

Decrease in Visual Acuity Revealing Lung Cancer: Case Report

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Abstract

Case Report

Cancers, especially bronchopulmonary ones, can sometimes be accompanied by paraneoplastic syndrome. These syndromes may appear during a known neoplasia or even be the mode of revelation. Among these manifestations, ophthalmic manifestations, especially retinal, are noted. We report the case of a 71-year-old patient with small cell lung cancer revealed by a decrease in visual acuity.

Keywords: Paraneoplastic, CAR, Lung Cancer.

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INTRODUCTION

Paraneoplastic ophthalmies are rare ocular manifestations, the most common being Cancer-Associated Retinopathy (CAR), estimated at approximately 0.01% of cancers [1, 2], which can precede or accompany a malignant tumor pathology, without direct invasion by the cancerous pathology or its metastases. They are secondary to the production by the tumor of circulating pathogenic peptide substances or immune dysregulation [3]. Bronchogenic carcinomas, especially small cell ones, are most often responsible for these paraneoplastic syndromes compared to gynecological or thymic cancers [4].

CASE REPORT

We report the case of a 71-year-old patient with a history of chronic smoking at a rate of 40 pack-years, who quit smoking 10 years ago. He also has hypertension treated with medication. The patient presented with a rapidly progressive decrease in visual acuity over 15

days, estimated at 4/10 OD and 6/10 OS. Ophthalmological examination was strictly normal. Brain MRI and Angio MRI were performed, revealing no abnormalities.

Paraneoplastic retinopathy was suspected due to the absence of obvious lesions explaining this sudden decrease in visual acuity. A thoraco-abdominal-pelvic CT scan was performed, revealing a well-defined oval mass in the left lower lobe with discreetly spiculated margins measuring 51mm x 41mm, along with mediastinal lymphadenopathy measuring 50mm x 20mm and 29mm x 20mm (Figure 1). A CT-guided transparietal biopsy was performed, and the histopathological study confirmed a diagnosis of small cell lung carcinoma.

The diagnosis of CAR secondary to lung cancer was established, and the patient received corticosteroid boluses with gradual tapering, as well as radio chemotherapy for cancer, resulting in slight improvement in visual acuity.

