exposed to medications that suppress immune function like methyl prednisolone administered immediately after traumatic injury (18). Other possible explanation that central nervous system injury evoked a T cells dependent neuroprotective response from damage the neuron tissues by immune system (17). Neuroendocrine immune system interactions was an important factor in the restoration of immune functions, this was fulfilled by Cruse *etal* that found both natural and adaptive immune responses were found strikingly decreases (natural killer cells functions, T cells functions, interleukins 2 receptors, interlukins 2, interlukins 6, inter cellular adhesions, plasma adrenocorticotrophic hormone (ACTH) and urine free cortizol) and after physical rehabilitation therapy over a period of six months after injury, these parameters restored to near normal levels in most patients (11, 12).

In our patients when there was cut in the spinal cord and release of sequestered antigens and stimulations of immune system. This did not lead to development of autoantibodies against nuclei, mitochondria and smooth muscle antigens in patients group.

Recommendations:

Other autoantibodies must be investigating against neurological tissues like HU, Ri, Yo and other tissues using indirect immunoflurescence and western blot methods.

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Patients And Methods:

- Patients group consists from forty-eight patients with spinal cord injury: twenty-eight were quadriplegics and twenty patients were paraplegics. Those patients were admitted to hospital of spinal cord injury (Ibn Al-Giff formerly) from 2002 to 2003. Their age were ranged from 20-65 years. Males were 38 and females were 10.
- Control group was 25 apparently healthy individuals, their age ranged from 18-60 years. Males were 14 and females were 11.
- Sera of both groups were separated from the blood and used for detecting the presence of autoantibodies against nuclear, mitochondria and smooth muscle antigens using indirect immunoflourescence method (13).

Results:

The autoantibodies were investigated against nuclear, mitochondria and smooth muscle in both patients and control groups. In case of antinuclear antibodies, there was no significant difference between two groups. The last other autoantibodies (mitochondria and smooth muscle) were not detected in control and spinal cord injured patients (Table-I-).

Groups	Antinuclear Autoantibodies No.	Antimitochondria Autoantibodies	Anti smooth muscle No.
Patients No.=48	4 (1)	-	-
Control No=21	2 (1)	-	-

(1) = Not significance

Table-I- Autoantibodies in spinal cord injury patients compared with control groups.

Using student t-test did statistical analysis.

Discussion:

Spinal cord injury is one of the neurological diseases that were investigated for the presence of autoanti bodies. Autoantibodies can be generated by a variety of mechanisms like release of sequestered antigens, molecular mimicry, inappropriate expression of class II major histocompatibility complex and polyclonal B cells activations (2,19,20). Blood brain barrier was an ill-defined obstacle normally excludes intravascular proteins including IgG and effective barrier to cell mediated response (3). Once circulating autoantibodies reach the central or peripheral nervous system, damage may ensue. Detection of new autoantibodies explains the pathogenesis of some neurological diseases like Lambert-Eaton myaesthenic syndrome was an excellent example of this (1).

Autoantibodies were associated with paraneoplastic neurological syndromes that were helpful in the diagnosis and treatment like Hu autoantibodies and it's association with small lung cancer (9). Unfortunately, these autoantibodies were not always of value in diagnosis of diseases because they were present in normal persons and other diseases (5). Posner provided useful guidelines for assessing the clinical relevance of antineuronal antibodies: a particular antibody must be present in more than one patient with similar neurological disordered and tumors and occurrence of both false positive and negative tests should be rare, concentration of antibodies in the serum should be relatively high, high titer in cerebro spinal fluid than in the serum would suggest intrathecal synthesis and neurological relevant antibody and this antibody should react with a symptomatic part of the nervous system and the nature of antigen must be identified by western blot and immunohistochemistry(6). In our study we did not detect anti mitochondria and anti smooth muscle antibodies. In case of anti nuclear antibodies there were no significant differences between patients group and control group. Our finding that was similar to Siosteen etal whom they did not detect anti sperm autoantibodies in the serum of patients with spinal cord injury (4).

Absence of these autoantibodies might be due to decrease expression of major histocompatibility complex class II and corticosteroid regulation of immunity that affect neuroimmune interactions (14), corticosteroid administration as one method of treatment of autoimmune diseases (15). Extra justification, the etiology of this was due to decentralization of the autonomic nervous system and may be due to over

Auto antibodies in Iraqi Patients with Spinal Cord Injury

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Summary:

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Background: *Autoantibodies can be generated by different mechanisms and detected in different autoimmune diseases, neoplastic conditions and other diseases. These autoantibodies are important in the diagnosis and management of these disordered.*

Patients And Methods: *Forty-eight patients with spinal cord injury, twenty-eight quadriplegics and twenty patients were paraplegics, resident in spinal cord accidents hospital (Ibn - Algiff formerly) from 2002 to 2003. The other group was twenty-five apparently healthy individuals. Both groups were investigated for the presence of autoantibodies against nuclear, mitochondria and smooth muscle using indirect immunofluorescense test.*

Results: *There was no significant difference in anti nuclear antibody between patients and control group. Other autoantibodies, mitochondria and smooth muscle were not detected in both groups.*

Discussion: *Spinal cord injury did not lead to formation of autoantibodies in spite of damage to spinal cord and exposed sequestered neuronal antigens to immune system probably due to decreased MHC class II expression on neuronal tissues and corticosteroid effect neuroimmune interactions that administered immediately after traumatic injury or cortisol formation in the body.*

Conclusion: *Autoantibodies (nuclear, mitochondria, smooth muscle) were not detected in both control and patients group.*

Recommendations: *Other autoantibodies directed against neurological tissues by using other methods like western blot method.*

Key wards: *autoantibodies , spinal cord , nuclear , mitochondria.*

Introduction:

The central and peripheral nervous system are not excluded from immune diseases since the immune system participates actively in nervous tissues. As else where, T and B lymphocytes will cause tissue damage by invading nervous tissue, some of B cells are responsible for the locally synthesized immunoglobulin found in the cerebrospinal fluid and serum and T cells can cause direct damage such as in chronic viral infection, post infective states and demylenation (1). Spinal cord injury or contusion one of neurological diseases characterized by distribution of microglia, macrophages, T-lymphocytes and astrocytes throughout. There was also increased in the expression of complement receptor type 3 (CR3) (14). In addition to that natural and adaptive immune responses were found to be decreased strikingly after spinal cord injury (11).

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Other immunological abnormalities that present in the neurological diseases were autoantibodies. They were present in neurological diseases of unknown etiology and in paraneoplastic neurological syndromes such as ovarian tumors had an association with Yo autoantibodies (it is an autoantibody that is directed against cytoplasm of neuronal Perkanji cells)(7,8). Other autoantibody was Hu antibody that had a relation with small cell lung tumors (Hu is an auto antibody directed against neuronal nuclei). (5,9).

These autoantibodies react with cerebral tissues and patients own tumors can readily be detected by indirect immunofluresence test using rodent, monkey or human cerebellum. The high titer of these autoantibodies in patients with typical clinical features was of considerable value in the diagnosis and management. Autoantibodies against neurological tissues in many other neurological diseases such as antigangliosides antibodies in polyneuropathies (10).

In this research we will try to investigate the autoantibodies, which are present in the serum of patients directed against nuclear, mitochondria and smooth muscle as a result of spinal cord injury.