
Bilateral chylothorax as a complication of internal jugular vein cannulation

Sir,

We thank the reader for the interest shown and an incisive analysis of our article.^[1] We would like to clarify the queries raised as follows:-

1. The mechanism of bilateral chylothorax after thoracic duct injury is not clearly known but has been hypothesized based on the various case reports that have been described in the past and in our article. Paw described a case of bilateral pleural effusion as a complication of left internal jugular venous catheterization for total parenteral nutrition.^[2] The author had suggested that the tip of the catheter might have migrated into the mediastinum and that fluid from the mediastinum had entered both the pleural spaces via anatomical communications, which have not yet been described. Similar instances where contralateral or bilateral effusions have occurred after central line insertion have been reported.^[3-7] Varache attributed it to a possible mediastinal pleural ruture.^[7] Naquib *et al.* have ascribed the phenomenon to the pressure difference between the mediastinal compartment and the pleural compartments, which leads to the extravasation of fluid from the mediastinum to bilateral pleural spaces. They also demonstrated the same with instillation of a radio-opaque dye through the central line and its appearance in the mediastinum^[4]
As described in our article, after the inadvertent injury to thoracic duct, during central venous cannulation, the chyle first leaks out either into the ipsilateral pleural space through a pleural breach that occurred during cannulation (which would explain the development of

unilateral chylothorax), or the chyle could extravasate into the mediastinal compartment. From there, after a latent period of about two days, the chyle enters one or both pleural spaces because of the pressure difference between the compartments. This would explain the instances of the contralateral and bilateral chylothoraces. The authors agree with the various mechanisms proposed by the reader except that occlusion or near occlusion of the thoracic duct (post-injury) were unlikely in our case considering the early and complete resolution that occurred. All the other stated mechanisms were plausible.

2. During our literature search we found five case reports where the mechanism of chylothorax was a direct injury to thoracic duct or lymphatic vessels. We had listed the same in our article and as shown in the table, all had either ipsilateral or bilateral chylothoraces. Contralateral chylothoraces have been described in those cases where the central venous cannulation had led to thrombosis of central veins. The extension of thrombi and back pressure effects may lead to ipsilateral, contralateral or bilateral chylothorax
3. The origin of the swelling in the supraclavicular fossa has not been conclusively determined, but has been observed in other reports too. Most experts believe that when the duct starts to leak, a mediastinal chyloma is first formed below the pleura which tracks up to appear above the sibson's fascia and causes the supraclavicular swelling. The same mediastinal chyloma may enter the pleural space to cause chylothorax.^[8-10] However, as mentioned earlier, the mechanism has not been conclusively elucidated.

We thank the reader again for bringing out these important anatomical considerations which are intuitively unclear and may require experimental studies for understanding the mechanism more explicitly.

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