

FATAL BACTERAEMIC PNEUMONIA DUE TO COMMUNITY-ACQUIRED METHICILLIN-RESISTANT STAPHYLOCOCCUS AUREUS

Dear Sir,

We read with interest Drs Chua and Lee's report of two fatal cases of pneumonia caused by methicillin-resistant *Staphylococcus aureus* (MRSA)⁽¹⁾. While we welcome any report that highlights the emerging problem of community-associated MRSA (CA-MRSA) in Singapore, there are some issues that concern us.

Firstly, although the terminology is confusing, it is clear that CA-MRSA and healthcare-associated MRSA (HA-MRSA) isolates are distinct entities on a molecular level, with differing genetic profiles and evolutionary paths⁽²⁾. MRSA infections that arise outside the hospital setting are conventionally labeled as "community-acquired" or "community-onset". These can be caused by both the newly-emerged CA-MRSA strains as well as HA-MRSA strains that have "escaped" out of the hospital via colonised patients. Conversely, in areas where the prevalence of CA-MRSA is high, nosocomial infections are increasingly being caused by CA-MRSA⁽³⁾. Therefore, using purely conventional epidemiological criteria – such as criteria suggested by the Centers for Disease Control and Prevention, Atlanta⁽⁴⁾ – to distinguish between them is unreliable. The duration of HA-MRSA carriage in patients discharged from hospitals may exceed two years⁽⁵⁾, and closer evaluation often results in the identification of healthcare links in "community-acquired" infections in Singapore⁽⁶⁾ as in other settings.

Secondly, susceptibility to non-beta-lactam antibiotics per se does not distinguish CA-MRSA from HA-MRSA even in Singapore. The past three years has seen the introduction and spread of a multidrug-susceptible epidemic HA-MRSA, UK-EMRSA-15, in local hospitals⁽⁷⁾. This MRSA has the same antibiotic susceptibility profile as the isolate from Patient 2 in the report⁽¹⁾. A minor variant UK-EMRSA-15 clone currently circulating in Singapore is even susceptible to both erythromycin and clindamycin as a result of the loss of the macrolide resistance *ermC* gene [unpublished data, Hsu LY]. An observation that we had made was that the majority (>99%) of local HA-MRSA isolates are resistant to ciprofloxacin, whereas the converse is true of local CA-MRSA isolates⁽⁸⁾.

It would have been of great interest if the two MRSA isolates described in the report had undergone molecular typing or had at least been tested for the presence of Panton-Valentine leukocidin genes. Without the molecular evidence, it is quite possible that either or both of these cases might have been caused by UK-EMRSA-15 that had been transmitted to the patients during the course of one of their outpatient follow-up appointments, resulting in colonisation and subsequent infection as reported. We hope that the authors could provide readers with some of the molecular evidence to confirm that these are indeed CA-MRSA strains.

The need for definitive identification of, and differentiation between, HA- and CA-MRSA is, in our opinion, neither trivial nor pedantic. The former hardly transmits successfully in the community with the exception of institutional settings such as nursing homes or dialysis centres; various strains of the latter, however, have true epidemic potential in the community⁽⁹⁾. Knowledge of the real extent of the problem of CA-MRSA is necessary for appropriate calibrated measures in terms of dynamic outbreak modelling, infection control and/or empirical antibiotic therapy for community-associated infections.

As a final and less relevant point, true CA-MRSA causing severe community-acquired pneumonia in Singapore has been previously reported in the literature⁽¹⁰⁾.

Yours sincerely,

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