Radiation Exposure and Bell’s Palsy: A Hypothetical Association

Khateri M.1, Cheraghi S.1,2, Ghadimi A.3, Abdollahi H.4*

ABSTRACT
Bell’s palsy is an idiopathic peripheral nerve palsy involving the facial nerve. It accounts for 60 to 75% of all cases of unilateral facial paralysis. The main mechanisms to induce BP remain unclear, but infection, ischemic condition and immunodeficiency may contribute to the development of Bell’s palsy. Accumulating evidence has shown several factors can trigger the reactivation of latent HSV including psychological stressors, physical stressors and immunosuppression. Ionization and non-ionization radiations are of importance of physical stressors. Some data have shown radiation can reactivate HSVs. Based on preliminary studies showing radiation reactivation of HSVs, we aimed to hypothesize radiation (in both forms of ionization and non-ionization) may cause Bell’s palsy. In the future, the role of radiotherapy, radiofrequency radiation from mobile phones and wireless devices in HSV reactivation and Bell’s palsy should be investigated.

Keywords
Radiation, Mobile Phone, Herpes Virus, Bell’s Palsy, Hypothesis

Introduction
Bell’s palsy (BP) is a common idiopathic neurologic disorder which involves facial nerve and leads to peripheral nerve palsy. Based on previous studies, BP accounts for approximately 60–75% cases of unilateral facial paralysis [1]. The annual incidence is 15 to 30 cases per 100,000 people [2]. The main mechanisms to induce BP remain unclear, but infection, ischemic condition and immunodeficiency may contribute to the development of Bell’s palsy [3]. As an interesting mechanism suggested by McCormick, reactivation of herpes simplex virus (HSV-1) is a possible cause of BP [4]. Accumulating evidence shows several factors can trigger the reactivation of latent HSV including psychological stressors, physical stressors and immunosuppression. Ionization and non-ionization radiations are of importance of physical stressors. Several studies have revealed that radiation can reactivate HSVs [5].

Riel-Romero et al. observed cranial irradiation might reactivate latent herpes simplex virus. They reported this finding from a 15-year-old male with a brainstem glioma treated with Temozolomide and conventional radiotherapy [6]. In a similar observation, Tohyama et al. reported HSV was reactivated in a 5-year-old male with a pontine glioma, two weeks after completing radiotherapy [7]. The reactivation of HSV in these studies has begun from early days to months after radiotherapy.

Epstein et al. reported that just 3.6% of the head and neck radiotherapy
patients were positive for HSV during radiation therapy [8].

Shimeld et al. demonstrated that ultraviolet radiation could induce the reactivation of herpes simplex virus type 1 in the corneas of latently infected mice [9]. In an experimental study, Perna et al. showed that ultraviolet light could reactivate herpes simplex virus in over 60% of all attempts [10]. Rooney et al. reported UV light is a potent stimulus for inducing reactivation of latent herpes simplex virus (HSV) infections [11].

In a recent study, Yang et al. showed induction of Epstein-Barr virus early antigen expression in Raji cells by GSM mobile phone radiation; meaning that radiofrequency radiation may reactivate herpes simplex virus [12].

Hypothesis

Based on preliminary studies showing radiation reactivation of HSVs, we aimed to hypothesize radiation (in both forms of ionization and non-ionization) may cause Bell’s palsy.

Evaluation of Hypothesis

With unknown cause, BP is a rapid facial nerve paralysis which serves as the most common neurological disorders. As an accepted pathway, the reactivation of latent HSVs using stressors can lead to BP. In the present study, we hypothesized radiation as HSV activator may lead to BP. Although, little evidence has been found associating radiation (ionization and non-ionization) and HSVs reactivation, but scientific evidence shows this fact, practically and theoretically. On the other hand, cancer statistics show head and neck cancers are common cases of cancer in developed countries [13] and as a main treatment approach, these patients receive radiotherapy in their course of treatment. In this light, the incidence probability of BP may rise due to radiotherapy-induced HSV reactivation. As a similar phenomenon, there are studies revealing radiotherapy hepatitis B virus reactivations in hepatocellular carcinoma [14-16].

As an important health concern, non-ionization radiofrequency radiation such as mobile phones, wireless and Wi-Fi are increasing in workplaces, homes and public places and remain as the newest environment pollutants [17-18]. In this era, this concerning situation may also be risen when these radiations have potential to reactivate HSVs and induce Bell’s palsy, particularly mobile phones that are used near the head.

An experiment that would help establish the groundwork for testing the hypothesis would be to expose mice head and necks to radiation followed by an examination of the reactivation of HSVs and BP manifestation.

Further investigation and new experiments in vivo are needed to determine whether radiation exposure particularly in head and neck regions is causing the reactivation of HSVs that would result in BP.

Conclusion

Radiation-induced Bell’s palsy has been hypothesized in the present paper. However, we believe that it can be modelled and studied as a new radiation induced health consequence in radiotherapy cancer patients and also all public which use mobile phones or have been exposed to UV radiation. In the future, the role of radiofrequency radiation from mobile phones and wireless devices in HSV reactivation and Bell’s palsy should be investigated.

