lead to miss the diagnosis of PNX. A double lung point can be detected in PNX secondary to trauma when pulmonary contusions can cause pleural adhesion or, less frequently, even in spontaneous PNX of young adults."

Volpicelli 2013

Sonographic signs of complex pneumothorax

“Double lung point: when for some reason, the air of a pneumothorax is not free to float inside the pleural space, a minimal amount of pleural air may remain in the lateral or dorsal chest without migrating in the most superior area in a supine patient, which corresponds to the anterior-inferior chest zone. In this case, the operator may visualize two lung points, ie, the alternating patterns of sliding and non-sliding lung intermittently appearing at the two opposite sides of the scan. These two lung points represent the visualization of the two edges of the air trapped in the pleural space. Pneumothorax with air trapping may be caused not only by pleural adherences in chronic pleural and pulmonary diseases but also by acute lung contusions in blunt torso trauma. Even without abnormal pleural adherences, very small spontaneous pneumothoraces may not have enough pressure to allow complete detachment of the pleural layers and the floating of air towards the most superior chest areas. Being aware of this condition or in case of strong suspicion, the operator should always complete the scan of the lateral chest in the supine patient to confirm lung sliding even when this latter is first visualized in the parasternal anterior-inferior chest. In the unstable patient, this extension of the technique is less important. Presence of lung sliding in the anterior-inferior chest may conclude the ultrasound examination, unless the patient is intubated for pressure ventilation or is going to be transported by helicopter. In these two latter cases, the lateral chest should always be scanned to rule out even the smallest pneumothorax that may need to be monitored or warrant prophylactic drainage.”

Response to “Protective effect of tetanus antibodies”

To the Editor,

We thank Dr Gozdas for opening discussion regarding our recent case report of tetanus infection despite a protective tetanus antibody level [1]. We agree that the increased level of tetanus antibody may be somewhat attributed to the active tetanus infection. However, the accepted level of protective tetanus antibody must also be discussed.

The accepted level of tetanus antibody considered to be protective is largely based on the pivotal trial conducted by Sneath et al [2] in 1937. In this trial, 13% of guinea pigs with antibody titers more than 0.01 IU/mL still developed tetanus, whereas 3 subjects with titers less than 0.01 IU/mL did not develop the disease despite injection with a lethal dose. In addition, Goulon et al [3] studied 64 guinea pigs who underwent serum antitoxin testing before serotherapy to validate the protective level. They found that 24 subjects (37.5%) had antibody levels ranging from 0.002 to more than 0.1 IU/mL. Furthermore, 10 (15.6%) had levels greater than 0.01 IU/mL, what is now considered the protective level of antibody. These results, coupled with multiple case reports of tetanus infection despite adequate antibody levels, call into question what is considered a protective level of antibody.

As mentioned in our original text, there have been multiple case reports of patients with active tetanus infection despite a protective level of tetanus antibody [4-11]. Of note, all of the reported antibody levels were drawn before administration of tetanus immune globulin. Passen and Andersen [7] reported a case of severe tetanus infection in an individual who sustained a puncture wound 52 hours before arrival yet had an antibody level of 0.16 IU/mL. This patient had reportedly received tetanus vaccines 4 and 8 years prior, as well. Alternatively, Abrahamician et al [9] reported a case of tetanus in an intravenous drug user who had injected 3 weeks before presentation and had an antibody level of 0.16 IU/mL. This patient had an unknown vaccination status. As you pointed out, antibodies may decrease the severity of the disease; however, there may be a ratio of toxin to antibody that, if surpassed, may lead to active infection, no matter what the antibody level may be.

Coupling the debate of the actual level of antibody considered protective with case reports providing inconsistent antibody levels given time of injury and time of presentation can lead to much discussion surrounding our case report. The discovery of tetanus largely remains a clinical diagnosis as the antibody levels often do not result until days after presentation.

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References


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To the Editor,

We read recently the published case report in your journal by Vollman et al [1] with great interest. The authors described an interesting case that developed mild tetanus infection despite protective antibody level. The last booster vaccination is stated to have been administered 13 years before presentation. The high tetanus antibody level, measured after 10 days of exposure when the clinical symptoms appeared, however, may not be protective. This tetanus antibody level might be related to the recent natural infection [2]. To our knowledge, antibodies could not prevent the occurrence of clinical disease, rather, they would provide that the disease course would be mild. If the tetanus antibody level could have been studied soon after exposure, this would reflect baseline protective level and would have substantiated the authors’ conclusion.

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