. Liver function tests showed low albumin of 26 and 29 g/L. The patient's alkaline phosphatase level was normal on admission at 73 but rose to 135 U/L (reference range < 125 U/L). Gamma-glutamyl transferase level was elevated at 62 and 103 U/L (reference range < 49 U/L). Alanine amino transferase level was normal at 23 and rose to 52 U/L (reference range < 50 U/L), and aspartate aminotrasferase level was normal at 20 and rose to 44 U/L (reference range < 36 U/L). Bilirubin levels were normal on two occasions at 4 and 9 μmol/L (reference range 3 to 17 μmol/L). Urinalysis showed trace protein but findings were otherwise negative. Hepatitis screen was nonreactive for anti-HBs and anti-HCV. HIV and syphilis serology were nonreactive. Sputum culture grew more than three respiratory flora and blood culture showed no growth after 5 days.

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Treatment

In the emergency department treatment was initiated with intravenous fluids and levofloxacin (500 mg every 24 hours). On the day of admission the patient developed a generalized erythematous macular rash. This was felt to be a drug eruption and the antibiotic regimen was changed from levofloxacin to intravenous cefuroxime (750 mg every 8 hours) and oral clarithromycin (500 mg twice a day). The rash faded over several days and the cough and emesis resolved. The left axillary node became nontender and gingivitis abated. By day 4 in hospital the patient was afebrile. He was discharged after 1 week with a prescription for clarithromycin (500 mg twice a day) for an additional week.

Follow-up

The patient returned to the office 1 week later for follow-up. He had finished the course of clarithromycin. Other than one episode of hemoptysis after smoking marijuana at a party he was asymptomatic. Chest auscultation was normal. No further antibiotic therapy was prescribed. Follow-up CBC, liver function tests, and chest X-ray were arranged at this time. These results all returned as normal.

After this visit I found on my desk a handwritten letter marked "personal and confidential." Past experience has taught me that such documents fit into one of two categories: complimentary or litigious. I was wrong. At the end of the day, in the solitude of my office, I read the two-page document. The first two sentences gave me pause for thought:

"I am writing this letter because I didn't have the ability to explain to you what caused my illness, so here goes. The illness was caused by mercury poisoning that was self-induced as I had a pound of liquid mercury which I heated up to release gas which I inhaled—the reason being that it would cause damage which would shorten my life expectancy so then I would maybe get a letter from you stating that I had a shortened life expectancy so that I could collapse my locked-in RRSPs. I know it wasn't very smart but this past year has been the worst of my life and I just wanted to be able to give my family something back after what I did to them, as emotionally I was unable to work. I don't know if I did any real damage to myself or not but I have to cross that bridge if I come to it. In closing I would just like to apologize for my actions.

"I think I am safe from inflicting any more hurt on myself but I guess only time will tell. Once again I am sorry but I just couldn't see any other way and I just want you to know."

