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Research Article

Postural Disorders in Neuromyelopathy by Deficiency in Vitamin B12

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Abstract

Neuromyelopathy by deficiency in Vitamin B12 manifests by loss of proprioception which is predominant in lower limbs. Spasticity is less important. These two manifestations lead to an ataxo-spasmodic gait. Posture is defined as the disposition of body segments each one to another. Posture is closely dependent of proprioception, vestibular and visual functions, and muscle tone. Thus, one may expect frequent postural disturbances in neuromyelopathy by Vitamin B12 deficiency. Only few studies on neuromyelopathy by Vitamin B12 deficiency exist. Our study aims to evaluate postural disorders in neuromyelopathy by deficiency in Vitamin B12.

Methodology

Patients with neuromyelopathy by deficiency in Vitamin B12, included and balance disorders evaluated at stand-up and in actions. It was a prospective study which done in Physical Medicine and Rehabilitation department of the Hospital University of Fann, Dakar.

Results

8 patients of which 4 women were included. The average age was 40 years. Palmar melanodermia was constant. The transfer on beds was possible in 7 cases and 1 case needed help. The postural equilibrium sitting position necessitated help in 1 case. The standing position was possible legs spread in 7 cases, impossible joined legs in 7 cases, anteroposterior pushes, laterals , eye closures, the picking up of an object in all the patients caused a destabilization. Head and trunk anteflexion in upstanding, with a pattern of bended head during walking was found is all of our patients. With Vitamin B supplementation and rehabilitation, the support on a leg at 6 months was impossible in more than 10 seconds in all the patients.

Discussion and Conclusion

Postural disorders in neuromyelopathy by deficiency in Vitamin B12 are certainly discrete but constant. They present predominantly by head and trunk anteflexion in upstanding, a loss of lateral gaze during walking and multidirectional oscillations worsened by eyes closure, and pick up of an object on the floor. This is in line with available data in the published literature. For economic reasons, Vitamin B12 dosage is difficult in our setting. Spinal cord degeneration or a sensitive ataxic neuropathy associated with a palmar melanodermia in a black African should prompt a therapeutic test with high doses of Vitamin B12. An accurate and early management often allows recovery of daily living activities.

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Introduction

The neurologic disorders associated with pernicious anemia were well described in the literature of the late nineteenth century and the first decade of this century [1-7]. Certain limitations including uncertainty as to underlying diagnosis and problems in evaluating therapy are noted in previous studies. Since the introduction of various modern diagnostic and therapeutic measures, many reports of neurologic disorders associated with pernicious anemia involving single patients or small numbers of patients have appeared [2,8-12]. Neuromyelopathy by Vitamin B12 deficiency means all manifestations in relation to spinal cord lesions or/and peripherals nerves lesions related to Vitamin B12 deficiency. It is more common in elderly and is under diagnosed because of subtle clinical manifestations [13]. Neuromyelopathy by deficiency in Vitamin B12 is due to a deficit in an intrinsic factor or in an inability to deliver cobalamin from the food or from its carrying proteins [14-16]. Neuromyelopathy by deficiency in Vitamin B12 manifests by profound sensibility [17-19]. Involvement of large sensitive fibers increases the susceptibility to postural disorders. In Tunisia, postural disorders during spinal cord degeneration were evaluated using a stabilimeter. They reported a postural instability in frontal and sagittal plans. Given the predominance of proprioceptive ataxia, these authors already highlighted the importance of postural rehabilitation [20].

Objectives

To evaluate postural disorders in neuromyelopathy by deficiency in Vitamin B12.

Methodology

We have carried out a study concerning patients followed up in the service of Physical Medicine and Functional Rehabilitation of Hospital University of Fann, Dakar for neuromyelopathy by deficiency in Vitamin B12. This was a case series study. Cases were collected over a period of 14 months. Neuromyelopathy diagnosis was made upon:

- A progressive onset combined spinal cord degeneration syndrome.
- And/or a sensitive predominant neuropathy.
- Melanodermia of skin and mucosa on black skin.
- A level of Vitamin B12 inferior to 200pg/nl.
- A positive therapeutic challenge with Vitamin B12.

Therapeutic challenge with Vitamin B12 consists in intravenous or intramuscular daily injection of 1000µg of Vitamin B12 for one week. This test is positive if melanodermia and full blood count disorders improve.

Socio-demographic characteristics, results of the neurological examination, upstanding and gait disorders were recorded.

Posture was assessed

- In the sitting position: Ability to maintain the sitting position without or with an aid for more than 15 seconds.
- In upstanding: Spontaneous body attitude and the ability to stand with legs open or closed, oscillations during eyes closure, destabilization by pulling and the ability to maintain unipodal support for more than 10 seconds.
- During action: Destabilization or oscillation when picking up an object on the floor, and gaze orientation during walking.

The abnormalities of full blood count, the level of Vitamin B12, the electromyography data and results of MRI were collected. Data analysis is done with Fischer method.

