Reply to the Letter to the Editor

Reply to Isaka et al.

Hideki Itano*
Department of Surgery,
Osaka Kosei Nenkin Hospital,
4-2-78 Fukushima, Fukushima-ku,
Osaka City, Osaka 553-0003, Japan
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We are thankful for this opportunity to reply to Dr Isaka’s letter to the Editor [1].

Our aim is to evaluate the optimal technique for combined application of fibrin sealant and bioabsorbable felt against alveolar air leakage, by comparing the newly developed Rub + Soak B method to various combined application techniques. Some studies have experimentally shown that the combined application of two materials is more effective than the fibrin sealant alone [2]; this combination is presently used widely in pulmonary surgery. In this context, we confirmed that the novel Rub + Soak B method was the most effective and reasonable combination technique wherein the sealing mechanism was supported by the physical properties of the fibrin sealant components [3]. However, Isaka has overlooked the main aspects of this study and has instead focused on a different issue; the pros and cons of possible pleural adhesion associated with the polyglycolic acid (PGA) sheet application. They have strayed from our study topic and have introduced other concepts. Authors should oppose and be wary of such an attitude.

Although the pros and cons of pleural adhesion have been debated, it should not be necessarily avoided only due to the undefined possibilities of re-thoracotomy unless the adhesion involves a massive portion of the lung surface. Pleural adhesion is a rather important useful phenomenon for the control of alveolar air leakage. In certain cases, immediate control of alveolar air leakage is prioritized to avoid complications. Avoidance of massive pleural adhesion should be considered for a more specific category of patients such as candidates for lung transplantation vulnerable to recurrent pneumothorax episodes (i.e., lymphangiomyomatosis or COPD).

With regard to Isaka’s experience of postoperative pneumothorax recurrence, the negative impact of adhesion of the covered site on patient management is unclear. Pleural coverage for alveolar air leakage control is usually limited and might not encumber re-thoracotomy when it is needed. Massive adhesion, e.g., adhesion of entire lobes, might be rare.

Furthermore, creation of an acidic environment by polymer degradation by pleural coverage with a PGA sheet is unconfirmed, as it is only based on two separate studies [4,5]. Pleural adhesion in a group with PGA sheet coverage reported in a previous study [5] might not be valid: pleural coverage with the oversized, comparatively bulky PGA mesh in a rat chest with a large thoracotomy (Fig. 1) considering the excessive PGA amount might not occur clinically considering the excessive PGA amount and its subsequent influence on a living body. Non-specific inflammatory response caused by the acidic environment causing pleural adhesion has not been proven either.

References


* Corresponding author. Tel.: +81 6 6441 5451; fax: +81 6 6445 8900.
E-mail address: itano@okn.gr.jp
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Letter to the Editor

Marijuana smokers and lung bullae

Alfonso Fiorello, Giovanni Vicidomini, Mario Santini* Thoracic Surgery – Second University of Naples, Italy
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We read with great interest the article by Beshay and colleagues [1] on the incidence of emphysema and secondary pneumothorax in young adults smoking cannabis. The link between cannabis and lung bullae is quite definite [2–4]. Retrospectively, Beshay compared the findings of 17 marijuana and nicotine smokers (4 of these added a sporadic use of cocaine, one LSD over 3 years, and one sporadic heroin abuse) affected by spontaneous pneumothorax for bullous lung emphysema (group I) with the findings of non-marijuana smokers affected by spontaneous pneumothorax in the same (group II) and in a different period (group III). The authors showed that the presence of lung emphysema on computed tomographic scan in group I was significantly different to groups II and III while no significant difference in the form of clinical, laboratory, histopathological findings and postoperative course was found. In the last 5 years at our institution, we observed seven Caucasian young male marijuana smokers affected by emphysema and secondary pneumothorax. All patients had a tobacco smoke history (mean 6 pack years): five patients were current smokers of marijuana only (MS) and two current smokers of cocaine plus marijuana (CMS) over 4 years.
In all patients α1-antitrypsin serum levels were normal (median 1.6 g/l). High resolution CT-scan showed that the emphysema was paraseptal in distribution and hence quite distinct from the more uniformly distributed bullae of centrilobular emphysema, typical changes associated with a lifetime of tobacco smoking. However CMS had multiple and larger emphysematous bullae with pleural thickening (size 12 cm) than MS affected by small bilateral bullae (size from 0.5 to 7 cm). Spirometric tests of CMS confirmed reduced FVC and FEV1 value with a reduced FEV1/FVC ratio suggesting mild airways obstruction in contrast to normal values of MS. All patients were treated by VATS for prevention of relapsing pneumothorax. Bullectomy specimen of MS showed minor accumulation of pigmented histiocytes (smoke’s macrophages) than of CMS where we found a coarser brown to black pigment [4]. The postoperative course of MS was unremarkable while CMS had prolonged air leaks and in one case was performed pleurectomy by thoracotomy. In contrast to Beshay who showed no difference among all patients of group I, we noted several differences for clinical, radiological and histopathological evaluation and in the postoperative course of CMS compared to MS. These observations suggest that cocaine, when smoked together with marijuana, might aggravate marijuana-induced lung injury. Is it coincidence? Fligiel and colleagues reported that the effects of cocaine and marijuana on the airway appear not to be additive [5]. However the authors report histopathologic features of mucosal biopsy but do not include alveolar lung tissue and bullous disease. Probably noxious components that are a mixture of cocaine smoke plus components included in marijuana smoke might stimulate an amplified inflammation response with a dramatic increase of injurious effects on the alveolar lung. Finally in the light of this hypothesis, further studies are required to assess the effects of marijuana when smoked by itself and in conjunction with other illicit substance on lung parenchyma.

References


Reply to the Letter to the Editor

Reply to Fiorello et al.

Ralph A. Schmid*, Morris Beshay
Division of General Thoracic Surgery, University Hospital Bern, CH-3010 Bern, Switzerland

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We thank Dr Fiorello [1] and his group for the additional information on the topic [2] and we assume we are not the only thoracic centre seeing this type of lung injury. It is true that we do not exactly know what these patients are smoking and additives as lead, talcum or other substances to increase weight when selling these drugs have been reported and it is of course difficult to identify the additives retrospectively in a specific patient.

We have to pay attention to this problem but also have to ask the question, as in a recent editorial in the European Respiratory Journal: ‘Cannabis: the next villain on the lung cancer battlefield?’ [3].

References


Letter to the Editor

Surgery for isolated pleural recurrence from thymoma

Stijn Heyman*, Paul Van Schil
Department of Thoracic and Vascular Surgery, Antwerp University Hospital, Wilrijkstraat 10, 2650 Edegem, Antwerp, Belgium

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Keywords: Thymoma; Pleural; Recurrence

We read with great interest the article of Lucchi et al. [1] on surgical treatment of pleural recurrence of thymoma. As such recurrences are rare we want to add an additional case we recently encountered [2]. Recurrent myasthenia gravis was observed in a 43-year-old patient 3 years after radical thymectomy for which a partial pericardial resection had been necessary to obtain a complete resection. An isolated pleural recurrence was discovered above the left diaphragm which could be removed by video-assisted thoracic surgery.