Objectives

Reactive oxygen species modulator 1 (Romo1) is a novel protein that localizes in the mitochondrial membrane and induces mitochondrial reactive oxygen species (ROS) generation. Romo1 is increased in most cancer cell lines and is related with resistance to chemotherapy in vitro. However, data on its expression in patients with malignancy is very limited. We evaluated the usefulness of serum Romo1 as a potential diagnostic biomarker in non-small cell lung cancer (NSCLC).

Materials and methods

We initially assessed the expression of Romo1 using Western blotting and enzyme-linked immunosorbent assay in paired lung tissue and serum specimen from NSCLC patients who underwent surgical resection. Then we evaluated and compared serum Romo1 level in a healthy population ($n = 55$), patients with benign lung diseases ($n = 63$) and NSCLC patients ($n = 58$). We explored the correlation between Romo1 expression and clinical parameters and assessed
diagnostic performance of serum Romo1 for NSCLC using receiver operating characteristic (ROC) curve analysis.

Results

Romo1 expression in lung cancer tissues was significantly increased compared with non-tumorous tissues ($p < 0.001$). Romo1 expression in cancer tissues positively correlated with that in serum ($r = 0.68$, $p = 0.009$). Serum Romo1 level in NSCLC patients significantly increased compared with that of healthy population or patients with benign lung diseases (both $p < 0.001$). ROC curve analysis using an optimal cutoff value of 329.7 pg/mL revealed sensitivity and specificity for the diagnosis of NSCLC of 81.9% and 89.8%, respectively, with an area under the curve of 0.847 (95% confidence interval: 0.789–0.892, $p < 0.001$). Serum Romo1 level was not related with age, gender, smoking status, tumor differentiation, histological type or stage.

Conclusions

Serum Romo1 discriminated NSCLC patients from the population without cancer with considerable sensitivity and specificity. Serum Romo1 could be a potential diagnostic biomarker for NSCLC.
Objectives

The role of estrogen signaling in lung cancer remains unresolved. We investigate the influence of serum estrogenic compounds and estrogen receptor (ERα and ERβ) mediated bioactivity on lung cancer outcomes.

Materials and methods

Serum samples were collected from 222 postmenopausal Chinese patients diagnosed with lung cancer in five Singapore hospitals. Levels of the estrogenic
compounds estradiol and estrone were measured using liquid chromatography tandem mass spectrometry. Free estradiol levels were calculated based on sex hormone binding globulin levels. ERα- and ERβ-mediated bioactivity in serum samples were analyzed using reporter gene bioassays in human cells.

Results and conclusion

High ERβ-mediated bioactivity predicted poorer lung cancer survival ($p = 0.001$) on multivariable Cox regression analysis with adjustment for age, stage of tumor, smoking status, body mass index and histology. In comparison, levels of estrogens and ERα-mediated bioactivity were not associated with prognosis. Compared to the lowest tertile of ERβ-mediated bioactivity, patients in the middle and highest tertiles had HR (95%CI) 1.60 (1.10–2.33) and 1.93 (1.32–2.82) ($p$ for trend = 0.001) higher risk of death from lung cancer. Using Kaplan–Meier survival curves, patients with high ERβ-mediated bioactivity correlated with poorer overall survival ($p = 0.033$). ERβ-mediated bioactivity did not differ in terms of age, use of hormone replacement therapy, smoking, stage of tumor or histological subtype. High ERβ-mediated bioactivity levels in patients’ serum were associated with poorer prognosis in lung cancer patients. Our findings suggest that that compound(s) other than endogenous estrogens may be exerting this ERβ bioactivity and studies to identify these compounds or groups of compounds need to be performed. Furthermore, the measurement of ERβ activity in sera could potentially serve as a prognostic marker to predict lung cancer survival, and selective blockage of ERβ signaling may have a role in lung cancer therapy.

3. Comparison between CT tumor size and pathological tumor size in frozen section examinations of lung adenocarcinoma

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Objective

We examined the appropriate measurement for pathological tumor size by comparing radiological and pathological tumor size of resected lung adenocarcinoma in FSE.

Materials and methods

We reviewed records of 59 resected specimens of lung adenocarcinoma for FSE from January to December 2008.
Specimens were well-inflated with saline by using an injector before cutting into segments. After selecting the tumor segment of maximal diameter, we compared three ways of measuring pathological tumor size by using paired \( t \)-test: (I) macroscopic tumor size (MTS), measured with a metal straight ruler, (II) microscopic frozen section tumor size (FSTS), and (III) microscopic paraffin section tumor size (PSTS). We compared each discrepancy rate (DR) \[ DR = \frac{\text{CT tumor size} - \text{pathological tumor size}}{\text{CT tumor size}} \times 100 \] between tumors that were air-containing type and solid-density type on CT scans, and also compared the tumors with lepidic component rates (LCR) \( \geq 50\% \) and LCR \( < 50\% \), by using Mann–Whitney \( U \)-tests.

