

## Sweating Disorder after Traumatic Brain Injury

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Received date: July 18, 2016; Accepted date: December 01, 2016; Published date: December 07, 2016

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### Introduction

Sweating Disorders after Traumatic Brain injury are common with multifactorial causes including injuries, tumours, infarcts or haemorrhages of the brain or medulla. Irritation of the central or peripheral nervous system may lead to an unexplainable spectrum of sweating ranging from anhidrosis to hyperhidrosis. Stroke in the region of the cerebral hemispheres, hypothalamus, pons & spinal cord has been known to cause hyperhidrosis. Unilateral hyperhidrosis may also be caused by disruption of pathways from Frontal Coeruleus Nucleus [1-2].

### Case Report

We present a case of 27 year old man, who presented to our hospital with severe Head injury following a road traffic accident; with Fronto-Temporal contusion. Magnetic resonance imaging (MRI) revealed a sub dural haemorrhage (SDH) & a sub arachnoid haemorrhage (SAH).

He underwent an urgent Decompressive craniotomy followed by Tracheostomy, in view of requirement of prolonged ventilation. Postoperative Glasgow coma scale (GCS) remained 8/15 (E2VtM5). He spontaneously maintained his ventilation on a T-piece.

After around 4 weeks of hospitalization the patient was noticed to have features of raised ICP (Bradycardia: HR<60/min; Blood Pressure: MAP>110) along with profuse sweating over the right side of skull (head) & face (Figure 1).

There was no ptosis, anisocoria, or changes in his respiration pattern. The patient had a left sided facial palsy. We observed a pinpoint pupil with a sluggish light reflex, without a corneal and oculocephalic reflex.

The patient was afebrile, electrolytes were normal, and cardiac arrhythmia was not observed. There was no flushing, lacrimation or vasodilation. The blood counts, renal function test (RFT) & liver function test (LFT) were within normal limits.

Starch-Iodide testing for sweating showed a zone of sweating on Rt side of face (Figure 2). MRI Brain revealed that the patient developed a dilatation of the Ventricular system and showed extensive areas of gliosis and encephalomalacia involving the left cerebral hemisphere including the left gangliothalamic complex.

MRI of the whole spine did not reveal any compression, cord transection or any other abnormality.



**Figure 1:** Starch-Iodide Test for hyperhidrosis. Anticlockwise from left top: 1. Hyperhidrosis seen on right side of forehead 2. Coating with Betadine 3. Coating with starch powder after drying of Betadine 4. Unilateral hyperhidrosis seen on the right side of forehead.



**Figure 2:** Unilateral hyperhidrosis on the right side of forehead as seen on Starch-Iodide Test for Hyperhidrosis.

Patient was taken up for placement of a VP Shunt to relieve the raised ICP. The patient's GCS subsequently gradually improved to 10/15 & his unilateral hyperhidrosis disappeared.

## Discussion

Sympathetic pathways (originating in the frontal operculum) which makes connections with the thoracic spinal cord on the contralateral side is mainly inhibitory to sweating. The increased sweating might be attributed to a lesion involving any part of this sympathetic inhibitory pathway. Unilateral hyperhidrosis has been attributed to the disruption of a sweating inhibitory pathway of cortical origin [3]. The etiopathogenesis of these lesions being multi-factorial like syringomyelia, intramedullary gliomas, vertebral tumours, structural lesions of the spinal cord or hypothalamus, head and spinal cord injury.

Unilateral hyperhidrosis has been reported post stroke affecting the cerebral hemispheres, hypothalamus, pons & even spinal cord [4,5]. Labar et al. reported that unilateral hyperhidrosis has been noted in patients with large infarctions and may be regarded as a clinical sign of severe neurological deficit and poor prognosis in stroke [6]. However, these prognostic results were not corroborated in a series by Korpelainen et al. [7,8]. Appenzeller in his case report described a patient with an opercular infarction who had unilateral hyperhidrosis, ptosis, miosis, and hemiplegia on the side contralateral to the infarction [3].

Unilateral localized hyperhidrosis has also been shown to occur secondarily to tumours of lungs involving parts of the sympathetic pathway, the phenomenon called as the Harlequin Syndrome. The lesion is assumed to be situated in the endo-thoracic fascia; here the cervical sympathetic fibres are closely related to the apex of the lung and its pleura [9,10].

The unilateral focal hyperhidrosis in our patient developed approximately 4 weeks after the injury and was associated with increased intracranial pressure (ICP). The increased ICP was managed by ventriculo-peritoneal shunt (VP Shunt), after which the unilateral focal hyperhidrosis resolved over next 1-2 days.

In our patient, there were multiple lesions which could explain the origin of focal unilateral hyperhidrosis. Based on the present knowledge of anatomic relationship and the physiology involving sweating, the focal unilateral hyperhidrosis of the right side of face in this case could have been due to the hypothalamic lesion caused by cerebral infarction in the posterior cerebral artery region.

Also the extensive gliosis and encephalomalacia involving the large sub-cortical areas on the left side of brain could have led to hypersensitive and hyper-stimulated sympathetic innervations of the contralateral right side. These hypersensitive and hyper-stimulated sympathetic innervations got stimulated due to a condition mimicking ischemic stroke because of a compromised blood (due to increased ICP). The increased ICP was controlled with a VP shunt leading to adequate blood supply thus relieving the unilateral hyperhidrosis.

The hyperhidrosis could also be as a result of paroxysmal sympathetic hyperactivity which is fairly common after TBI.

## Conclusion

Sweating Disorders after Traumatic Brain injury are common with multifactorial causes; lesions of the cerebral hemisphere,

hypothalamus, and pons have been thought to be involved in the phenomenon of hyperhidrosis. Unilateral hyperhidrosis contralateral to lesions after TBI is observed infrequently. The available data is inconclusive on the exact origin, the pathways involved and prognosis of the patient.

## Consent

Written informed consent was obtained from the NOK of patient for publication of this manuscript and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

## Competing Interests

The authors declare that they have no competing interests.

## Authors' Contribution

RM and AG performed the clinical examination, analyzed and interpreted the diagnostic findings. Conception and discussion was done by NSL. All authors read and approved the final manuscript.

## Acknowledgments

We thank our patient's NOK who gave written permission to publish his case.

No external funding was obtained.

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