Autoimmune diseases in a Nigerian woman – A case report

***O.A. Talabi, M. O. Owolabi and B. O. Osotimehin**
Neurology unit, Department of Medicine
University College Hospital, Ibadan, Nigeria.

Summary

Autoimmune diseases (AD) are conditions in which there is the development of antibodies against self cells/ organs. AD could either be organ-specific or non-organ specific (systemic) in clinical presentation.

Commonly reported ADs includes: Myasthenia gravis, Hashimoto thyroiditis, Guillian-Barre syndrome, vitiligo, type 1 diabetes mellitus, Graves diseases, Goodpastures syndrome, pemphigus, rheumatoid arthritis, systemic lupus erythematosis, Addisons disease, multiple sclerosis, pernicious anaemia, autoimmune haemolytic anaemia, chronic active hepatitis, idiopathic thrombocytopenic purpura. There is paucity of locally documented information on the occurrence of AD in same patient in our environment. We therefore report the case of a 66 year old woman who presented at the University College Hospital (UCH), Ibadan, with a spectrum of the AD, Vitiligo, rheumatoid arthritis, myasthenia gravis, impaired glucose tolerance.

Keywords: ***Autoimmune, Disease, Suspicion, Index, Spectrum.***

Résumé

Des maladies auto-immunes (AD) se caractérisent par l'apparition d'anticorps nuisibles à l'organisme. AD pourrait êtré soit un organisme spécifique soit un organisme non-spécifique (systémique) dans une présentation clinique.

Des ADs communément rapportés sont: myasthénie gravis, thyroidite de hasimoto, syndrome de Guillain-bare, vitiligo, diabètes mellités type 1, maladies graves, syndrome Bon paturage, pemphigus, arthrite rhumatoide, lupus erythémateux systémique, maladie d'adisons, sclérose multiple, hépatite chronique active, purpura idiopathique thrombocytopenique. On remarque qu'il y a un manque d'information locale bien documentée sur l'incidence de AD chez la même patiente dans notre milieu.

Donc, nous présentons le rapport d'un cas d'une femme âgée de 66 ans qui c'est présentée au College Hospitalo Universitaire (UCH), d'Ibadan, avec un spectre de AD, vitiligo, arthrite rhumatoide, gravis myasthénie, tolérance glucose détériorée.

Case report

A 66 year old lady A. A hospital number 1002606 was referred to the Department of Medicine of the University College Hospital (UCH), Ibadan, Nigeria following a -week history of double vision, and generalised body weakness and easy fatigability. She was actually referred to our centre as a possible case of raised intracranial pressure, with a computerised tomography of the brain. She was apparently in good health until she noticed a rather sudden onset of diplopia and difficulty in opening her eyes (ptosis) while sitting in her shop. About the same period she also noticed a gradual weakness of her body and easy fatigability. There was fluctuancy in the strength of her muscles as she got progressively worse with activity.

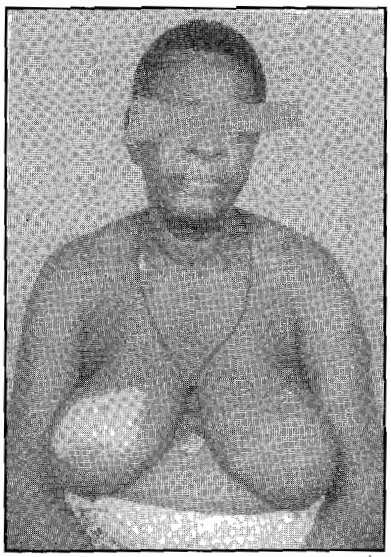

Fig. 1 ***Non-anaesthetic hypopigmented macular patches over the scalp, trunk, breast and limbs.***

There was no vomiting, headache, seizures or sphincteric disturbances and her speech was normal. There was neither difficulty with a swallowing nor breathlessness.

