Persistent Visual Noise (Visual Snow Syndrome)

Introduction

Visual Snow Syndrome is a disorder of altered visual perception in which the patients see continuous flickering tiny black and white dots across the entire visual field of both eyes similar to the pixels of an old television.

The visual noise occurs 24/7 with eyes open and closed. Visual Snow is a part of unique syndrome that is different from visual aura in migraine. It was diagnosed for the first time in 1995 by Dr. Schankin MD Fellow in the department of neurology, University of California, San Francisco. Patients may describe other visual symptoms like floaters, afterimages, flashes in addition to headache, tinnitus, anxiety or depression. Most of the affected patients are young and otherwise healthy, often in the second to the fourth decade of life. The cause of syndrome is unclear [1].

The supposed mechanism is excessive activity or excitability of the cerebral cortex neurons that including the thalamic reticular nucleus, Parietal lobe and prefrontal lobe. There is no cure for this syndrome until now [2-4].

Method

26 y old female, has attended our clinic, complaining from persistent noise in her vision (day and night 24/7) that described as black & white dots in the entire field of her vision.

Also she reported difficulties in night vision in addition to other non-ophthalmic symptoms like headache, tinnitus, loss of appetite and pain in tempero-mandibular joint (Figures 1 and 2).

The patient’s past medical history & drug history were negative.

Full ophthalmic examination was done for anterior and posterior segments of the both eyes. In addition to performing swept source OCT (SS-OCT) imaging to the retina and the optic nerve and Visual field test (Humphrey 24-2).

The patient was referred to neurologist to exclude migraine aura or any other neurologically associated condition.

The neurologist report was normal with no any clear link between the patient complain and migraine with normal MRI Neuroimaging and normal EEG as shown below in (Figure 3).

FDG PET/CT scan findings: There is evidence of asymmetrical temporal lobe metabolism which appears to be slightly reduced on the left compared to the right, yet within acceptable limits for age and with no locality.

Normal metabolic activity within both parietal lobes.

Normal metabolic activity within both occipitoparietal regions, and occipital lobes with no evidence of any hyper or hypometabolic regions (Figure 4).

Discussion and Conclusion

“Visual Snow” is a life disabling disorder with patients complaining of continuous flickering dots (TV Noise) in the entire visual field [1]. The ocular and neurological
Figure 1(A): Shows the visual noise that seen by the patient of visual snow syndrome. (B): Explains the dark adaptation problems in those patients.

Figure 2(A): showed the ophthalmic examination that was normal with 6/6 vision, normal color vision on isihara test & the Humphry visual fields that were normal for both eyes. (B): SS-OCT image of the optic nerve head that showed a normal peripapillary nerve fiber layer (NFL) and normal ganglion cell layer (GCL) for the both eyes. (C): SS-OCT scan for the foveal area that showed bilateral normal foveal contour with intact photoreceptors layer (intact IS-OS junction) in both eyes.
exams and neuroimaging studies are unremarkable. The suggested pathophysiology is chemical imbalance between glutamic acid & gamma amino butyric acid (GABA) in the high visual centers of thalamus, parietal & prefrontal lobe [4]. It is believed that deficiency of (GABA), the most significant inhibitory neurotransmitters of the CNS is the trigger for high center over activity in visual snow syndrome.

The neuroimaging studies of the visual snow syndrome cases showed a hyper metabolism in visual cortex and lingual gyrus in compare to healthy control.

In our case FDG PET scan didn’t show any metabolic hyperactivity in the high visual centers rather than showing asymmetrical temporal lobe metabolism with normal metabolic activity which not goes with visual snow syndrome findings in the literatures.

The syndrome is a variant of migraine aura also known as Persistent Migraine Aura. It is still complex and not well understood, differential diagnosis includes migraine, visual hallucination, concussions and drug side effects, it usually affects the young age group and rarely disappears once it appears. In the last few years medical community has begun to recognize this syndrome. The newest data from 2 studies presented at European Headache Migraine Trust (EHMTIC) in September 2016 that tested 69 (STEROIDS, NSAID, ANTI-DEPRESSANT–etc.) drugs on 88 patients. Of 44% had a response inform of improvement or worsening? Latest recommended updated helpful medication for this syndrome according to the (a case series of 54 adults in UK by Dr. Mark Weatherall) were: (Riboflavin 400mg, Magnesium citrate 600mg, acetazolamide and levetiracetam) [5]. Riboflavin 400mg dose usually used to reduce the frequency and intensity of migraine and act as a precursor in the mitochondrial electron transport that might help to improve brain mitochondrial dysfunction that associated with headache and migraine [6]. Some researches showed a low magnesium level during migraine, that is why magnesium is given to reduce the migraine attacks. Magnesium also improves blood pressure and improves cerebral blood flow as shown in many studies which gives it some benefits in visual snow syndrome [7]. We need more studies in the future to find a good treatment for this extremely life disabling syndrome that may be sometimes a life threatening by causing chronic depression and constant thoughts of suicide.

Conflict of Interests

The authors declare that there is no conflict of interests regarding the publication of this paper.
Figure 4: FDG PET/CT scan: Following IV injection of 18-fluoro-2-deoxyglucose (FDG) and a standard uptake period, 60 minutes, the patient was imaged on a Biograph mCT, Siemens (TOF, 128 slice) scanner. After a noncontrast enhanced CT image was acquired for photon attenuation correction, multiple three-minute bed position acquisitions were obtained from the vertex to the skull base. DOSE: 150 MBq.

References

5. Dr Mark Weatherall PhD FRCP Edin Consultant Neurologist, Consultant Neurologist Charing Cross Hospital, Fulham Palace Road, London, W6 8RF.