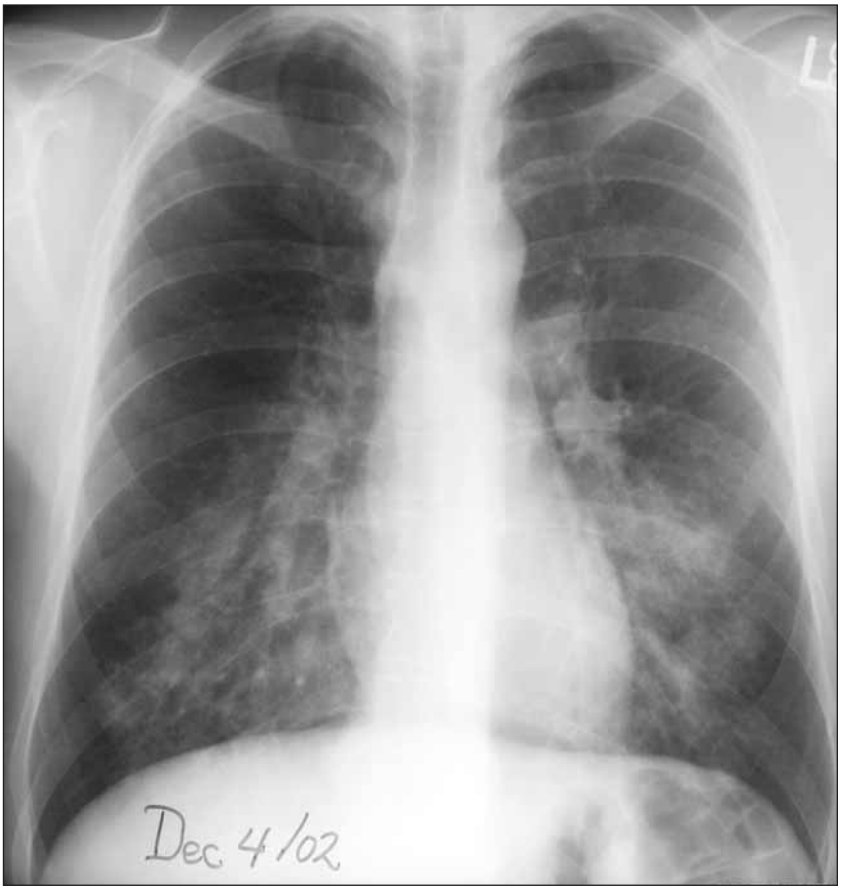

Figure 1. Chest X-ray on admission showing bilateral lower lobe patchy consolidations.

Treatment following disclosure

My past experience with acute or chronic mercury poisoning was nonexistent. The toxicologists and consulting physicians at the BC Poison Control Centre proved to be an invaluable resource and led me by the hand. They recommended a 24-hour urine sample for mercury analysis and mailed a photocopied chapter on mercury from *Goldfrank's Toxicologic Emergencies*.[2] This document filled in many pieces of the puzzle, including the gingivitis, gastrointestinal symptoms, and possibly the rash. It did not explain the anemia, lymphadenopathy, or hepatic dysfunction.

A 24-hour urine sample was collected and tested for mercury 1 month after the patient was admitted to hospital. Results were reported 2 weeks later at 2377 nmol/L (reference range < 50 nmol/L)—well into the toxic range. A consultant at the Poison Control Centre noted that this level could lead to permanent neuropsychiatric changes and recommended immediate therapy with a chelating agent, meso 2, 3-dimercaptosuccinic acid (DMSA or succimer). This required submitting a

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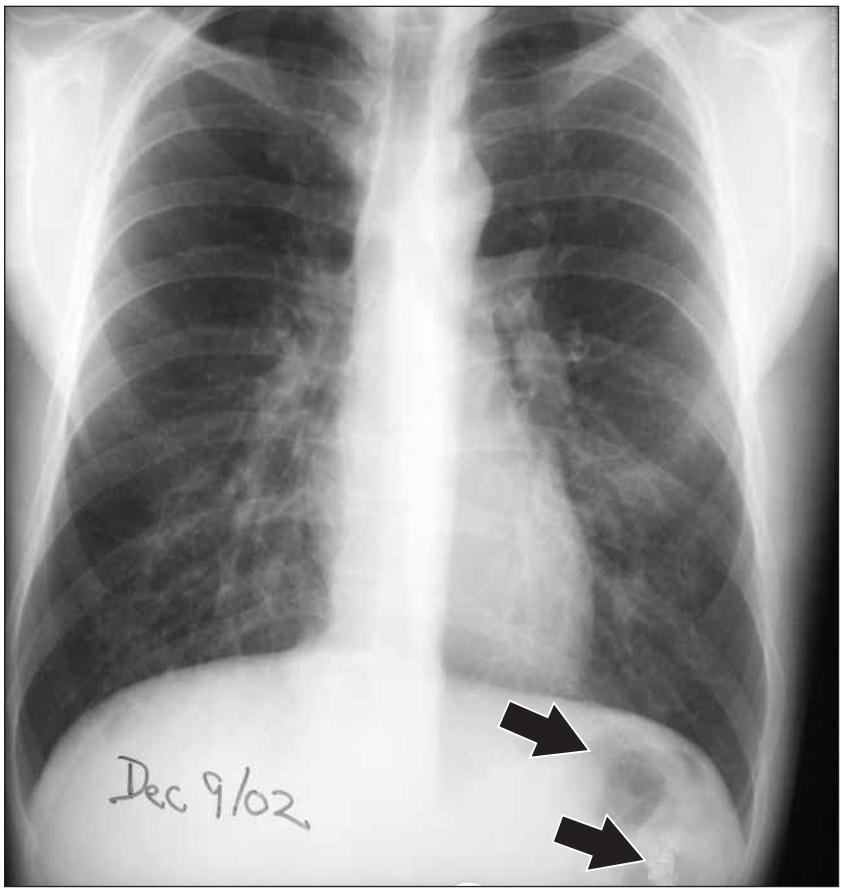

Figure 2. Resolving bilateral lower lobe pneumonia. Arrows indicate the radiodense globules located in the splenic flexure.

request to the Special Access Programme in Ottawa. A 19-day course of therapy was then supplied to the patient, who took 700 mg (10 mg/kg) every 8 hours for 5 days and 700 mg every 12 hours for an additional 14 days.

