

Figure 2: Intraoperative photograph of the calcified amorphous tumor

The second one has the same size, but it was implanted next to the stoma of the superior vena cava. Both masses were removed, taking the endocardium on their implantation bases.

Furthermore, there was a small mass in the right ventricle measuring 4mmx3mmx2mm implanted behind the anterior leaflet of the tricuspid valve and a second small mass implanted on the side and front wall of the pulmonary artery; It continuous with an extension which completely blocks the left pulmonary artery and rushes into 1cm (Figure 3). A total removal of this mass was performed after dissection from its attachments.

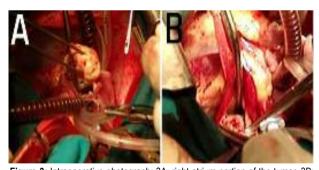


Figure 3: Intraoperative photograph: 3A: right atrium portion of the tumor. 3B: The tumor blocking the pulmonary artery.

The patient presented at third postoperative day for fever with a diagnosis of right pneumonia. Under antibiotic treatment there was a good clinical and radiological evolution.

On the basis of the pathological examination, the cardiac mass was demonstrated with calcified amorphous tumor of the heart.

Echocardiography revealed that there was an expansion of the right atrium and increase of the severity of the

tricuspid regurgitation. After six months, tricuspid regurgitation became severe (grade 3-4) and the right cavities are always dilated. The patient was doing well with no evidence of the recurrence of the syncope 48 months after operation.

References

- Suh JH, Kwon JB, Park K, et al. Calcified amorphous tumor in left atrium presenting with cerebral infarction. Journal of Thoracic Disease. 2014;6(9):1311-4.
- Vlasseros I, Katsi V, Tousoulis D, et al. Visual loss due to cardiac calcified amorphous tumor: a case report and brief review of the literature. International journal of cardiology. 2011;152(3):e56-7.

Central serous chorioretinopathy after nasal corticosteroids in the aviator

Chorioretinite séreuse centrale après corticothérapie nasale chez le pilote

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Central serous chorioretinopathy (CSC) is a frequent unilateral maculopathy in young adults, characterized by an idiopathic retinal detachment of the neuroepithelium in the macular region. It is a multifactorial disease whose pathogenesis is still poorly understood [1]. Endogenous and exogenous glucocorticoids have been frequently implicated in the pathogeny of the CSC[1].

In the aviation medicine, where the nasal corticosteroids are commonly prescribed, CSC takes a particular importance and severity due to its negative impact on visual function of the pilot and flight status.

We report the case of two pilots who developed CSC after the use of nasal steroids.

Observation N°1:

A 40 year- old male, airline pilot, consulted for a sudden decrease in visual acuity in the right eye and metamorphopsia lasting for four days.

The patient was non alcoholic, non-smoker, non-hypertensive, with no notable medical history. Questioning revealed the use of nasal corticosteroid for sinusitis 10 days before the onset of the symptoms.

The visual acuity was 4/10 in the right eye and 10/10 in the left eye. The examination of the anterior segment was normal in both eyes. The glare test was disturbed as the test of the stereoscopic vision. Color vision was normal. Fundus examination detected a bleb of serous retinal detachment macular at the right eye. Fundus fluorescein angiography (FFA) showed macular leakage point (Photo $N^{\circ}1$).

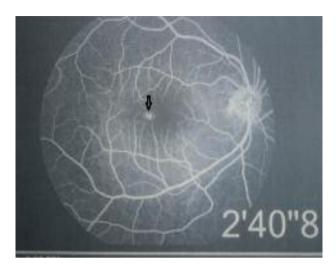


Figure 1: fundus fluorescein angiography showing macular leakage point in the right eye.

Coherence tomography (OCT) confirmed the diagnosis of CSC (Photo N°2). The dosage of serum and urinary cortisol was normal.