Conflict of Interest

None

References

1. Adour KK, Byl FM, Hilsinger RL, Jr., Kahn ZM, Sheldon MI. The true nature of Bell’s palsy: analysis of 1,000 consecutive patients. Laryngoscope. 1978;88:787-801. doi.org/10.1002/lary.1978.88.5.787. PubMed PMID: 642672.
2. Newadkar UR, Chaudhari L, Khalekar YK. Facial Palsy, a Disorder Belonging to In-
fluential Neurological Dynasty: Review of Literature. N Am J Med Sci. 2016;8:263-7. doi.org/10.4103/1947-2714.187130. PubMed PMID: 27583233. PubMed PMCID: 4982354.

3. Eviston TJ, Croxson GR, Kennedy PG, Hadlock T, Krishnan AV. Bell’s palsy: aetiology, clinical features and multidisciplinary care. J Neurol Neurosurg Psychiatry. 2015;86:1356-61. doi.org/10.1136/jnnp-2014-309563. PubMed PMID: 25857657.

4. McCormick DP. Herpes-simplex virus as a cause of Bell’s palsy. Lancet. 1972;1:937-9. doi.org/10.1016/S0140-6736(72)91499-7. PubMed PMID: 4112101.

5. Furuta Y, Fukuda S, Chida E, Takasu T, Ohtani F, Inuyama Y, et al. Reactivation of herpes simplex virus type 1 in patients with Bell’s palsy. J Med Virol. 1998;54:162-6. doi.org/10.1002/(SICI)1096-9071(199803)54:3<162::AID-JMV3>3.0.CO;2-3. PubMed PMID: 9515763.

6. Riel-Romero RM, Baumann RJ. Herpes simplex encephalitis and radiotherapy. Pediatr Neurol. 2003;29:69-71. doi.org/10.1016/S0887-8994(03)00044-4. PubMed PMID: 13679127.

7. Tohyama Y, Sako K, Daita G, Yonemasu Y, Shuke N, Aburano T. Dissociation of 99mTc-ECD and 99mTc-HMPAO distributions in herpes simplex encephalitis. Childs Nerv Syst. 1997;13:352-5. doi.org/10.1007/s003810050096. PubMed PMID: 9272290.

8. Oakley C, Epstein JB, Sherlock CH. Reactivation of oral herpes simplex virus: implications for clinical management of herpes simplex virus recurrence during radiotherapy. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 1997;84:272-8. doi.org/10.1016/S1079-2104(97)90342-5. PubMed PMID: 9377190.

9. Shimeld C, Easty DL, Hill TJ. Reactivation of herpes simplex virus type 1 in the mouse trigeminal ganglion: an in vivo study of virus antigen and cytokines. J Virol. 1999;73:1767-73. PubMed PMID: 9971753. PubMed PMCID: 104415.

10. Perna JJ, Mannix ML, Rooney JF, Notkins AL, Straus SE. Reactivation of latent herpes simplex virus infection by ultraviolet light: a human model. J Am Acad Dermatol. 1987;17:473-8. doi.org/10.1016/S0190-9622(87)70232-1. PubMed PMID: 2821086.

11. Rooney JF, Bryson Y, Mannix ML, Dillon M, Wohlenberg CR, Banks S, et al. Prevention of ultraviolet-light-induced herpes labialis by sunscreen. Lancet. 1991;338:1419-22. doi.org/10.1016/0140-6736(91)92723-F. PubMed PMID: 1683420.

12. Liu Y, Wang ML, Zhong RG, Ma XM, Wang Q, Zeng Y. The induction of Epstein-Barr Virus early antigen expression in Raji cells by GSM mobile phone radiation. Biomed Environ Sci. 2013;26:76-8. PubMed PMID: 23294619.

13. Siegel RL, Miller KD, Jemal A. Cancer statistics, 2015. CA Cancer J Clin. 2015;65:5-29. doi.org/10.3322/caac.21254. PubMed PMID: 25559415.14.

14. Kim JH, Park JW, Kim TH, Koh DW, Lee WJ, Kim CM. Hepatitis B virus reactivation after three-dimensional conformal radiotherapy in patients with hepatitis B virus-related hepatocellular carcinoma. Int J Radiat Oncol Biol Phys. 2007;69:813-9. doi.org/10.1016/j.ijrobp.2007.04.005. PubMed PMID: 17524569.

15. Huang W, Zhang W, Fan M, Lu Y, Zhang J, Li H, et al. Risk factors for hepatitis B virus reactivation after conformal radiotherapy in patients with hepatocellular carcinoma. Cancer Sci. 2014;105:697-703. doi.org/10.1111/cas.12400. PubMed PMID: 24654677. PubMed PMCID: 4317906.

16. Elwood JM. Epidemiological studies of radio frequency exposures and human cancer. Bioelectromagnetics. 2003;Suppl 6:S63-73. doi.org/10.1002/bem.10142. PubMed PMID: 14628307.

17. Kesari KK, Kumar S, Behari J. Effects of radiofrequency electromagnetic wave exposure from cellular phones on the reproductive pattern in male Wistar rats. Appl Biochem Biotechnol. 2011;164:546-59. doi.
18. Feychting M. Mobile phones, radiofrequency fields, and health effects in children-epidemiological studies. *Prog Biophys Mol Biol*. 2011;107:343-8. doi.org/10.1016/j.pbiomolbio.2011.09.016. PubMed PMID: 21958911.