She had early morning stiffness of the wrists as well as the right ankle in the last eight months, swollen joints of the hands. She also experienced recurrent right knee pain, which made walking difficult for the last 2 years. She was treated with various non-steroidal anti-inflammatory agents. The blood pressure had

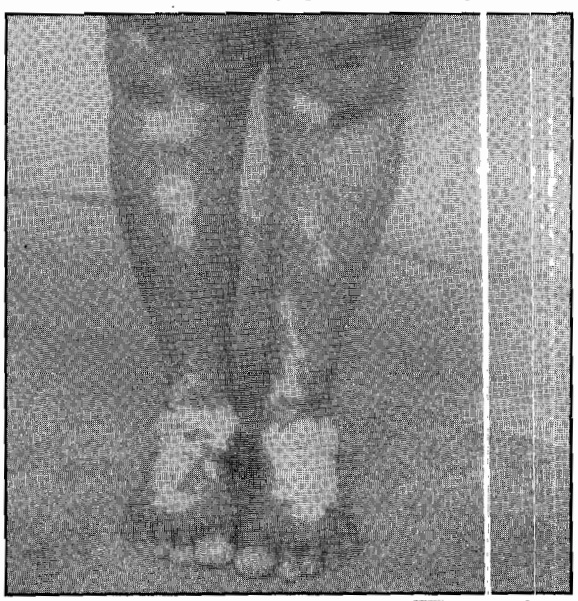

Fig. 2 ***Non-anaesthetic hypopigmented macular patches over the lower limbs.***

**Correspondence*

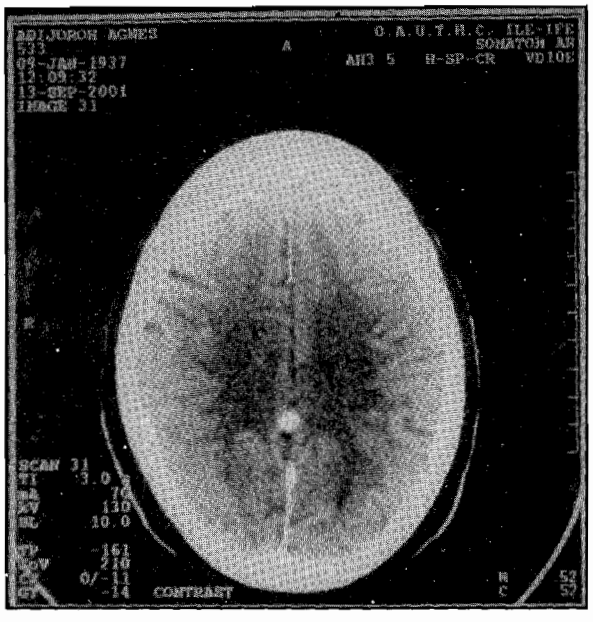

Fig. 3. ***Solitary contrast-enhancing hyperdensity superior to the corpus callosum.***

been elevated since the onset of the noticed illness. There was no history of diabetes mellitus. Her mother had stroke about 8 months prior to the illness, while the Father died of an undisclosed illness. She did not use alcohol or cigarette. She was married with 9 children, 6 girls and 3 boys all alive and well. One of her daughters had wide spread vitiligo at 24 years of age.

Examination revealed that she had wide spread multiple, non-anaesthetic hypopigmented macular patches involving the lower limbs, trunk, and the scalp (Figures 1 and 2. She also has prominent ptosis marked on the right eye, worse with activity. She had bilateral immature cataracts with normal visual fields. The hands had swollen and tender interphalengeal, metacarpophalegeal joints, with ulnar deviation. The right knee was swollen with crepitus and reduced range of movements. The blood pressure was elevated at 130 – 160/90 – 104mmHg. Tensilon (Edrophonium test was positive with sustained improvement in muscle strength. The clinical diagnosis comprised myasthenia gravis, vitiligo, and rheumatoid arthritis. Investigations carried out included, packed cell volume of 45%, total white blood count of 4,200/cc, with normal differentials. Platelet count was normal. Urinalysis revealed trace of protein while electrolytes and urea were within normal limits. The erythrocyte sedimentation rate (ESR), was 5mm/hour (Westergreen) while the fasting and 2 hour post prandral plasma glucose were 117mg/dl and 197mg/dl respectively. The oral glucose tolerance test revealed 84, 152, 203, 213, 193mg/dl at 0, 30, 60, 90, 120 minutes respectively. The chest X-ray and the liver function tests were normal. Anti-hepatitis C virus antibody was negative.

