



## PARANEOPLASTIC EOSINOPHILIA IN LUNG CANCER

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**ABSTRACT**

Diverse medical conditions, including allergic disorders, parasitic and fungal infections, vasculitis and drug reactions, as well as hematologic and non-hematologic malignancies can be associated with eosinophilia.<sup>1</sup> We describe a case of metastatic poorly differentiated squamous cell lung cancer and a case of metastatic small cell lung cancer complicated by eosinophilia.

**KEY WORDS :** Hypereosinophilia, TBNB , IHC**INTRODUCTION**

Hypereosinophilia is a rare finding in the course of a malignant disease in general and lung cancer in particular. Very seldom it is the first sign of malignancy; and when it occurs it is often associated with disseminated metastatic end stage disease and a poor prognosis.<sup>2</sup> We report the occurrence of highly elevated numbers of eosinophils in the peripheral blood of a 32-year-old lady and a 65-year-old man. The diagnostic work-up revealed the presence of a non-small-cell lung carcinoma in the former and a small cell lung carcinoma in the latter; a disease state rarely reported to have hypereosinophilia.<sup>3</sup>

**Case 1**

A 32 year old female presented with complaints of shortness of breath and cough for 1 month. Shortness of breath was of insidious onset progressive from grade II to grade III MMRC, cough with expectoration- mucoid and scanty, not blood stained. Loss of weight and appetite present. No h/o fever, chest pain or palpitation. General examination was normal. Respiratory system – trachea central, absent movements on the right. Stony dullness to percussion and absent breath sounds in right hemithorax.

Routine labs showed a total leukocyte count of 17100 with eosinophils of 40.8 %. Other blood routines were within normal limits.

Chest x-ray showed right massive pleural effusion, trachea central. Diagnostic and therapeutic thoracentesis was done. Pleural fluid analysis showed ADA of 10.8, LDH 19017, culture showed no growth, cell block cytology negative for malignant cells. PET CT showed FDG avid (SUV max 11.20) mass seen in the upper and mid lobe of right lung (72x76x55 mm), large right pleural effusion, enlarged mediastinal lymph nodes (T4N3M1c).

Patient underwent bronchoscopy for tissue diagnosis- findings were nodules over medial wall of left main bronchus, right intermediate bronchus was narrowed, bled on touch and scope could not be negotiated distally. TBNB & core biopsy was taken from subcarinal station and endobronchial biopsies were taken from nodules in the left main bronchus. TBNB and nodule biopsy histopathology and IHC was suggestive of metastatic moderate to poorly differentiated squamous cell carcinoma, focally positive vimentin and positive p63. TTF1 and CD56 negative.

Patient also underwent right intercostal drain insertion and was planned for thoracoscopy and pleurodesis. Her condition however worsened rapidly and she developed progressive dyspnea. She required intensive care and non-invasive ventilation. Acute cardiac

decompensation was ruled out. The family was informed that no curative measures were possible due to stage IV disease. Comfort care was initiated. They opted for the same at home. She was discharged with the ICD insitu, on home bilevel positive airway pressure and she succumbed within a few days of discharge.

**Case 2**

A 65 year old male presented with complaints of shortness of breath for 2 months, cough for 10 days. Shortness of breath was insidious in onset, progressive from grade II to grade III MMRC, cough with expectoration- mucoid and scanty, occasionally blood stained. Loss of weight and appetite present. He was a known case of systemic hypertension on treatment. Was also a chronic smoker (40 pack years)- quit 5 years back. General examination was normal. Respiratory system – trachea central, decreased movements on the right. Resonant to percussion and decreased intensity of breath sounds in right hemithorax.

Routine labs showed a total count of 10400 with eosinophils of 14.7 %. Other blood routines were within normal limits.

Chest x-ray showed a right hilar mass. CT thorax showed a homogeneously enhancing mass lesion in right upper and middle lobe with spiculated margins involving perihilar region measuring 7.3x7.8x7.6 cm medially infiltrating the mediastinum with superior vena cava appearing severely compressed due to mass effect- 180 degree encasement, complete encasement of right pulmonary artery, right upper lobe pulmonary vein and right main bronchus.

Radiation oncology opinion sought for suspected superior vena cava syndrome and need for emergent radiation but was deferred as patient had no symptoms of superior vena cava obstruction. Patient underwent bronchoscopy and endobronchial biopsy from growth. Histopathology and IHC were in favour of small cell carcinoma. CD 56, TTF1, INSM-1 positive. MRI brain showed no evidence of space occupying lesion. Patient was then referred to medical oncology for palliative chemotherapy.

