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Patient born with insensitivity to pain acquires neuropathic pain following childbirth

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A medical case report of a female patient who had felt no pain since childhood but who, following childbirth, was left with a variety of pain symptoms, has given insights into the manifestation of neuropathic pain. The clinicians involved, Professor Geoff Woods and colleagues from Cambridge University Hospitals, also claim that their findings reveal a great deal about the subjective nature of pain.

The report, published on F1000Research and titled Neuropathic pain in a patient with congenital insensitivity to pain has just passed peer review. It concerns a unique case of a woman with Channelopathy-associated Insensitivity to Pain (CIP) Syndrome, who developed features of neuropathic pain after sustaining pelvic fractures and an epidural hematoma that impinged on the right fifth lumbar (L5) nerve root. These injuries were sustained during a painless labour, which culminated in a Caesarean section.

The patient had been diagnosed with CIP as child. This was later confirmed by tests showing bi-allelic mutations in the gene that codes for the voltage-gated sodium channel Nav1.7, a protein considered necessary for the sensation of pain. Testing two months after childbirth showed that her ability to sense mechanical stimulation of her feet had increased more than 10-fold compared with tests performed before her pregnancy. However, the authors note that the value of verbal descriptors is somewhat limited in a person who has never felt pain before.

She now suffers from continuous, unpleasant, buzzing sensations and electric shocks in her legs and a vice-like squeezing in the pelvis when she walks. Even though she has not reported sharp, stabbing or burning sensations, her symptoms fall under the International Association for the Study of Pain definition of pain, and she frequently used terms such as 'hurt', 'cramp' and 'aching'.

Professor Woods and his colleagues state: "The Nav1.7 channel plays a crucial role in pain transmission; however, this case shows that neuropathic pain can be initiated and maintained in its absence in humans, as well as in knockout mice. Our data provides a further basis for seeking specific molecular substrates for neuropathic pain, some of which could act as mechanistic targets for new therapies for patients with such symptoms."

This case shows that, contrary to popular belief, pain (or at least a form of it) can be induced in humans without Nav1.7 channels. This finding raises the possibility that pain-killers developed to target this channel may only be able to relieve certain aspects of neuropathic pain.

Rebecca Lawrence, Managing Director of F1000Research, said: "This fascinating case report represents important work in the field of pain transmission and ultimately, management. Through F1000Research, the authors have been able to share their findings immediately, and with the widest audience."

More information: Case Report: Neuropathic pain in a patient with congenital insensitivity to pain, f1000research.com/articles/3-135/v2

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