Reply to the Letter to the Editor

Reply to Goldsmith

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Keywords: Air leak; Lung resection; Sealant; Randomised trial

We thank Dr Goldsmith for the stimulating comments [1] concerning our recent study.

As a conclusion of this randomised study, we have stated that the Coseal sealant proves to be effective in reducing intra-operative and postoperative air leaks. This is clearly shown by the evidence that the number of patients presenting air-leak cessation in the Coseal group was more than the control group either intra-operatively (85.3% vs 59.4%) or postoperatively at 24 h (80.4% vs 59.4) and at 48 h (76.5% vs 58.4%) with statistically significant differences (intra-operative, \(p < 0.001\); 24 h, \(p = 0.001\); 48 h, \(p = 0.006\)). It is true, as Dr Goldsmith points out, that the number of patients without air leak was slightly fewer at 48 h than intra-operatively in both groups with a more evident reduction in the Coseal group (−8.8% in the Coseal group and −1% in the control group), but these data have no considerable impact on the statistical significance of the differences observed also at 48 h (\(p = 0.006\)), and therefore it should not modify the scientific message reported.

Revision of the records of the Coseal group patients showing re-appearance of air leaks at 48 h after initial air tightness has allowed us to establish (as reported in the text) that, in most of them, multiple air-leak sites were visible after resection, requiring the division of the standard quantity of the sealant in smaller doses for each parenchymal area to be sealed. This evidence may suggest the need for increased dose of sealant in patients with multiple sites of air leakage.

Our study showed no significant difference in the duration of hospital stay, as reported in most of the similar studies in literature [2,3]. However, this result should not be surprising since air leaks are not the only cause determining prolonged hospitalisation.

In Dr Goldsmith’s opinion the higher rate of postoperative re-appearance of air leaks after intra-operative cessation in the Coseal group patients may explain why there was no significant difference in the duration of hospital stay between the two groups. We believe this phenomenon could not justify the non-significant difference in the length of hospitalisation, because the mean air-leak duration resulted in significantly shorter duration in the Coseal group. It is therefore more likely that other medical and surgical factors may have influenced patients’ recovery.

References


Letter to the Editor

Tracheal rupture after endotracheal intubation

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Keywords: Tracheal lacerations; Conservative treatment; Good results

This article [1] describes a compilation of articles — historical ones, more than 30 years old, as well as the more recent ones.

Case reports are mixed, with smaller and more extended series, including a total of 178 cases within 50 publications (on average, approximately 3.6 per author) enriched with four of the author’s own cases (published in 2007 in Eur J Emerg Med, quoted in (1)).

Therefore, the value of each statistical analysis is more than self-limiting.

Moreover, to be precise, iatrogenic injuries and traumatic ruptures are caused by two absolutely different mechanisms. This makes it not only a linguistic difference.

Iatrogenic injury means: local tearing — low impact — longitudinal run — laceration. There is always a continuity of the airways.

Traumatic accidental injury means: high (general) impact — horizontal run — rupture. Consequently, there is usually or frequently no continuity between the proximal and distal part of trachea or bronchi.

Thus, it follows that ruptures have to be dealt with operatively, and lacerations can be dealt with conservatively — this differentiates between the two.

Although mentioned in the discussion but not described in the introduction, the incidence of tracheal lacerations is well defined [2,3].

We encounter tracheal lacerations in almost 1:20 000 cases, independently from the anaesthetist’s experience and the training status.

Therefore, although seemingly a rare event, in fact, it is not as unusual, because in high-volume clinics, more than 30 000 intubations are performed in a year.

References


The striking prevalence in women is well documented since many years, with about 80–90% clearly resulting from the smaller dimensions of the airways in women (see also the comment by Sameh Sensar in ICVTS 3(2004) 404).

Thus, there is no consensus about the treatment modalities, but there is an explicit trend to conservative treatment since one of the earlier publications of a series of conservatively treated patients in 1994 [4].

Indications for conservative treatment are listed briefly in Lampl [3].

Based on a personal experience with 25 cases (17 conservative and eight operative), it should be mentioned that (1) tiny tears of less than 2 cm are usually non detected (probably clinically unapparent); (2) the preferred site of lacerations is the lower third of the trachea, so it is nearly impossible to block the endotracheal tube cuff distal to the tear without proceeding endobronchially.

We could demonstrate substantially low mortality in the conservative group (6%; 1 in 17) as well as in the operative group (12.5%; one in eight); however, it does not compare with 71% in an operative series.

There is no doubt that the trans-cervical approach and its modifications, as well as the trans-thoracic repair, offer a good treatment option, especially in case of an intubation injury in preparation to endotracheal surgery.

References


The authors of the original paper [1] were invited to reply to this Letter to the Editor but they did not respond.

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Letter to the Editor

CD133 and non-small-cell lung cancer

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Keywords: CD133; Lung cancer

I read the recent publication by Tirino et al. with a great interest [1]. Tirino et al. reported that CD133-positive cells isolated from non-small-cell lung cancer were able to give rise to spheres and act as tumour-initiating cells [1]. Indeed, the clinical correlation between CD133 and non-small-cell lung cancer is the present focus in medical oncology. Hilbe et al. noted that CD133-positive cells might contribute to the tumour vasculature in non-small-cell lung cancer [2]. This result is concordant with the study by Tirino et al. [1]. However, there are also other interesting reports to be mentioned. Salnikov et al. reported that CD133 was indicative for a resistance phenotype but was not a prognostic marker for survival of non-small-cell lung cancer patients [3]. In addition, Meng et al. reported an interesting finding that ‘CD133 alone cannot be used as a stem cell marker for the lung cancer cells A549 and H446, and both the CD133+ and CD133− subpopulations contain similar numbers of cancer stem cells’ [4]. It can be seen that the exact role of the CD133 in lung cancer is still not completely known at present and further studies on this topic are needed.

References


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Reply to the Letter to the Editor

Reply to Wiwanitkit

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Keywords: Lung cancer; Cancer stem cells; CD133

We agree with the comments made by Dr Wiwanitkit [1] on publication by Tirino et al. [2], presumably based on his own authoritative experience and on the references he quotes in his letter. In this context, there is a small detail worth noting: apart from Ref. [2] — parenthetically consistent with our working hypothesis — the other two were published in 2009 (one not even out in print), while our paper was presented in June 2008.