Results

FSE could diagnose malignancy with 100\% accuracy. The mean CT tumor size was 18.36 mm, and the mean pathological tumor sizes (MTS, FSTS, and PSTS) were 17.81, 14.29, and 14.23 mm, respectively. FSTS and PSTS were significantly smaller than CT tumor size \((p < 0.001)\). The DR calculated with PSTS was significantly larger in air-containing than in solid-density tumors, and also larger in LCR \( \geq 50\% \) than in LCR \( < 50\% \) tumors.

Conclusion

FSE with the inflation method diagnosed malignancy with 100\% accuracy. The lung specimen must be sufficiently inflated to prevent tissue shrinking, and we propose MTS as the definition for pathological tumor size in FSE. The greater discordance observed between CT tumor size and microscopic tumor size was assumed to be due to shrinkage of the lepidic component in the tumor.
4. Use of adjuvant chemotherapy (CT) and radiotherapy (RT) in incompletely resected (R1) early stage Non-Small Cell Lung Cancer (NSCLC)

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Background

Early stage Non-Small Cell Lung Cancer (NSCLC) is potentially curable with surgery. ESMO guidelines recommend cisplatin-based adjuvant chemotherapy (CT) for completely resected stage II–III NSCLC. There is limited evidence for the use of adjuvant CT and/or radiotherapy (RT) in incompletely resected (R1) early stage NSCLC.

Materials and methods

A European survey of thoracic oncologists was conducted to evaluate use of adjuvant CT and RT for R1-resected NSCLC and to identify factors influencing treatment decisions. Demographics and information on clinical stage, regimens, cycles planned, radiotherapy sites, multidisciplinary management and discussion about inconclusive evidence with the patient were collected. Univariate and multivariate analyses were performed.

Results

768 surveys were collected from 41 European countries. 82.9% of participants were medical oncologists; 49.3% ESMO members; 37.1% based in University Hospitals; 32.6% practicing oncology for over 15 years and 81.4% active in research. 91.4% of participants prescribed adjuvant CT and mostly cisplatin/vinorelbine (81.2%) or cisplatin/gemcitabine (42.9%). 85% discussed limited clinical evidence with the patient. In the univariate analysis, a statistically significant association with CT prescription was found for medical oncology specialty ($p < 0.001$), ESMO membership ($p < 0.001$), activity in clinical research ($p = 0.002$) and increased frequency of ESMO guidelines consultation ($p$ for trend $<0.001$). 48.3% of participants prescribed adjuvant RT and its prescription was associated with radiation oncology specialty ($p < 0.001$), not being an ESMO
member ($p < 0.001$), years practicing specialty ($p$ for trend $= 0.001$), workload of lung cancer patients ($p$ for trend $= 0.027$) and decreased frequency in consulting ESMO guidelines ($p < 0.001$). In the multivariate analysis, medical oncology and radiation oncology were the best discriminator for prescription of adjuvant CT and RT, respectively.

Conclusion
This survey demonstrates that adjuvant CT and RT are commonly used in clinical practice for R1-resected NSCLC despite limited evidence. Prospective trials are necessary to clarify optimal management in this setting.

Objectives
Although the National Lung Screening Trial (NLST) lauds the efficacy of low-dose computed tomography (LDCT) at reducing lung cancer mortality, it has not been widely used for population-based screening. By examining the availability of U.S. LDCT screening centers, and underlying rates of lung cancer incidence, mortality, and smoking prevalence, the need for additional centers may be determined.

Materials and methods
Locations of 203 LDCT screening centers from the Lung Cancer Alliance Screening Centers of Excellence database, a list of active NLST and International Early Lung and Cardiac Action Program (I-ELCAP) screening centers, and an independently conducted survey of Society of Thoracic Radiology members were geocoded and mapped. County-level rates of lung cancer incidence, mortality, and smoking prevalence were also mapped and overlaid with the locations of the 203 LDCT screening centers.

Results and conclusions

Results showed the majority of LDCT screening centers were located in the counties with the highest quartiles of lung cancer incidence and mortality in the Northeast and East North Central states, but several high-risk states had no or few identified screening centers including Oklahoma, Nevada, Mississippi, and Arkansas. As guidelines are implemented and reimbursement for LDCT screening follows, equitable access to LDCT screening centers will become increasingly important, particularly in regions with high rates of lung cancer incidence and smoking prevalence.

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