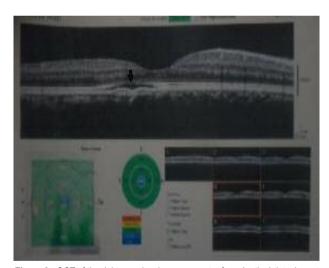


Figure 2 : OCT of the right eye showing serous retro foveal retinal detachment

The pilot was temporarily unfit and was treated by spironolactone (25 mg per day for 3 months) with eviction of all steroids and followed up every two weeks.

The evolution was marked by a complete recovery of the visual acuity in the right eye, improvement of functional signs and normalization of stereopsis and glare test within six weeks. The optical coherence tomography (OCT) showed a total recovery of the serous retinal detachment bleb.

One month later, the pilot has developed a recurrence of

CSC in the same eye. He was prescribed inhibitor of carbonic anhydrase (acetazolamide 250 mg twice per day during fifteen 15 days, then one tablet daily for 15 days). The pilot was temporarily unfit and was removed from flight status for three months. He resumed his flight status after the normalization of visual function and total disappearance of functional complaints.

Observation N°2:

A 43 year-old man, a helicopter pilot with no particular medical history, consulted for sudden decrease in visual acuity in the right eye with metamorphopsia especially during night mission, with onset a week prior his presentation.

Questioning revealed a nasal corticosteroid treatment one week before and a high level of environmental stress.

The visual acuity was 8/10 in the right eye and 10/10 in the left eye.

The examination of the anterior segment was normal in both eyes. Color vision was normal.

The glare test and test of stereopsis were disturbed.

The fundus examination showed a right macular bleb. A point of extra foveal macular leak was objectified by fundus fluorescein angiography (Photo N°3). OCT confirmed the diagnosis of CSC (Photo N°4).

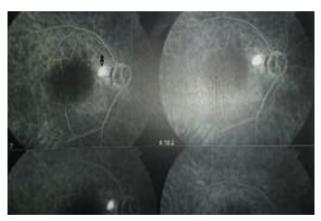


Figure 3: fundus fluorescein angiography showing point of extra foveal macular leak

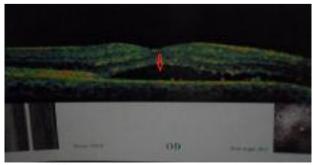


Figure 4 : OCT showing retrofoveolar serous retinal detachment

The pilot was declared unfit temporary. The evolution was marked by spontaneous remission without treatment after four months and by the occurrence of two recurrences of CSC a year apart. First recurrence lasted nine months and the second lasted seven months. After all, the pilot recovered normal visual function.

Conclusion

The CSC is an idiopathic maculopathy. Symptoms include sudden blurred vision, functional macular syndrome. The disease usually regresses spontaneously within three to four months, however with a risk of recurrence. The most common initial approach to treatment is observation [1]. Recently, spironolactone, aldosterone antagonist is used in the treatment of the CSC [2]. The use of Glucocorticoids in any form (oral, intravenous, subcutaneous, epidural and intra-articular) was involved in the development or worsening of the CSC [3,4,5,6]. The link between the use of intranasal corticosteroids and CSC was also reported by several authors [7-9].

The CSC is a common disease in the flight [10]. The particular operational stress among military pilots especially in anxious nature promotes its development. CSC affects, to varying degrees, various parameters of pilot visual function. The resumption of flights should only be decided after the normalization of these parameters, the reapplication of the neurosensory retina and the disappearance of functional macular syndrome.

Place of nasal corticosteroids in the development of the CSC has some particularities in the flight. In fact, in current practice, aerospace ENT is frequently confronted with acute inflammatory condition type inflammatory rhinitis associated with tubal dyspermeability, causing possible otological barotrauma in the pilot. In these cases, the use of inhaled corticosteroids provides, due to their anti-inflammatory action, fast sinonasal and tubal decongestion by reducing significantly the catarrhal edema.

