Plague—Older Than We Knew

Rasmussen and colleagues sequenced 89 billion bacterial DNA reads recovered from the teeth of 101 individuals in Europe and Asia who lived in the Bronze Age and identified 7 with sequences resembling Yersinia pestis. The teeth of the 7 were recovered in modern day Estonia, Russia, Poland, Armenia, and Siberia.

Molecular clock analysis estimated the date of divergence of early branching of the organism from its ancestor to be approximately 5783 years ago, 2 millennia earlier than previous estimates. The investigators were able to track the evolution of elements related to the pathogenicity of Y. pestis, finding that 6 of the 7 Bronze Age organisms lacked a gene present in current plague bacilli, ymt, that encodes a phospholipase that is necessary for its survival in its flea vector. The youngest sample of the 7, from an individual in Armenia dating to 951 cal BC, however, did carry ymt, indicating that the ability to be transmitted by fleas had been acquired by that time. Although the ancient organism carried Pla, a protein necessary for tissue invasion, it lacked a mutation required for the development of bubonic (but not pneumonic) plague.

These findings led the investigators to conclude that the ancestor of contemporary Y. pestis was present by the end of the fourth millennium BC and had spread across Eurasia, from modern day Estonia to Siberia, by the early third millennium BC—at least 3000 years before the Plague of Justinian, the first of 3 known plague pandemics, which began in the sixth century AD. The ancient version of the organism, however, lacked the ability to be transmitted by fleas and to cause buboes, but they could presumably cause pneumonic and septicemic infections. The ability to survive in fleas was, however, acquired by the beginning of the first millennium BC, setting the stage for the known (and perhaps unknown) plague epidemics and their profound effects on human evolution and civilization.

Trypanosoma cruzi Infection and Nonischemic Cardiomyopathy in California

Many Latin American immigrants are infected with Trypanosoma cruzi, a cause of nonischemic cardiomyopathy (NIC) that is often complicated by malignant arrhythmias. As a consequence, this infection must be considered as a potential etiology of NIC in immigrants from endemic countries.

Traina and colleagues examined the prevalence and impact of Chagas disease in all Latin American immigrants seen at Olive View–UCLA Medical Center in Los Angeles with newly diagnosed NIC. The diagnosis of Chagas disease required seropositivity in both of 2 tests at enrollment performed by the US Centers for Disease Control and Prevention (CDC): an immunofluorescence assay and an enzyme-linked immunosorbent assay (Chagatest ELISA recombinant version 3.0; Wiener Laboratorios, Rosario, Argentina).

The 135 enrollees were followed for a median duration of 43 months; 25 (19%) had T. cruzi infection. The median age of the total group was 57 years. The prevalence of Chagas cardiomyopathy (CCM) was 38% for immigrants from El Salvador, 25% for those from Guatemala, and 8% for those from Mexico. There were no differences in location of their residence (rural vs urban) in these countries or in type of house (concrete, adobe, or other) in a comparison between CCM and non-CCM groups. Those with CCM, however, had spent more time in their native country (41 vs 26 years; P = .002) and correspondingly less time in the United States (13 vs 24 years; P = .01).

The primary study endpoint, consisting of all-cause mortality or cardiac transplant, occurred in 9 of the 25 (36%) with Chagas disease and 11 (10%) of those without Chagas disease (hazard ratio [HR], 4.46; 95% confidence interval [CI], 1.8–10.8; P = .001). Reaching the primary endpoint was the result of death in 8 of 9; only 1 patient underwent cardiac transplant. The secondary endpoint, heart failure-related hospitalization, was reached in 14 (52%) and 35 (32%), respectively (HR, 2.22; 95% CI, 1.2–4.2; P = .01). Consistent with the high risk of life-threatening arrhythmias associated with Chagas disease, both receipt of amiodarone (40% vs 6%; P < .001) and the presence of an implantable cardioverter-defibrillator (36% vs 7%; P = .001) were more common in the CCM group. Patients with CCM were also more likely to have a right-bundle branch block (20% vs 1%; P < .001).

It is estimated that 8 million people in the endemic countries are infected with T. cruzi and the CDC estimates that >300 000 infected individuals reside in the United States. Although almost all of these acquired the infection in their countries of origin, autochthonous infections occur in Texas; of 17 prospective blood donors at a Houston donation site who were seropositive for T. cruzi, 5 (29%) were believed to have acquired their infection within the state [1]. Three of the 5 had electrocardiographic changes consistent with the presence of CCM. The 19% prevalence of T. cruzi infection among Latin American immigrants with CCM is similar to the 13% previously reported among a smaller number of subjects in New York City [2].
Preventive activities in the United States primarily consist of screening of blood and tissue donors, although vector control in areas such as Texas may also prove to be important. The identification of early infections and infection in pregnant women is critical as treatment with benznidazole is effective in preventing progression to cardiomyopathy and in the prevention of congenital infection. Benznidazole treatment of patients with established CCM, although effective in reducing parasitemia, unfortunately does not prevent clinical cardiac deterioration [3].

**References**


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**Case Vignette: Staphylococcus aureus: It’s MSSA—No, It’s MRSA**


A 60-year-old man with a chronic tracheostomy who was known to be colonized with methicillin-resistant *Staphylococcus aureus* (MRSA) developed bacteremia due to *S. aureus*. The organism was reported to be methicillin-susceptible *S. aureus* based on the results of cefoxitin disk diffusion, oxacillin screening agar, and the Vitek AST-GP67 susceptibility test. Polymerase chain reaction testing, however, demonstrated that the isolate carried *meca*, and a latex particle agglutination test demonstrated the presence of PBP2a. The isolate appeared susceptible to oxacillin (minimum inhibitory concentration [MIC] < 2 µg/mL) by microbroth dilution with incubation up to 48 hours; with continued incubation, the MIC had increased to 32 µg/mL by day 5 – consistent with inducible resistance. No information is provided on the antibiotic therapy the patient received.

This case illustrates some of the complexities that have been emerging with regard to the recognition and treatment of *S. aureus* infections. The bloodstream isolate from this patient (MRSA was recovered from his respiratory secretions) appeared oxacillin susceptible by standard phenotypic testing, but the detection of *meca* and PBP2a provided evidence of resistance (although mutated *meca* that fails to encode a functional PBP2a is reported). Finally, inducible resistance, something that would not be recognized by routine testing in the clinical laboratory, was present.

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