Once the patient's abnormal urine mercury level was identified, the Poison Control Centre notified the public health office to ensure that an environmental health hazard did not result from this man's action. An on-site inspection was carried out at his residence, where spot testing in the garage and house revealed traces of mercury on a propane bottle and near a stove fan. A discussion by conference call led to the conclusion that there was no health risk.

After the patient completed the first course of succimer, a follow-up 24-hour urine mercury test found a markedly reduced level of 424 nmol/L at 3 months postadmission. This level was still in the toxic range, so after discussion with the Poison Control Centre consultant a second identical course of medication was requisitioned at 4 months postadmission. After the patient completed this course of succimer, a follow-up urine mercury test found a level of 56 nmol/L at 7 months postadmission. A toxicologist at the Poison Control Centre felt that this level was nontoxic and no further chelation was required. The patient was notified of the result and stated that he remained asymptomatic.

During treatment the patient was also seen by Dr J. Glezos, respirologist, regarding the pneumonia and potential interstitial lung disease from the mercury inhalation. Pulmonary function studies showed a mild degree of airway obstruction that improved with bronchodilators. A high-resolution CT chest scan did not reveal evidence of diffuse interstitial lung disease or emphysema. Recently the patient's case history and serial X-rays were presented at a BC Thoracic Surgery meeting. During the discussion and radiological review, an unusual finding was noted on the chest PA view of day 5 (**Figure 2**): The radiodense globules in the region of the stomach and hepatic flexure of the colon were felt to indicate heavy metal ingestion, illustrating the infinite resolution of the retrospectoscope!

It is difficult to imagine the desperation that led him to heat a pound of elemental mercury with a propane torch under the stove hood in his kitchen in order to create toxic vapors that could shorten his life and thus provide access to locked-in RRSP funds.

Lessons learned

There are several lessons to be learned from this patient's presentation. The initial diagnosis of pneumonia was based on the history of pyrexia, productive cough, and radiological evidence of bilateral consolidation. The last feature was unusual and suggested the possibility of underlying immunosuppression or aspiration. Other unusual features for a diagnosis of bacterial or atypical pneumonia included the mild anemia, normal white blood count, mild elevation of liver enzymes, profound nausea and vomiting, and marked gingivitis. However, it was the unusual abdominal X-ray finding on day 5 that was the missing piece of the puzzle and could have led to an earlier diagnosis if the patient had been questioned about heavy metal exposure. This oversight, coupled with a delayed laboratory result, led to a 1-month lag in initiating chelation therapy. Despite this, the patient appears to have survived without any long-term sequelae.

In medical school we were taught that the majority of diagnoses are made on history and supported by physical examination. With this case, the diagnosis was provided belatedly by the history in the patient's disclosure, albeit after hospitalization and resolution of the acute phase. It is the first time that I have received significant diagnostic information in a letter. For this patient it was significant in that it triggered a chain of communication that culminated in two courses of chelation therapy. There was an initial delay in therapy caused by the need to consult about the unusual history and by laboratory lag time. Despite this, the patient responded to the therapy, his mercury levels dropped to the nontoxic level, and to date he is unscathed by permanent sequelae.

Until this encounter I was naive about the destructive potential of gambling addiction. This man's 5-year battle led to the loss of his house and financial duress for his family. It is difficult to imagine the desperation that led him to heat a pound of elemental mercury with a propane torch under the stove hood in his kitchen in order to create toxic vapors that could shorten his life and thus provide access to locked-in RRSP funds. The resulting cost to the health care system was not insignificant, taking into account 1 week of hospital care, outpatient therapy, and environmental inspection. My experience with this patient has increased my understanding of opposition to legalized gambling in our province. No doubt he represents the tip of the iceberg in my practice.

This man will require close follow-up for renal, pulmonary, and neuropsychiatric complications. Five years after presentation he remains well, continues to work at his trade, and—with the support of his family—is keeping the gambling monkey caged as well as possible.

Since this man's presentation I have taken on the challenge as a GP clinical instructor for first- and second-year medical students. I use this vignette to illustrate the importance of considering a differential diagnosis, using all resources available, and eradicating the word "assume" from our vocabulary.

Fortunately my list of personal practice exceptions still remains at two.

Competing interests

None declared.

References

1. Glezos JD, Albrecht JE, Gair RD. Pneumonitis after inhalation of mercury vapours. Can Respir J 2006;13:150-152.
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Acknowledgment

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