Acute onset phantom pain

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Abstract

Phantom pain is severe pain feeling in an organ which is surgically removed. Examples are phantom limb pain, where the painfull limb was amputed and still the patient feels severe pain in amputed leg. This phenomenon is explained on the basis of central sensitization, (cerebral reorganization and spinal spasticity) where because of long standing pain the projection area in the brain, cerebral cortex and spinal cord, undergoes certain changes which gives continuous feeling of the pain even after that part is surgically removed from the body. Well established spinal anaesthesia in patient is a condition where all the sensation below the level of ananesthesia must lost. A patient with avulsion injury of heal after one month and prior three surgeries in the period having intense pain in heel after a completely well established spinal anaesthesia with inj bupivacain 0.5% H.

Key Word: Phantom pain, central sensitization, spinal spasticity, cerebral reorganization.

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INTRODUCTION

The concept of phantom limb pain (PLP) as being the pain perceived by the region of the body no longer present was first described by Ambrose Pare, a sixteenth century French military surgeon . Silas Weir Mitchell, a famous Civil War surgeon in the nineteenth century, coined the term "phantom limb pain" and provided a comprehensive description of this condition. It continues to remain a poorly understood and difficult to treat. Initially it was suspected that a patient with amputed parts feels pain was his own imagination. But latter on the studies proved that the feeling of such intense pain is because of some chemical and structural changes in the central nervous system because of chronic intense continuous pain signals from that part, is called as central sensitazion(spinal spasticity and cerebral reorganization). In a patient with injury to lower limbs the pain of patient must be completely relieved after successful spinal

anaesthesia. Severe pain in injured heal region after successful spinal anaesthesia is unexplainable on the neuro-anatomical and functional basis. As all senses below the spinal level must lost.

CASE REPORT

A 45 year's old male having road side accident with avulsion injury to left heal. He was known case of diabetes mellitus since last 5 years with good glycemic control. No other significant history. He was posted for the surgery and a rotational flap was planned after thorough cleaning and debridement. He was posted for the surgery for total four times in a span of one month. The spinal anaesthesia was given three times before with inj. sensorcain 0.5% heavy. With good effect. When he was posted for final surgery i.e. fourth time, spinal anaesthesia with inj sensorcain 0.5% heavy 3.5 ml. patient was having sensation of getting numb both the lower limbs and unable to move the limbs, but at the same time he was complaining severe pain in the injured heal. As the density of the block was increasing, the pain in the heal was more intense and patient was shouting that he was having very severe pain in the injured heal. Patient was sedated with inj. Midazolam 1.5 mg and inj fortwin 30 mg IV., but still patient was complaining severe pain in the heal. Only after giving inj. Ketamin 20 mg IV. The patient's pain relieved and he could sleep.

DISCUSSION

Series of mechanisms are involved in generating phantom pains and that these include elements in the periphery, spinal cord and brain. It is likely that the first events occur in the periphery, which subsequently generates a cascade of events that sweep more centrally and also recruit cortical brain structures. The latter may be responsible for the complex and vivid sensation that characterizes certain phantom pain sensations. The unraveling of neuroplastic changes in periphery, spinal cord, and brain are also reflected in many of the features seen in phantom pain phenomena. Successful spinal anaesthesia is said when all the sensation below the level is no more felt, (chemical temporary amputation) In order, the temperature, sharp and crude/crush pain, motor function and then lastly the proprioception goes. In the case, the patient is having total motor paralysis, he was not feeling any kind of sense in both the limbs except the severe pain in the injured heal. This cannot be explained on the neuro-anatomical and routine functional basis. Then what is the cause of such severe pain in successfully anaesthetized patient? In the 1990s researchers found that neuroplasticity—the ability of neurons in the brain to modify their connections and behavior—could explain pain phenomena that had been observed in association with phantom limb syndrome. Phantom limb pain was found to be explained specifically by map expansion neuroplasticity (cortical reorganization), in which local brain regions, each dedicated to performing one type of function and reflected in the cerebral cortex as "maps," can acquire areas of the unused phantom map. It can be explained on the basis of central sensitization(spinal spasticity and cerebral reorganization) and is well responded to the IV inj. Ketamin. From this can be explained the acute onset of the phantom limb pain if the pain control of the injured leg is not done in proper and efficient way in initial days.

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