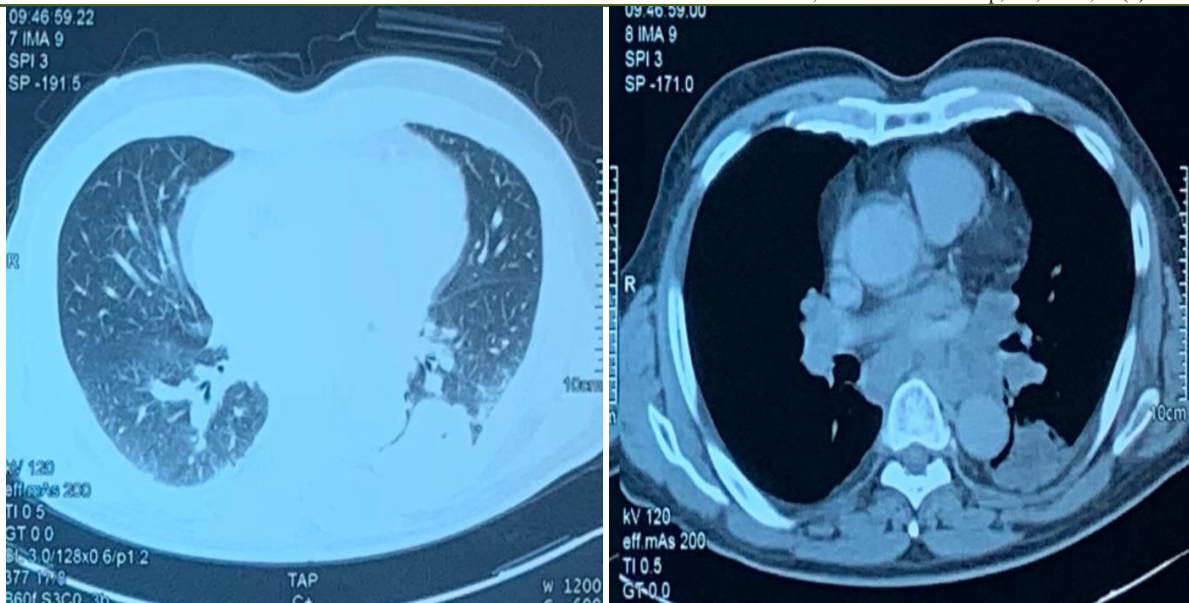


Figure 1: Left Lower Lobe Mass on CT Scan

DISCUSSION

CAR (Cancer-Associated Retinopathy) is the most common paraneoplastic retinopathy. The cancers responsible for CAR are numerous, with the most common being pulmonary, breast, ovarian, and uterine cancers [1, 5, 6].

It is an autoimmune paraneoplastic disease affecting the photoreceptors. Consequently, the symptomatology will include, if cones are affected, decreased visual acuity, color vision disturbances, photopsia, and photophobia. When rods are affected, hemeralopia will be observed with difficulties adapting to darkness. Most of the time, the ophthalmological clinical examination is normal, although it may sometimes reveal signs of vasculitis, vitritis, or macular edema [5]. Rarely, signs of retinitis pigmentosa may develop, with thin vessels and scattered pigmentary migrations [7]. Electroretinogram most often reveals overall retinal dysfunction.

It is possible to request measurement of anti-retinal antibodies, understanding its limitations: firstly, there are many false positives, especially in other general or ocular pathologies (including cataracts...), and secondly, only 60% of patients will have positive anti-retinal antibodies, and their negativity does not exclude the diagnosis. The most well-known antigen is recoverin [8], a protein aiding in rhodopsin phosphorylation and whose role in phototransduction is essential: anti-recoverin antibodies are positive in 10% of cases. Its pathogenic role is demonstrated: studies have shown destruction of photoreceptors and bipolar cells in rats by intravitreal injection of anti-recoverin antibodies [9]. Another auto-antibody is directed against α -enolase, present on photoreceptors, ganglion cells, and Müller cells: it is positive in one-third of cases. There are many other autoantibodies, whose role is less clear (anti-

transducin- α antibodies, anti-carbonic anhydrase II or CAII, anti-tubby-like protein 1 or TULP1, anti-arrestin, anti-heat shock cognate 70 or HSC70, anti-nuclear receptor specific to photoreceptors or PNR...).

Due to this antigenic complexity, CAR encompasses different phenotypes, of which at least three are well known: CAR with anti-recoverin antibodies affect both cones and rods and progress more rapidly, with a worse prognosis. Those with anti- α -enolase antibodies are rather responsible for a more slowly progressive involvement, affecting only cones and often manifesting years after the diagnosis of a known cancer. Those with anti-transducin antibodies affect rods more and progress very slowly [10, 11].

In CAR, cancer treatment rarely improves visual function [12]. Therefore, it is appropriate to use long-term immunomodulatory or immunosuppressive treatments (corticosteroids, plasma exchanges, polyvalent immunoglobulins, azathioprine, mycophenolate mofetil, rituximab...). Teams have effectively supplemented general immunosuppressive treatment with intravitreal injections of dexamethasone or triamcinolone [13], allowing more effective reduction of inflammation directly in contact with photoreceptors. Intravitreal injections of anti-VEGF are discussed in this indication since they would combat VEGF potentially secreted by the tumor mass and could possibly decrease the permeability of the hemoretinal barrier to pathogenic antibodies [14].

CONCLUSION

Bronchopulmonary cancers, especially small cell carcinomas, can initially manifest with paraneoplastic syndromes, notably ocular, sometimes preceding their discovery. This is why it is important to recognize these manifestations and conduct thorough

investigations to determine the cause for possible early treatment.