Results

8 patients of which 4 women were included. They were all black Africans. The average age was 40 +/- 10.57 years. A patient had a previous acute poliomyelitis during childhood cramps. Seven patients had no contributively past medical history. Swollen extremities, sensation of disequilibrium in the course of moving were revealing signs in all the patients. Urinary disorders were present and transitory in 1 case. The physical examination has objective proprioceptive ataxia and a palmar melanodermia in all the patients. The other signs were a partial muscle weakness (5 cases), severe (1 case) a tactile hypoesthesia and painful in socks (3 cases), respiratory disorders and an asymmetric amyotrophic of inferior members (1 case). Osteotendinous reflexes were quick in 3 cases and abolished to the inferior members in 2 cases. Polyneuropathy (2 cases), myelopathy (6 cases) were neurological syndromes found. The transfer on beds was possible in 7 cases and 1 case needed help. The postural equilibrium sitting position necessitated help in 1 case. The standing position was possible legs spread in 7 cases impossible joined legs in 7 cases anteroposterior pushes, laterals, eye closures, the picking up of an object in all the patients caused a destabilization. A trunk anteflexion was found during upstanding in all the patients. Walking with aid and loss of lateral gaze was recorded in seven patients. Walking was not possible for one patient. Electroneuromyography is done in 2 cases, MRI (2 cases), dosage of Vitamin B12 (4 cases). All patients have blood account and a macrocytosis was found in 3 cases. Supplementation of Vitamin B12 is done in all patients. Two patients received a blood transfusion. Walking without aid was possible for seven patients, and in one patient, walking was possible with an aid. The support on a leg at 6 months was impossible in more than 10 seconds in all the patients. No fall was noted.

Discussion

Neuromyelopathy by deficiency in Vitamin B12 concerns mainly adults. Mean age at onset is in the fifth decade in North African studies [17,19]. It is more level in Healton **s**tudy [7]. The mean age of our series is lower than which reported in the literature. In North African series, posture was not assessed, but proprioceptive ataxia was constantly recorded, as well as in studies from USA [2,7,21]. In Edwards Study, the most common abnormality was diminished vibratory sensation which was found in 87.7% of cases and proprioception was diminished or absent n the toes or anklers in 59% [7]. Results of the previous studies can let presume the existence of postural disorders in these series. In a study from Tunisia, postural disorders evaluated using a stabilimiter were constantly recorded [20]. These authors found instability in the frontal plan in one patient, and in the sagittal plan in the other. However, the limited number of participants included (two) do not permit to draw any conclusion on this issue**.** Postural disorders in neuromyelopathy by deficiency in Vitamin B12 are certainly discrete [18] but constant. Walking under visual supervision of foots is an adaptative mechanism for proprioception and balance deficits. Thus, balance rehabilitation may be helpful in this case. Kerdoncuff et al., recommend eyes closed balance rehabilitation to reduce visual dependence [22]. The dosage of Vitamin B12, intrinsic factors and the realization of the schiller test are rarely carried out. In our current practice due to limited means [13]. In these conditions in front of a combined spinal cord degeneration syndrome or a sensitive predominant neuropathy, associated with a palmar melanodermia in a black African, a therapeutic test with high doses of Vitamin B12 is warranted. This goes to the direction of observation of Bagé et al., [23]. The treatment of neuromyelopathy by deficiency of Vitamin B12 must be introduced early because of the relationship found between severity of neurological symptoms and duration [24,25]. Neurologic disorders in patients with Cobalamin deficiency not related to overall severity of deficiency of vitamin [7]**.** The previous observers found that profoundly anaemia patients frequently had no neurologic signs or symptoms [25,26]**.** An early addition of Vitamin B12 associated with rehabilitation of postural equilibrium stand and proprioception can restore professional activities and daily living activity [23]**.** Interest of balance rehabilitation in spinal cord degeneration was already outlined by Abdelkafit et al., [20]**.** Healton study mentioned a complete resolution of all signs and symptoms [7].

Conclusion

Postural disorders are constant in neuromyelopathy by deficiency in Vitamin B12. They mainly affect upstanding posture, walking and pick up of objects on the floor. In neuromyelopathy by deficiency in Vitamin B12, walking is under visual control of steps. Open and close eyes balance rehabilitation are mandatory for the patient to resume his daily living activities. Eyes closed balance rehabilitation aims to reduce the visual dependence constantly observed in neuromyelopathy by Vitamin B12 deficiency.

Study Limitations

The small size of our series imposes to continue this study and include more patients. Our results could be more objective with the use of a stabilimeter**.** Nevertheless, an accurate clinical evaluation can yield relevant results.

References

1. Ahrens RS (1932) Neurologic Aspects of Primary Anemia. Arch NeurPsych 28: 92-111.
2. Chanarin I (1979) The megaloblastic anemia, (2nd edn), Oxford Blackwell Scientific Publication, New Jersey, USA.
3. Goldhamer SM, Bethell FH, ISAACS R, Sturgis CC (1934) The Occurrence and Treatment of Neurologic Changes in Pernicious Anemia. JAMA 103: 1663-1667.