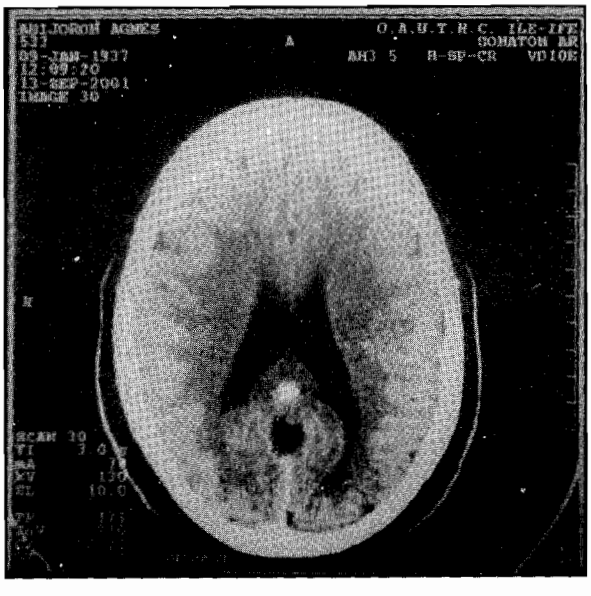

Fig. 4. ***Solitary contrast-enhancing hyperdensity superior to the corpus callosum.***

A cranial tomographic scan (Figures 3 and 4) showed solitary round, contrast-enhancing hyperdensity located superior to the corpus callosum posteriorly with the consideration of an arterio-venous malformation (AVM), while tuberculoma, calcified parasite and astrocytoma were considered differentials. Rheumatoid factor, autoantibodies electromyography and angoigraphy were not done for lack of facility at our centre. The patient was managed with Tab. Pyridostigmine 30mg thrice daily, tab Nifedipine 20mg twice daily, and tab diclofenac sodium (Voltaren®) 100mg daily with good response.

Discussion

Autoimmune Diseases are often autoantibody mediated and T-cell dependent human diseases. There is little documented in the local literature on the co-existence of autoimmune diseases in Africans. The major issue in ADs is the initiation, propagation and persistence of immunologically mediated inflammatory changes in the absence of any clearly defined pathogen(s). The ultimate outcome is the destruction of tissues so targeted which is often devastating with attendant consequences. The exact initiation process remains unclear, however, the persistent activation of the helper T-lymphocytes against self proteins seems to be a nearly constant feature in autoimmune diseases[2,3]. The roles of genetic, environmental and immunoregulatory factors were outlined by Van Noort et al[2], when they reinforced the multifactorial origin of autoimmunity. It must be emphasized that the presence of autoantibody does not necessarily mean that it is the cause of the presenting disease; it may just be entirely secondary in origin as it is with syphillis and the anti cardiolipin. In the same vein the absence of autoantibody does not exclude an autoimmune disease; as it may be purely T-cell mediated disease. Special considerations were given to the contribution of infectious events as well as that of stress proteins in the generation of disordered immune process[2]. However the genetic influence via the histocompatibility leucocyte antigen (HLA) is prominent among the autoimmune diseases. HLA B8 DR3 have been found in association with different organ-specific diseases to which it is linked. The HLA status of the presented patient and her daughter are not known, but the presence of combinations of autoimmune disease spectrum in this case as well as the presence of vitiligo in her daughter may well suggest a genetic linkage. It is important to note that few African physicians do not see vitiligo as a purely autoimmune affair because they tend to see autoimmune diseases in isolation. I believe this may be due to a relatively poor search for the existence of other autoimmune conditions. This is the purpose of this case report.

The role of genes in tolerance and immunoregulation in systemic autoimmunity was highlighted by Moller[3], Theofilopoulos and Kono[4] and Lipsitz et al[5]. They not only found significant role for the genetics in the autoimmune pro-

cess initiation but also identified the usefulness of the experiences gained with the experimental models in attempts at blockage of the activation of self-reactive T-lymphocytes. The use of experimental animal models in the cases of diabetes mellitus and multiple sclerosis have experimental animal models in the cases of diabetes mellitus and multiple sclerosis have led to the application of therapies to block the activation of self-reactive T-lymphocytes[2]. The ultimate is to develop antigenic-specific strategies to block the process of autoimmune in humans. The search from mammalian genome to identify the predisposing loci for the various autoimmune diseases has begun among some researchers. This will be a great relief to the efforts aimed at preventing this often devastating condition[6,7]. Gene therapy is another area receiving attention and trials. It is targeted at the correction of the identified immunoregulatory aberrations which has become a new direction in the treatment of autoimmune diseases[8-9].

