pleural space was demonstrated. We believe the variable anatomy of the thoracic duct is probably not an explanation of why clinically suspected leaks were not demonstrated by lymphangiography, but rather resolution of the chylothorax by healing during the period of preparation for lymphangiography.

Injury to the lymphatic system, be it feeder lymphatic, thoracic duct, or one of its many anatomic variants, is the main cause of chylothorax in this surgical series. Yes other factors, such as increased lymph flow, altered lymphatic permeability, distal lymphatic obstruction, and venous occlusion, may complicate or aggravate surgical injury of the thoracic lymphatic system.

For any one patient, day-to-day clinical decisions can be made on total daily chest tube drainage. However, for summary statistics of this highly variable measurement (standard deviation > mean), the median, and not mean value, must be used.

References


Letter to the Editor

Does size matter?

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Keywords: Lung cancer; Non-small cell lung cancer (NSCLC); Stage IA; Size; Survival; Screening

Medical publications contain conflicting evidence on the important question of whether NSCLC size within stage IA impacts survival. Veeramachanenia et al. from Washington University present important new data to answer this question in February’s EJCTS entitled ‘Risk factors for occult nodal metastasis in clinical T1N0 lung cancer: a negative impact upon survival [1].’

Their retrospective review of 292 patients with clinical stage IA NSCLC demonstrates that a size increase of 1 cm increases the risk of nodal metastasis by a factor of 3.5. Such occult nodal metastasis results in strikingly reduced survival. Because cases with nodal enlargement on CT or hilar or mediastinal PET uptake were excluded, and because systematic mediastinal node dissection was not routinely performed, this figure may underestimate occult nodal metastasis. In their literature review, the authors cite multiple prior studies offering similar data and conclusions.

But does not this information merely confirm common-sense notions regarding tumor biology? Why is this information important?

Although most publications, including this one, suggest decreasing survival as small NSCLC grow larger, two influential articles from Duke University dispute this association. In 2000, Patz published a review of 510 NSCLC patients with pathologic stage IA disease and found no association between size and survival [2]. In 2001, the same investigators reviewed 620 NSCLC 3 cm or smaller and concluded that increasing size within the group had no significant effect on final stage of tumors [3]. They conclude that, because they found no evidence that size matters with regard to either stage or survival, the theoretical benefit of detecting very small lung cancers by computerized tomographic screening is questionable. One of the Duke investigators, Philip C. Goodman MD, has included this data and conclusions prominently in paid depositions and courtroom testimony for tobacco companies in two lawsuits that seek jury verdicts compelling tobacco companies to pay for medical monitoring of individuals with high lung cancer risk.

Who is correct? With regard to survival, the 2000 Duke data contains a very important intrinsic bias. By excluding clinical IA cases that subsequently are classified in higher final pathological stages, they publish misleading data leading to incorrect conclusions. The Washington University investigators have not repeated this mistake, carefully identifying those patients who are so reclassified and the resulting wide variance in survival. With regard to size and stage, the issue is more complex. In the 2001 Duke series, 16.8% of cIA NSCLC had pII-IV comparable to 16.1% in the Washington series but patients upstaged to IB are not identified, nor is survival of 104 higher-stage patients displayed. Furthermore, their statistical analysis contains a questionable manipulation of data. Although a statistically significant increase in stage IIIB by size is noted, they conclude that ‘with stage IIIB patients being excluded, no statistically significant difference was found to exist.’

My conclusion is that size does appear to matter, and that Veeramachanenia et al. have demonstrated that a striking increase in nodal metastasis with increasing size at least partially explains why this is true.

References

Letter to the Editor

Re: Is the Allen test reliable enough?∗

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Keywords: Radial artery; Coronary artery bypass grafting; Allen test

We agree with Kohonen et al. [1] that a negative Allen’s test is safe to harvest the radial artery. This is well known. We have harvested the radial artery in 881 patients with a negative Allen’s test without postoperative hand ischaemia [2]. Barner has done the same in 1364 patients [3] and Meharwal and Trehan in 3977 cases [4].


References


Letter to the Editor

Re: Perivascular tissue of internal thoracic artery releases potent nitric oxide and prostacyclin-independent anticontractile factor

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We read with interest the recent article by Malinowski et al. [1] on the release of a soluble anticontractile factor from perivascular tissue (PVT) of the internal thoracic artery (ITA). In this study the authors show that the PVT surrounding the ITA, the ‘gold standard’ graft in coronary artery bypass surgery (CABG), releases a nitric oxide (NO) and prostacyclin-independent anticontractile factor. The authors suggest that the presence of an active PVT might explain the functional difference between skeletonised and non-skeletonised ITA and influence vascular function after graft implantation. Possible drawbacks of removing PVT should be taken into account since preservation of this tissue might be beneficial.

Malinowski et al. suggest that the influence of PVT removal on the function of other vessels used for CABG, such as the saphenous vein, the radial or the gastroepiploic arteries should be analysed. Although we have not performed functional studies, we have recently shown that the PVT surrounding saphenous veins used as grafts in patients undergoing CABG exhibits positive endothelial nitric oxide synthase (eNOS) immunoreactivity, contains eNOS mRNA and