EDITORIAL

Does Presence of Sepsis by itself Predispose Patient to HCAIs?

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Health care-associated infection (HCAI) is one of the most serious complications in critically ill patients as it leads to increase in ICU and hospital length of stay and increase in mortality. Medical literature is full of articles describing the risk of HCAIs in various patient populations. In this issue of IJCCM, Venkataraman et al. have tried to address the same.[1]

Most of the studies have shown that there are multiple risk factors for developing HCAI and include old age (>65 years), emergency admission to ICU, duration of hospitalization before ICU admission, presence of indwelling devices, such as central line, endotracheal tube, and urinary catheters, prolonged and extensive surgery, trauma, neutropenia, and parenteral nutrition. Over and above these factors, the health care practices, trained personnel, basic hygiene, nurse–patient ratio also contribute to the development of HCAI.[2]

Since these factors are common in the ICU population, whether critically ill patients have sepsis or not, there is no reason why the incidence of HCAI should be different.

The main reason for the belief that HCAIs incidence is likely to be more in septic patients, is the belief that septic patients tend to develop immunoparalysis, in addition to the other factors mentioned above.[3,4] However, this is not entirely true, as immunoparalysis in a critically ill patient is a very complex and ill-understood phenomenon. Immunoparalysis is seen in critical illness and involves alterations in both innate and adaptive immune responses, including neutrophil dysfunction, altered monocyte phenotype and antigen presentation capacity, lymphopenia and impaired lymphocyte responses, and elevated pro-inflammatory cytokines.

Immunoparalysis is the result of multiple factors and includes patient's genetic predisposition (predisposition to a compensatory anti-inflammatory response), which is a heritable trait.[5] It is also related to the degree of initial inflammation observed with a critical illness or injury, such as surgery, trauma, burns, or infection. Brain injury is also associated with immunoparalysis.

In 1986, Polk et al. undertook a systematic study of host defense processes in badly injured patients. They studied 20 adults with severe trauma to correlate the immunologic factors associated with the development of secondary infection and late mortality. They found that persistent impairment in monocyte antigen-presenting capacity was associated with the development of nosocomial sepsis.[6]

Many drugs, such as steroids, opioids, sedatives, catecholamines, and blood transfusion used in critically ill patients are known to contribute to immunoparalysis.[7,8] Device use is recognized as a high risk for device-associated nosocomial infections in ICU patients.[9,10]

Though it is not a consistent finding, studies also suggest that a persistently high level of therapeutic activity and persistently depressed consciousness after ICU admission are associated with the acquisition of nosocomial infection by critically ill patients.[11] Thus, the risk of acquiring HCAI will depend on the immune status of the patient, intensity of interventions, and of course adherence to infection control practices.

Hence, it makes sense to correlate the presence of immunoparalysis, presence of devices, degree of therapeutic activity, and mentation with HCAI rather than just whether the patient is septic or nonseptic.

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