REFERENCES

- Darnell, R. B., Darnell, R., & Posner, J. B. (2011). *Paraneoplastic Syndromes*. Oxford University Press, USA.
- Darnell, R. B., & Posner, J. B. (2003). Paraneoplastic Syndromes Involving the Nervous System. *N Engl J Med*, 349(16), 1543-1554. doi: 10.1056/NEJMra023009.
- Alabduljalil, T., & Behbehani, R. (2007). Paraneoplastic syndromes in neuro-ophthalmology. *Curr Opin Ophthalmol*, 18(6), 463-469. doi: 10.1097/ICU.0b013e3282f0b497.
- Adamus G. (2009). Autoantibody targets and their cancer relationship in the pathogenicity of paraneoplastic retinopathy. *Autoimmun Rev*, 8, 410-414.
- Rahimyet, E., & Sarraf, D. (2013). Paraneoplastic and non-paraneoplastic retinopathy and optic neuropathy: evaluation and management. *Surv Ophthalmol*, 58(5), 430-458. doi: 10.1016/j.survophthal.2012.09.001.
- Kim, S. J., Toma, H. S., Thirkill, C. E., & Dunn Jr, J. P. (2010). Cancer-associated retinopathy with retinal periphlebitis in a patient with ovarian cancer. *Ocular Immunology and Inflammation*, 18(2), 107-109. doi: 10.3109/09273940903457441.
- Heckenlively, J. R., & Ferreyra, H. A. (2008, April). Autoimmune retinopathy: a review and summary. In *Seminars in immunopathology* (Vol. 30, pp. 127-134). Springer-Verlag. doi: 10.1007/s00281-008-0114-7.
- Whitcup, S. M., Vistica, B. P., Milam, A. H., Nussenblatt, R. B., & Gery, I. (1998). Recoverin-associated retinopathy: a clinically and immunologically distinctive disease. *Am J Ophthalmol*, 126(2), 230-237. doi: 10.1016/S0002-9394(98)00149-4.
- Adamus, G., Machnicki, M., Elerding, H., Sugden, B., Blocker, Y. S., & Fox, D. A. (1998). Antibodies to Recoverin Induce Apoptosis of Photoreceptor and Bipolar Cells in vivo. *J Autoimmun*, 11(5), 523-533. doi: 10.1006/jaut.1998.0221.
- Adamus, G. (2011). Diversity in autoimmunity against retinal, neuronal, and axonal antigens in acquired neuro-retinopathy. *J Ophthalmic Inflamm Infect*, 1(3), 111-121.
- Weleber, R. G., Watzke, R. C., Shults, W. T., Trzuppek, K. M., Heckenlively, J. R., Egan, R. A., & Adamus, G. (2005). Clinical and electrophysiologic characterization of paraneoplastic and autoimmune retinopathies associated with anti-nucleolar antibodies. *American journal of ophthalmology*, 139(5), 780-794. doi: 10.1016/j.ajo.2004.12.104.
- Maeda, T., Maeda, A., Maruyama, I., Ogawa, K. I., Kuroki, Y., Sahara, H., ... & Ohguro, H. (2001). Mechanisms of photoreceptor cell death in cancer-associated retinopathy. *Investigative ophthalmology & visual science*, 42(3), 705-712.
- Huynh, N., Shildkrot, Y., Lobo, A. M., & Sobrin, L. (2012). Intravitreal triamcinolone for cancer-associated retinopathy refractory to systemic therapy. *Journal of ophthalmic inflammation and infection*, 2, 169-171. doi: 10.1007/s12348-012-0067-9.
- Cao, R., Xue, Y., Hedlund, E. M., Zhong, Z., Tritsarlis, K., Tondelli, B., ... & Cao, Y. (2010). VEGFR1-mediated pericyte ablation links VEGF and PlGF to cancer-associated retinopathy. *Proceedings of the National Academy of Sciences*, 107(2), 856-861. doi: 10.1073/pnas.0911661107.