International Journal of Science and Research (IJSR)
ISSN: 2319-7064
SJIF (2022): 7.942

A Case of Nummular Dermatitis / Eczema Caused by Compressive Myelopathy Diagnosed after 20 Years

Dr. V. P Jerath MD[1], Dr. Anurag Arora MD[2]

[1]ECFMG (Certified) USA, Fellow Indian College of Allergy & Applied Immunology, Delhi, Jalandhar City.

[2]DM (Neuro Consultant Neurologist Aastha Neuro Center Jalandhar City)

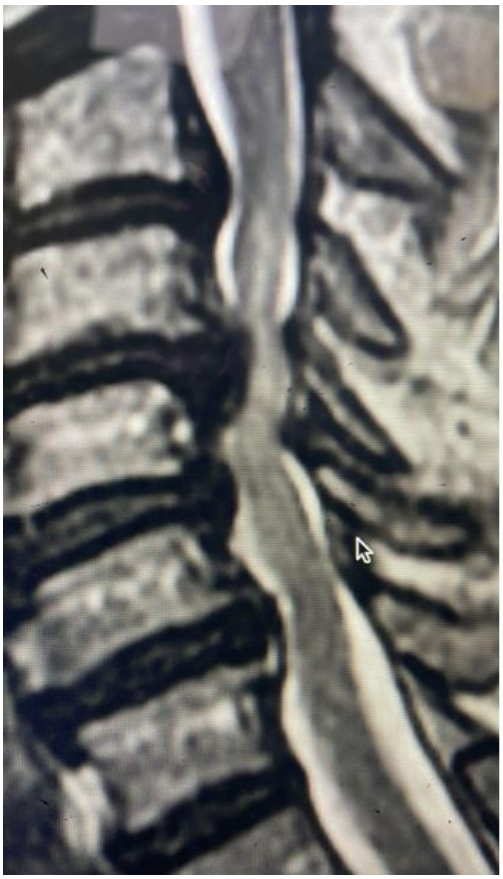
Abstract: ***A 45 years old male was diagnosed as a case of nummular dermatitis by me (1st author) seen 20 times in twenty years except 2019 - 21 (Covid period) and also examined by noted dermatologists of all institutions in Delhi. In the end of 2020, he got some pain in the legs and numbness and later some limping and the dye for diagnosis was cast and MRI for cervical spine was suggested which showed a ring of calcium deposit around the spinal cord which reduced the spinal cord to nearly half. He was operated upon in two sittings, and is well now and skin lesions decreasing, but one of the rare cases. The calcium ring could not be analysed pathological as the patient was operated at another hospital***

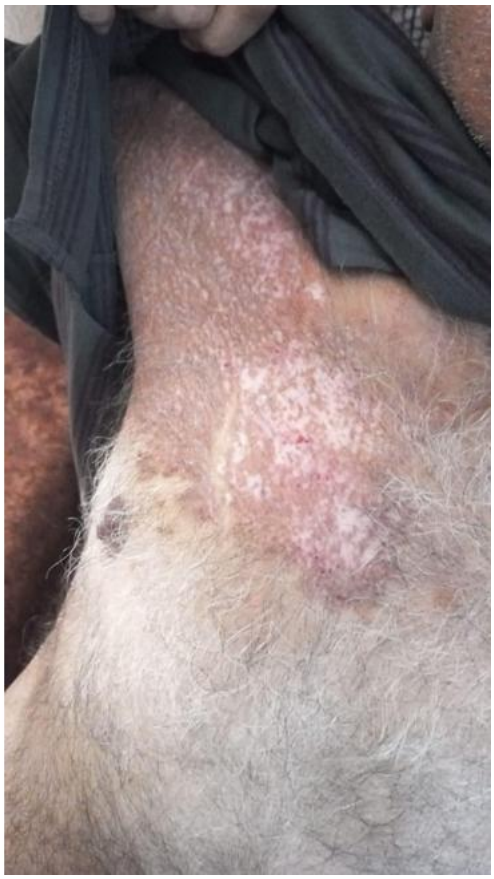
Keywords: compressive myelopathy, nummular dermatitis

1. Case History

A male patient about 45 years was first seen by me (1st author) and diagnosed nummular dermatitis and given topical steroids. Before that, he had consulted some popular dermatologists and diagnosis was the same. Patient takes alcohol 200ml for 3 to 4 times a week and smokes 5 to 7 fags per day. I have seen this patient for at least 20 times in 20 years and tried azithromycin & dapsone after G6PD deficiency test which was within normal limits. He got the whole body blood tests, all normal. In between he showed the noted dermatologists and big medical colleges, but to no effect. I could not see the patient from 2019 to 2022 due to COVID- 19.

Of late for the last 3 years he complained of pain in the lower legs and later he walked by limping & still later he could hardly walk or walk with support. Here the dye was almost cast in the direction of the diagnosis and his MRI cervical spine was done as suggested by some orthopedician as a last die effort and the MRI showed spinal cord reduced to half in size and a ring of calcium deposits all around that part of spinal cord. Two operations were conducted on the patients to remove the calcium deposit 4 months apart and now the patient is well, most eczamatious lesion healing or healed, numbness diminishing and able to walk slowly without support too; 6 months after the operation.