In view of the recent report of possible association between hepatitis C infection and the development of some autoimmune responses, particularly concerning MG, our patient was screened and the hepatitis C antibody was negative[10,11,12,13,14]. In this cas report, the co-existence of numerous autoimmune diseases in a patient is highlighted. Though the diagnosis here is largely clinical with little laboratory support, vitiligo, myasthenia gravis and rheumatoid arthritis are recognised autoimmune conditions. The emphasis here is that the clinical identification of an autoimmune classified disorder should necessarily initiate the search for the possible presence of other autoimmune diseases. This could be initially clinical but laboratory confirmation is often necessary. It is not common in our practice to find patients presenting with combinations of the AD as reported in this case where myasthenia gravis, vitiligo, rheumatoid arthritis and impared glucose tolerance (IGT) occurred together. It is well acknowledged fact that IGT often ends up with overt diabetes mellitus[15,16].

The normal PCV and FSB are not uncommon in some autoimmune diseases. The thyroid function tests, autoantibodies, antibody against the intrinsic factors in the stomach were not carried out for lack of facilities. The brain CT findings appears incidental and have no known recognised association with the patients presentation of autoimmune diseases.

References

1. Penn AS. Myasthenia gravis and related disorders; Am. NY Acad. Sci, 1993; 86: 1– 611.
2. Van Nort JM, Amor S. Cell biology of autoimmune diseases. International Review of Cytology 1998; 178: 127 – 206.
3. Moller E. Mechanisms for induction of autoimmunity in humans. Acta Paediatrica. Suppl: 1998; 424: 16 – 20.
4. Theofilopoulos AN,Kono DH. The genes of systemic immunity. Proceedings of the Association of American Physicians 1999; 111: 228 – 240.
5. Lipsitz D, Berry JL, Shelton GD. Inherited predisposition to myasthenia gravis in Newfoundland. J. Amer. Vet Med. Assoc. 1999; 215: 956 – 958.
6. Witkowska D. Molecular mimicry of bacteria as a factor in bacterial pathogenicity. Postepy Hygienyi Medycyny doswiadczalney 1999; 53: 545 –559.
7. Kumar V, Sercarz E. Distinct levels of regulation in organ-specific autoimmune diseases; Life Sciences 1999; 65: 1523– 1530.
8. Tsokos GC, Nepo GT. Gene therapy in the treatment of autoimmune diseases. J. Clin. Invest. 2000; 106 181 – 183.
9. Fathnan CG, Costa GL, Seroogy CM. Gene therapy for autoimmune disease. Clin. Immunol. 95 (suppl.): 2000; S39 – S43.
10. Reading PJ, Newman PK. Untreated hepatitis C may provoke myasthenia gravis. J. Neurol. Neurosurg. Psychiatry. 1999; 62: 302 – 303.
11. Halfon P,Levy M, San Marco M, Gerolami V, Khiri H, Bourliere M et al. Myasthenia Gravis and hepatitis C virus infection. J. Viral Hepatitis 1999; 3: 329 – 332.
12. Harada H, Tamaoka Kohno, Ymochizuki A, Shoji S. Exacerbation of myasthenia gravis in patients after interferone beta treatment of chronic hepatitis C Neurol. Sci. 1999; 165: 182 – 183.
13. Eddy Wim R, Peter VE, Tanja RT, Werner VS. Myasthenia gravis: another autoimmune disease associated with hepatitis C virus infection. Digestive disease and Sciences 1999; 44: 186 – 189.
14. Talabi OA. Paucity of Hepatitis C virus antibody in patients with myasthenia gravis in Ibadan, Nigeria. Nig. J. Med 2002; In Press.
15. Saa MF, Knowler WC, Petti DJ, Nelson RG, Mott DM, Bennett PH. The natural history of impaired glucose tolerance in Prima Indians. New England J. Med. 1988; 319: 1500 – 1506.
16. Kean H, Jarret RJ, Mc Cartney P. The ten year follow up of the Bedford survey (1962 – 1972): glucose tole ance and diabetes mellitus. Diabetlogica 1982; 22: 73 – 78.