Using Drugs for Pain Prevention: Primary Prevention Across Areas

Introduction

Chronic pain may occur after surgery, trauma, cancer treatment or infection. In the majority of patients enduring chronic pain, a neuropathic or nociplastic component is present which increases pain severity and worsens the quality of life [8]. The transition from acute to chronic pain is a complex process involving multiple biopsychosocial mechanisms. Current knowledge favors mechanisms in relation to both peripheral (at the site of tissue trauma) and central (spinal and supraspinal) sensitization. Drugs have been used to prevent the development of chronic pain, specifically drugs which target spinal excitatory processes and/or which display anti-inflammatory properties able to modulate the release of cytokines from peripheral immune cells and central glial cells.

Drugs for Primary Pain Prevention

The largest bulk of research on the prevention of chronic pain has focused on chronic postsurgical pain, as surgery constitutes one of the common causes of pain and is often scheduled which makes preventive strategies easier to apply. Nonsteroidal anti-inflammatory drugs, gabapentinoids, ketamine and memantine have shown some but inconstant efficacy as preventive analgesics for surgical patients (Please see the IASP fact sheet “Prevention of pain after surgery”).

Complex Regional Pain Syndrome type 1 (CRPS-I) is a debilitating chronic pain condition which develops after an inciting event, often minor trauma or surgery, and involves chronic regional pain disproportionate to the causal event. CRPS-I should be distinguished from CRPS type 2, previously called causalgia, where the symptoms are due to nerve damage. The knowledge of the mechanisms of CRPS-I is constantly evolving. CRPS exhibits classic neuropathic pain characteristics but is associated with higher physical disability which considerably lengthens the time of recovery [2]. People who have developed CRPS-I may be at a high risk of recurrence following another trauma or surgical event. Vitamin C supplementation (500 to 1000mg a day for at least 50 days intake) is reported to decrease the risk of developing CRPS type I following wrist fracture and/or extremity surgery [1, 4]. Vitamin C is a well-documented antioxidant with low risk of toxicity.

Cancer treatment with chemotherapy is known to cause neuropathic pain in 25–50% of patients [5] resulting in an impairment of quality of life. Prevention of chemotherapy-induced pain with anticonvulsants or tricyclic antidepressants has so far not proved very successful [6], but there is some evidence for duloxetine or memantine [7]. Randomized controlled trials and observational studies are needed to achieve clinical significance.

Infection may also have long-standing pain consequences. For example, herpes zoster infection, a painful, blistering skin eruption in a dermatomal distribution caused by reactivation of a latent varicella
zoster virus in the dorsal root ganglia may result in chronic neuropathic pain (post-herpetic neuralgia). Although varicella and zoster vaccines have made major inroads into reducing the burden of disease globally, thus reducing the risk of post-herpetic neuralgia [9], there is currently insufficient evidence to determine the beneficial effect of other antiviral treatments [3].

Conclusion

The prevention of chronic pain remains an area of unmet clinical need. Additional well-designed studies are necessary to determine the overall effectiveness, adverse effects as well as duration of treatment and optimal dosage of preventive drugs. An important step has been done by the inclusion of chronic pain whatever its origin in the current International Classification of Diseases (ICD-11) [8]. Hopefully, this will increase focus on chronic pain and promote research in the field, including the development of preventive strategies.

REFERENCES

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