— F-273 —

Small Cell Lung Cancer Associated With Ectopic ACTH Syndrome

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About 20% of small cell lung cancer (SCLC) is associated with paraneoplastic syndrome. This is mostly caused by neoplastic cells secreting ectopic hormones. It has been known that ectopic ACTH syndrome is associated with about 5% of small cell carcinoma. SCLC associated with ectopic ACTH syndrome can be suspected when hypokalemia and metabolic alkalosis are present. This is also charaterized by generalized edema, hypertension, generalized weakness, muscle atrophy and glucose intolerance. According to recent reports, they the prognosis of median survival may be less than 4 months. We report here on a case of SCLC associated ectopic ACTH syndrome. A 46-year-old man came to our hospital presented with a cough, sputum and right chest wall pain which had persisted 2 months. A lung mass on the right upper lobe, multiple liver metastasis and metastasis in both adrenal glands is were also noted on chest CT scan. Bronchoscopic biopsy revealed SCLC. His blood test showed that K+ level was 2.3 mEq/L, glucose level was 389 mg/dL. ABGA showed that PH 7.5, PaCO2 51mmHg, PaO2 99mmHg, HCO3- 46mEq/L, O2saturation 98%. The ACTH level was over 1590 pg/dl, serum cortisol was 68.2 ug/dL, 24hr urine cortisol 9650 ug/day, cortisol 79.4 ug/dL on an overnight dexamethasone suppression test suggesting ectopic ACTH syndrome. He was diagnosed as SCLC associated with ectopic ACTH syndrome. He was treated with etoposide and cisplatin for 1st line chemotherapy. After one cycle of chemotherapy, pneumonia developed and progressed to lung abscess. After treating the lung abscess, he was treated with irrinotecan and cisplatin. However hypokalemia was not controlled even after chemotherapy for SCLC. He died of aspiration pneumonia after the 2nd cycle of chemotherapy, 2 months after diagnosis.

— F-274 —

Correlation of CD44, VEGF-C and COX-2 with Clinicopathologic Parameters and Clinical Outcomes: Non-Small Cell Lung Cancer

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Background : Recent studies suggest that CD44-ligation induces expression of vascular endothelial growth factor-C (VEGF-C) expression and that the induction of the cyclooxygenase (COX-2) is achieved through an upregulation of VEGF-C. It is postulated that individual role of CD44, VEGF-C and COX-2 involves cellular proliferation, angiogenesis and metastasis of cancer. We investigated the correlation of CD44, VEGF-C and COX-2 with clinicopathologic parameters and clinical outcomes in surgically resected NSCLC patients. **Methods :** Using immunohistochemial staining, we analyzed the protein expressions of CD44s, CD44v6, COX-2 and VEGF-C on the tissue array specimens from 180 patients (adenocarcinoma (AC), n=90; squamous cell carcinoma (SCC), n=90) with completely resected NSCLC patients. **Results :** The median age was 64 (range, 19-88) and M:F ratio was 141:39. According to pathologic stage by AJCC, stage I, II, III was 101 (56.1%), 35 (19.4%) and 44 (24.4%). The expressions of CD44s, CD44v6, COX-2 and VEGF-C were observed in 65.7%, 37.6%, 40% and 60%, respectively. COX-2 overexpression was found in 51.1% of AC subtype and 28.9 % of SCC with statistically significant difference. The expressions of both CD44s and CD44v6 were found to be more frequent in SCC with significance, compared to those of AC (44.6% vs. 26.2%, $p<0.001$; 6.6% vs. 34.1%, $p<0.001$). Overexpression of VEGF-C was correlated with CD44 and COX-2 expression. It also had strong correlation with tumor size and differentiation ($p=0.026$, $p=0.004$). In the multivariate analysis, only lymph node metastasis was an independent prognostic factor (HR 2.367, $p<0.001$), but none of these molecules showed statistical correlations. **Conclusions :** To be taken, the present study revealed that VEGF-C, correlated with CD44 and COX-2, was a strong predictive marker for tumor size in early stage of NSCLC.