LETTER IN REPLY

Oman Medical Journal [2019], Vol. 34, No. 1: 84-85

Letter in Reply: "Neonatal Bell's Palsy and Possible Correlation with Human Leukocyte Antigens"

Abdulhafeez M. Khair*

Pediatric Neurology Fellow, Thomas Jefferson University, Philadelphia, US

ARTICLE INFO

Article history:

Received: 28 August 2018

Accepted: 28 August 2018

ONLINE:

DOI 10.5001/omj.2019.17

Dear Editor,

We thank you for your interest in our recent case reort. You have raised a very valid argument with regard to not discussing the genetic etiology as a likely explanation of Bell's palsy (BP), given that it is a neurological phenomenon rarely observed in neonates and young infants.

To begin with, we believe that it is worth remembering that BP is the other known name of what can be called idiopathic facial nerve palsy.[1] By emphasizing on the idiopathic nature of the definition, we presumed that all other potential etiologies mentioned (including congenital, inflammatory, traumatic, infectious, syndromic, and ischemic factors) were already carefully considered and excluded before arriving at the diagnosis of BP. Thus, we stressed the importance of clinical and diagnostic evaluation before the diagnosis of BP is entertained or clinically managed.

We reported what we believed is the youngest child to have a diagnosis of BP in the current literature. It is known that neonates with facial nerve palsy are either victims of traumatic deliveries or are due to congenital facial nerve palsy. Our patient had no evidence of any trauma before or during delivery. He had normal physical examination on three different occasions making the possibility of congenital facial nerve palsy very unlikely if at all possible. In addition, our patient did receive a course of steroid therapy with complete recovery, again arguing against congenital palsy as the underlying etiology.

Regarding the possibility of genetic makeup predisposing children to BP, we believe that this debate does and will continue. For a lot of idiopathic neurological disorders, we are learning almost daily about a new genetic alteration that might or might not be significantly related to that specific disorder. BP is not an obvious exclusion. Whether this relationship is of causative, triggering, correlating, associating nature cannot be concluded with sufficiently strong evidence to date.[2]

Familial BP is a closely related interesting phenomenon, which was not discussed in detail in our paper. This is a very rare disorder contrary to the usual BP with strong familial inheritance component as the major differentiating factor. Zaidi et al,[3] reported a family of whom seven members were diagnosed with BP. Interestingly, six of them were females.[3] More recently, a group of researchers from Denmark reported a three generation family with recurrent BP with a pedigree pattern suggestive of autosomal dominant inheritance with near full penetrance.[4] However, the authors were not able to identify any particular genetic mutation or alteration in their study, leaving the door open for further studies.

Regarding human leukocyte antigen (HLA) and possible association with the development of clinical BP, studies are old yet sparse. The author has referred to some old literature arguing about a possible association with HLA backgrounds like DR2, DR4, DRW6, and DRW7 antigens, which we agree as a principle. These old retrospective studies did often lack statistically appropriate sample size and did not differentiate between purely idiopathic or familial

*Corresponding author: ✉drabboody@hotmail.com

variant of the disorder. The laboratory results were not repeated down the road for those patients to ensure consistency of results. Moreover, there is no more recent follow-up studies. We believe (among other scientists) that these HLA themes might represent a state of immune system dysregulation during the clinical presentation of BP, which might be a transient phenomenon.[5] Considering all of these observations and data, none of these patients were as young as our patient. Either way, we agree with the interesting theory and results of previous retrospective studies above. However, we believe in the need for more rigorous analysis and studies to evaluate objectively the concept of genetic drive with or without autoimmune trigger for developing BP, particularly in young children.

REFERENCES

1. Hashisaki GT. Medical management of Bell`s palsy. Compr Ther 1997. Nov;23(11):715-718.
2. Ciorba A, Corazzi V, Conz V, Bianchini C, Aimoni C. Facial nerve paralysis in children. World J Clin Cases 2015 Dec;3(12):973-979.
3. Zaidi FH, Gregory-Evans K, Acheson JF, Ferguson V. Familial Bell's palsy in females: a phenotype with a predilection for eyelids and lacrimal gland. Orbit 2005 Jun;24(2):121-124.
4. Gronhoj Larsen C, Gyldenove M, Jonch AE, Charabi B, Tumer Z. A Three-generation family with idiopathic facial palsy suggesting an autosomal dominant inheritance with high penetrance. Case Rep Otolaryngol 2015;2015:683938.
5. Kubik M, Robles L, Kung D. Familial Bell's palsy: a case report and literature review. Case Rep Neurol Med 2012;2012:674981.