VACCIREVIEW

Two-case cluster of rapidly progressive influenza b and staphylococcus aureus pneumonia with one death

Bibliography

Zheng C, Zhang Y, Wang Y, et al. Two-case cluster of rapidly progressive influenza B and Staphylococcus aureus pneumonia with one death. Int J Infect Dis. 2026. doi: 10.1016/j.ijid.2026.108442

Summary

This case report describes two previously healthy construction workers (39 and 36 years) who developed fulminant pneumonia after a shared occupational exposure, with one fatal and one surviving case. Both dismantled a brick wall without respiratory protection and developed high fever, dry cough, and sore throat within 48 hours, consistent with an initial influenza-like illness.

By day 5, Case 1 presented in extremis with diffuse bilateral consolidation and early cavitation on chest CT and died within hours of admission from respiratory failure; no microbiologic samples were obtained, but the pattern was highly suggestive of necrotizing pneumonia. Case 2 was admitted on day 6 with severe pneumonia, septic shock, and multi-organ failure (lactic acidosis, acute kidney and liver injury, myocardial and skeletal muscle involvement), alongside extensive bilateral pulmonary infiltrates with right-sided predominance. Initial external PCR testing yielded a complex mixture of signals (S. aureus and rhinovirus in blood; Klebsiella pneumoniae and rhinovirus in sputum), leading to empiric therapy with teicoplanin, meropenem, peramivir, and low-dose corticosteroids.

In-house metagenomic next-generation sequencing (mNGS) of sputum, and later BALF, clarified the picture by identifying influenza B virus and Panton–Valentine leukocidin (PVL)–positive methicillin-susceptible Staphylococcus aureus (MSSA) as the clinically relevant co-pathogens, while reclassifying Klebsiella and rhinovirus as incidental. Despite transient improvement in systemic biomarkers (lactate, CRP, PCT), Case 2 developed persistent high-grade fevers, rising leukocytosis, and radiographic progression to bilateral necrotizing pneumonia with cavitation, prompting bronchoscopy. BAL culture and mNGS confirmed abundant MSSA, and whole-genome sequencing showed a PVL-positive ST22 clone with a resistance profile limited to penicillin G, consistent with emerging community-associated MSSA in China.

Teicoplanin was then replaced by linezolid to optimize lung tissue penetration, without other major changes in supportive care at that time. After this switch, fever and leukocytosis resolved, and CT on day 36 showed substantial radiological improvement,

allowing discharge on day 38. The authors conclude that PVL-positive MSSA, in synergy with influenza B, was the main driver of necrotizing lung injury and outcome, and they advocate for early mNGS and use of lung-penetrant anti-staphylococcal agents in similar scenarios.

Comment

This report is a compelling illustration of post-influenza necrotizing pneumonia in previously healthy adults in which PVL-producing MSSA, enabled by influenza-mediated epithelial and innate-immune disruption, is the dominant driver of fulminant lung destruction rather than "severe viral pneumonia" alone. The tightly aligned temporal data (brief flu-like prodrome, abrupt respiratory collapse, early cavitation, leukopenia followed by marked neutrophilic rebound) and the mNGS-based clarification of PVL-positive ST22 MSSA plus influenza B as the true pathogen pair are major strengths, as is the pharmacologic rationale for switching from teicoplanin to linezolid, even though this remains an uncontrolled n=1 observation. Key limitations are the lack of microbiology in Case 1 and the absence of environmental or carriage studies, which preclude firm conclusions about transmission pathways. Clinically, the message is clear: in rapidly progressive, cavitating pneumonia after influenza, S. aureus—often PVL-positive MSSA—should be assumed until disproven, with early deep sampling, high-resolution diagnostics and lung-penetrant anti-staphylococcal therapy; **more broadly, seasonal influenza vaccination, even in healthy young adults, may prevent not only viral illness but also such catastrophic bacterial superinfection and death.**

*Brought to you by Chief Editor **Joe Schmitt** – Supported by AI*