**DISCUSSION**

Peripheral eosinophilia associated with a solid malignancy is rare, although it has been described; with lung carcinoma being the most rare. The pathogenesis of paraneoplastic eosinophilia is unclear. Numerous mechanisms have been postulated and bone marrow stimulation by cytokines secreted by tumor tissues, including granulocyte macrophage-colony stimulating factor (GM-CSF), G-CSF, IL-3 and IL-5, is most acknowledged.<sup>4</sup> IL-5 has emerged as the main controlling cytokine for eosinophilia production, activation, and recruitment.<sup>5</sup> In our first case IL-5 assay was not done since the family

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refused further intervention and treatment due to the poor prognosis.

Patients with paraneoplastic eosinophilia are typically asymptomatic. However, in a number of cases, a markedly elevated eosinophil count may be associated with shortness of breath and wheezing. In this case, the patient developed rapidly progressive dyspnea and hypoxia. It is generally considered as a poor prognostic feature reflecting extensive disease and dissemination. Normally, tumor removal and anticancer therapies also resolve the eosinophilia. However, case reports suggest that this is not always feasible, as these patients usually have extensive metastases and poor outcomes.<sup>6</sup>

Primary eosinophilic syndromes are managed successfully with corticosteroid therapy. However, a number of patients are non-responsive to corticosteroids, but respond well to hydroxyurea. Hydroxyurea is also reported to be an effective first-line agent in hypereosinophilic syndrome. A combination of hydroxyurea and corticosteroid increases the response rate. The significant effect of corticosteroid and hydroxyurea in reducing the eosinophil count may play a role in improving and stabilizing paraneoplastic eosinophilia and act as a bridge to more anticancer therapies. It is suggested that lung cancer patients who present with abnormally high counts of eosinophils, should receive a combination of corticosteroids, hydroxyurea and anticancer drugs to prevent the development of aggressive and life-threatening eosinophilia, even if they are asymptomatic initially.<sup>7</sup>

## CONCLUSION

In conclusion, this case series reports a young female with poorly differentiated squamous cell lung cancer who developed respiratory insufficiency as symptoms of paraneoplastic eosinophilia and an incidentally detected eosinophilia in a middle aged male with small cell lung cancer. While the incidence, pathogenesis and effect on outcome of eosinophilia in non-small-cell lung cancer has already been established, the occurrence or the probable pathogenesis of eosinophilia in small cell lung cancer is neither defined nor reported in literature. Larger studies may be required to establish a definite association between the latter.

## REFERENCES

1. Kayar Y, Baysal B, Kayar NB, Kyio NH, Mahdi NM, et al. (2015) Hypereosinophilia Induced by Lung Adenocarcinoma: A Rare Case. *J Carcinog Mutagen* 6: 216. doi:10.4172/2157-2518.1000216
2. Abughanimeh O, Tahboub M, Abu Ghanimeh M. Metastatic Lung Adenocarcinoma Presenting with Hypereosinophilia. *Cureus*. 2018;10(6):e2866. Published 2018 Jun 22. doi:10.7759/cureus.2866
3. AS Verstraeten, A De Weerd, G van Den Eynden, E Van Marck, A Snoeckx & PG Jorens (2011) EXCESSIVE EOSINOPHILIA AS PARANEOPLASTIC SYNDROME IN A PATIENT WITH NON-SMALL-CELL LUNG CARCINOMA: A CASE REPORT AND REVIEW OF THE LITERATURE, *Acta Clinica Belgica*, 66:4, 293-297
4. El-Osta H, El-Haddad P, Nabbout N. Lung carcinoma associated with excessive eosinophilia. *J Clin Oncol*. 2008 Jul 10;26(20):3456-7. doi: 10.1200/JCO.2007.15.8899. PMID: 18612162.
5. Han X. The eosinophilic infiltration of lung cancer tissue and its relationship with the expression of IL-5. *European Respiratory Journal*. 2011;38(55):2794.
6. Venkatesan, Rohit & Salam, Amir & Alawin, Issam & Willis, Maurice. (2015). Non-small cell lung Cancer and elevated eosinophil count: A case report and literature review. *Cancer Treatment Communications*. 19. 10.1016/j.ctrc.2015.05.002.
7. Lo CH, Jen YM, Tsai WC, Chung PY, Kao WY. Rapidly evolving asymptomatic eosinophilia in a patient with lung adenocarcinoma causes cognitive disturbance and respiratory insufficiency: Case report. *Oncol Lett*. 2013;5(2):495-498. doi:10.3892/ol.2012.1020