The Long Suffering of Frederic Chopin, Revisited

To the Editor:

The article by Kubba and Young attributing Chopin’s illness to α1-antitrypsin deficiency must be contrasted with the opinion of the world’s leading authority on diseases of the great composers, Anton Neumayr, MD. Neumayr, in an exhaustive, scholarly tome based on primary sources and a thorough understanding of 19th century medicine, concludes that both Emilia and Frederic Chopin died of tuberculosis. To cite only one of numerous disparities in the basic “facts,” Emilia’s death, according to Neumayr, was preceded by increasing cough and hemoptysis, not hematemesis.

Also, Solange is more correctly identified as the teenage daughter of George Sand and a central character in the final break between her mother and Chopin, rather than as a “friend.”

Finally, the link between bronchiectasis and α1-antitrypsin deficiency is extremely weak, casting further doubt on the proposed diagnosis of Kubba and Young.

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Chopin’s Malady

To the Editor:

I read with delight the article by Kubba and Young concerning the cause of Frederic Chopin’s chronic ailments. The general contention is that Chopin had long-standing tuberculous (TB) which eventually caused his death. However, Kubba and Young provide arguments against this, and while they freely admit that their evidence is not conclusive, they suggest that Chopin suffered from either atypical cystic fibrosis (CF) or α1-antitrypsin (α1AT) deficiency. I believe that neither of these two illnesses adequately explains Chopin’s malady and that TB is still most likely cause.

It is very unlikely that Chopin had α1AT deficiency because this disease rarely presents with respiratory problems before 25 years of age. Chopin’s chronic respiratory symptoms began at approximately 16 years of age, and his sister succumbed from a prolonged, possibly hereditary, respiratory illness when she was 14 years old. Seger et al evaluated 150 adolescents with α1AT deficiency, and they concluded that children with α1AT deficiency had a “favourable prognosis and normal lung development up to 16 years of age.” This is considerable evidence against Chopin having α1AT deficiency.

I also believe that Chopin did not have CF. If he had CF with pancreatic insufficiency, he would have died in infancy from malnutrition. Certainly, some of the rarer gene mutations are associated with more mild respiratory disease and pancreatic insufficiency. However, if Chopin had one of these “mild” genotypes, then his gastrointestinal (GI) symptoms would not be readily explained by CF, as he should have been pancreatic sufficient. Male infertility is almost universal in CF, but Chopin’s prolonged association with George Sand without production of children is not proof that he was sterile.

Chopin’s intimate relationship with Sand spanned 8 years (1838-1846), but only during the first year did they have sexual relations. On May 12, 1846 Sand wrote to a friend “For seven years I have lived as a virgin with him [Chopin] and all others.” The most compelling proof against CF is Chopin’s lack of digital clubbing. di Sant’Agnese and Davis reported the characteristics of 75 adults with CF, and they all had clubbing. Digital clubbing is a hallmark of CF, especially in its advanced stages, and the fact that Chopin did not have clubbing is strong evidence against a diagnosis of CF. Of note, the incidence of digital clubbing in males with advanced pulmonary TB is approximately 40%, so Chopin’s lack of clubbing certainly does not rule out TB.

Chopin probably did die from TB. Kubba and Young state that “it seems unlikely that with no treatment, someone would have over 24 years of recurrent tubercular infection,” but it is certainly possible to suffer for many years with pulmonary TB before dying. Rene Laennec, inventor of the stethoscope, first manifested symptoms of TB in 1803 and died from it in 1826, and Anton Chekhov contracted TB in his early 20s (approximately 1852) and succumbed to it in 1904. Recurrent hemoptysis, which characterized Chopin’s illness, is actually more frequent in chronic TB than it is in CF. Osler, in his textbook The Principles and Practice of Medicine, noted that 60-80% of patients with pulmonary TB had episodes of hemoptysis, and that it was more frequent in males. While mild hemoptysis is common in CF, the incidence of massive hemoptysis (>300 mL/day) is only approximately 7%. Also, TB could easily have caused the diverse respiratory signs/symptoms manifested by various members of Chopin’s family. We only have hearsay evidence from Chopin’s sister concerning the results of his autopsy, which by her account were not consistent with TB. Unfortunately, the official report of the autopsy has been destroyed, so it is difficult to accept her statement as fact.

The cause of Chopin’s GI complaints remains a conundrum. It is unlikely that he had pancreatic insufficiency and survived so long, but he might have had peptic ulcer disease (explains hematemesis) and/or gall bladder disease (explains fatty food intolerance). His icterus suggests that he had liver involvement. Chronic TB could have caused most of Chopin’s GI complaints. Osler wrote that secondary involvement of the bowels was very common in chronic pulmonary TB and that the liver could be involved, to include tuberculosis cirrhosis. However, he noted that TB of the liver rarely caused clinically important disease. Cor pulmonale resulting from chronic hypoxia due to end-stage lung disease could have caused Chopin’s peripheral edema and also his icterus (from hepatic congestion).

In conclusion, I believe that CF and α1AT deficiency are both unlikely causes of Chopin’s malady. While the actual etiology is

References

1 Kubba AK, Young M. The long suffering of Frederic Chopin. Chest 1998; 113:210-216

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