2. Observation

Are that the patient could not be diagnosed for 20 years by any of top dermatologists of new Delhi and myself, which is unfortunate. It is good that the patient is improving in all the parameters including numbness, eczema & walking. however from South Korea there is a retrospective study showing skin changes after spinal injury in 1408 patients and they observed infectious skin lesions, percentage wise like, fungal infections, eczema, seborrheic dermatitis & acne vulgaris to be the most common skin lesions besides some others.

3. Discussion

The relationship between skin lesion and neurological involvement is well known. Most of the times these skin lesion and neurological involvement are part of one disease like neurofibromatosis (NF1, NF2) because of involvement of a particular gene. But indirect effects of the neurological involvement due to spinal cord injury are not that well documented. Spinal cord as we all know is the seat of autonomic function of the body. Due to involvement of autonomic nervous system, It can lead to affect such as sweat gland, sebaceous gland & normal functioning of skin. Indirect effect as sensory receptors of spinal cord injury has been well documented.

The present case shows that cord compression led to calcium deposition and skin problem. The fact that they fully resolved after surgery further proves the point that the two were causally related.

Exact mechanisms of pathological damages that occur in such cases have never been documented. The present case thus provides us to look for the skin lesion in cases involving spinal cord compression or injury. Skin lesion after spinal cord compression has not been documented. However in spinal cord injuries such changes have been described in these parts distal to the injury.

Lin etal [(1)] at all have reported a high stimulation threshold and reduced aptitude of motor examination. In Spinal cord injuries have been described in these parts distal to the injury, Far away from the site of injury these likely represent transcriptic degeneration [(2, 3).]

Direct assessment of peripheral nerve morphology in spinal cord injury is not available till date. However recent uses of nerve transfer surgery in people with cervical spinal cord injury has provided a unique opportunity by providing the sample of peripheral nerve [(4, 5).] Fox etol [(5)] have described a decreased fiber density in the nerve.80% of the nerves shows abnormality most common being myeline thickening and folding, demylenation and a reduction of large unmyelinated axon density.

Local ischemeia mainly affects the spinal cord grey matter due to damage to anterior sulcal arteries and damage to spinal microvasculation within the spinal cord [(6).] Restoration of vasculation to save blood flow causes further damage by release of inflammatory cytokines such as TNF alpha, interlukins and interferones. Enhanced levels of inflammatory products may affect channel function in peripheral axons [(7).]

Immuno factors such as anti ganglioside GM 1, antibody have been shown to promote formation of antibody complement complexes that could block sodium channels to disrupt node of Ranvier [(8).] People with SCI have chronic Inflammation and immune destruction which makes them susceptible to channelopathies [(9)].

4. Conclusion

It is safe to conclude that in the present case the skin problem was directly related to spinal cord compression as is borne out by the fact that the skin lesions disappeared after the spinal cord decompression. In all such cases detailed evaluation for all the possible causes including rare causes should be looked for

References

[1] Lin SY, Macefield VG, Elam M, Wallin BG, Engel S, Kiernan MC. Axonal changes in spinal cord injured patients distal to the site of injury. Brain.2007; 130: 985 - 994. [CrossRef]
[2] Kirshblum S, Lim S, Garstang S, Millis S. Electrodiagnostic changes of the lower limbs in subjects with chronic complete cervical spinal cord injury. Arch Phys Med Rehabil.2001; 82: 604 - 607. [CrossRef]
[3] Aisen ML, Brown W, Rubin M. Electrophysiologic changes in lumbar spinal cord after cervical spinal cord injury. Neurology.1992; 42; 623 - 626. [CrossRef]
[4] Van Zyl N, Hahn JB, Cooper CA, Weymouth MD, Flood SJ, Galea MP. Upper limb reinnervation in C6 tetraplegia using a triple nerve transfer: Case report. J Hand Surg.2014; 39: 1779 - 1783. [CrossRef]
[5] Fox IK, Davidge KM, Novak CB, Hoben G, Kahn LC, Juknis N, et al. Use of peripheral nerve transfers in tetraplegia: Evaluation of feasibility and morbidity. Hand (N Y).2015; 10: 60 - 67. [CrossRef]
[6] Tator CH, Koyanagi I. Vascular mechanisms in the pathophysiology of human spinal cord injury. J Neurosurg.1997; 86: 483 - 492. [CrossRef]
[7] Allison DJ, Green LA, Gabriel DA, Roy BD, Inglis JG, Ditor DS. Elevated concentrations of circulating cytokines and correlations with nerve conduction velocity in human peripheral nerves. J Neuroimmunol.2014; 277; 134 - 139. [CrossRef]
[8] Takigawa T, Yasuda H, Kikkawa R, Shigeta Y, Saida T, Kitasato H. Antibodies against GM1 ganglioside affect K+ and Na+ currents in isolated rat myelinated nerve fibers. Ann Neurol.1995; 37: 436 - 442. [CrossRef]
[9] Allison DJ, Ditor DS. Immune dysfunction and chronic inflammation following spinal cord injury. Spinal Cord.2015; 53: 14 - 18. [CrossRef]