4. Longdon FW (1905) Nervous and Mental Manifestations of Pre-Pernicious Anemia. JAMA 45: 1635-1638.

5. Minot GR, Murphy WP (1927) A Diet Rich in Liver in the Treatment of Pernicious Anemia. JAMA 89: 759-768.

6. Ungley CC, Suzman MM (1984) Subacute combined degeneration of the cord. Symptomatology and effects of liver therapy. Brain 52: 271-294.

7. Healton EB, Savage DG, Brust JC, Garrett TJ, Lindenbaum J (1991) Neurologic aspects of cobalamin deficiency. Medicine (Baltimore) 70: 229-245.

8. Arias IM, Apt L, Pollycove M (1955) Absorption of radioactive vitamin B12 in nonanemic patients with combined-system disease. N Engl J Med 253: 1005-1010.

9. Fine EJ, Soria E, Paroski MW, Petryk D, Thomasula L (1990) The neurophysiological profile of vitamin B12 deficiency. Muscle Nerve 13: 158-164.

10. Hyland H, Farquharson RF (1936) Subacute combined degeneration of the spinal cord in pernicious anemia. Arch Neurol Psychiatr 36: 1166-1205.

11. Killander A (1958) Subacute combined degeneration of the spinal cord; the diagnostic value of serum vitamin B12 assay. Acta Med Scand 160: 75-84.

12. McCombe PA, McLeod JG (1984) The peripheral neuropathy of vitamin B12 deficiency. J Neurol Sci 66: 117-126.

13. Andrès E, Loukili NH, Noel E, Kaltenbach G, Abdelgheni MB, et al. (2004) Vitamin B12 (cobalamin) deficiency in elderly patients. CMAJ 171: 251-259.

14. Carmel R (2013) Diagnosis and management of clinical and subclinical cobalamin deficiencies: why controversies persist in the age of sensitive metabolic testing. Biochimie 95: 1047-1055.

15. Dali-Youcef N, Andrès E (2009) An update on cobalamin deficiency in adults. QJM 102: 17-28.

16. Andrès E, Perrin AE, Demangeat C, Kurtz JE, Vinzio S, et al. (2003) The syndrome of food-cobalamin malabsorption revisited in a department of internal medicine. A monocentric cohort study of 80 patients. Eur J Intern Med 14: 221-226.

17. Bouchal S (2012) Les manifestations neuropsychiatriques révélant la maladie de biermer (neurobiermer) et leur évolution après supplémentation en vitamine b12 (a propos de 30 cas). Thèse n°/161 12, Université sidi Mohammed Ben Abdellah, Faculté de médecine et de pharmacie, Fes, Morocco. Pg no: 1-96.

18. El Mostafa Al (2014) Les atteintes neurologiques par carence en vitamine B12, Etude d'une série de 11 cas colligé au service de neurologie de l'hôpital de Meknés. These 74/14. Université sidi Mohammed Ben Abdellah, Faculté de médecine et de pharmacie, Fes, Morocco. Pg no: 1-153.

19. Maamar M, Tazi-Mezalek Z, Harmouche H, Ammouri W, Zahlane M, et al. (2006) [Neurological manifestations of vitamin B12 deficiency: a retrospective study of 26 cases]. Rev Med Interne 27: 442-447.

20. Nesrine A, Migaou H, Salah E, Brahim HB, Boudokhane S et al. (2016) Zohra Ben Salah Frih. Sclérose combinée de la moelle et rééducation de l'équilibre: A propos de 2 cas. 2 eme Journées Maghrébines de MPRF, 20 éme Congrés National de la Société Tunisienne, de Médecine Physique, Réadaptation Fonctionnelle, Hammamet, Tunisia.

21. Sethi N, Robilotti E, Sadan Y (2004) Neurological Manifestations of Vitamin B-12 Deficiency. The Internet Journal of Nutrition and Wellness 2: 1-7.

22. Kerdoncuffa V, Duruflea A, Petrillib S, Nicolasb B, Robineaub S, et al. (2004) Intérêt de la rééducation par biofeedback visuel sur plateforme de stabilométrie dans la prise en charge des troubles posturaux des hémiplégiques vasculaires. Annales de réadaptation et de médecine physique 47: 169-176.

23. Bagé H (2008) Un nouveau cas de sclérose combiné de la moelle révélatrice d'un déficit en vitamine B12 decouverte à l'occasion de la rééducation. Journal de Réadaptation Médicale: Pratique et Formation en Médecine Physique et de Réadaptation 28: 7-14.

24. Bethell FH, Sturgis CC (1948) The relation of therapy in pernicious anemia to change in nervous system. Early and and late results in a series of cases observed for periods of not less than ten years and early results with treatment of folic acid. Blood 3: 57-67.

25. Cox EV (1962) The Clinical manifestations of Vitamin B12 deficiency in addisonian pernivious anemia In: Heinrich HC (ed.). Vitamin B12 an intrinsic factors Europaisches Symposium hamburg. Stuttgart, Germany. Pg no: 590-602.

26. Davidson SP, Gulland GL (1930) Pernicious anemia. London Kimpton.