Although the contribution of nasal corticosteroids are efficient in aviation ENT therapy, the risk of eye complications such CSC motivate avoiding them among pilots at risk of developing this disease. It is advisable that pilots, undergoing steroid treatment in any form, be alerted to the risk of sudden visual impairment.

References

- Salehi M, Wenick AS, Law HA, Evans JR, Gehlbach P. Interventions for central serous chorioretinopathy: a network metaanalysis.
 - Cochrane Database of Systematic Reviews 2015, Issue 12. Art. No.: CD011841. DOI: 10.1002/14651858.CD011841.pub2.
- Evaluation de la Spironolactone dans le traitement des choriorétinites séreuses centrales non résolutives à trois mois. Essai clinique 2016. Agence française de sécurité sanitaire des produits de santé (Saint-Denis)
- Jin-Liang L, Shu-Lye a. Can Long-Term Corticosteroids Lead to Blindness? A Case Series of Central Serous Chorioretinopathy Induced by Corticosteroids. Ann Acad Med Singapore 2006: 35: 496-9.
- Carvalho-Recchia Ca, Yanuzzi La, Negrao S, Spaide Rf, Freund Kb, Rodriguez-Coleman H. Corticosteroids and Central Serous Chorioretinopathy. Ophhalmology 2002: 109: 1834-7
- 5. Bevis T, Ratnakaram R, Smith Mf, Bhatti Mt. Visual Loss Due to Central Serous

- Chorioretinopathy During Corticosteroid Treatment for Giant Cell Arteritis. Clin Experiment Ophthalmol 2005: 33: 437-9.
- Levy J, Marcus M, Belfair N, Klemperer I, Lifshitz T. Central Serous Chorioretinopathy in Patients Receiving Systemic Corticosteroid Therapy. Can J Ophthalmol 2005; 40: 217-21
- Gunjan Prakash, Jain Shephali, Nath Tirupati, Pandey D Ji. Recurrent Central Serous Chorioretinopathy with Dexamethasone Eye Drop Used Nasally for Rhinitis. Middle East Afr J Ophthalmol 2013; 20(4): 363-365.
- Haimovici R, Gragoudas Es, Duker Js, Sjaarda Rn, Eliott D. Central Serous Chorioretinopathy Associated with Inhaled or Intranasal Corticosteroids. Ophthalmology 1997; 104: 1653-60.
- Kleinberg Aj, Patel C, Lierberman Rm, Malnik Bd. Bilateral Central Serous Chorioretinopathy Caused by Intranasal Corticosteroids: A Case Report and Review of the Literature. Laryngoscope 2011; 121: 2034-7.
- Corbé C, Lebail B. Appareil Visuel Et Aviation. Encycl Méd Chir (elsevier, Paris), Ophtalmologie, 21-810-a-05, 1996, 8p.

Un cas de Trisomie 9 Complète avec association phénotypique inhabituelle

Complete Trisomy 9 with unusual phenotypic associations

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La trisomie 9 complète et homogène est une anomalie chromosomique rare (moins de 50 cas décrits) (1). Elle conduit dans près de 80% des cas à un arrêt spontané de la grossesse ne dépassant pas les 22-24 SA et dans moins de 20% des cas à un décès quelques heures après la naissance (2). Elle est à l'origine d'un syndrome malformatif associant une dysmorphie faciale, des malformations du système nerveux central, des anomalies cardiaques, de l'appareil génito-urinaire et des malformations ostéo-articulaires (1-3). Nous présentons, un cas d'un fœtus porteur d'un syndrome polymalformatif découvert à l'échographie prénatale.

Observation

Il s'agit d'une patiente âgée de 41 ans, aux antécédents d'une première grossesse arrêtée (au premier trimestre) et d'une deuxième menée à terme (enfant vivant en bonne santé). La grossesse actuelle était mal suivie (mesure de la clarté nucale et étude des marqueurs sériques maternels non réalisées). La première