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Paravertebral block in the management of liver capsule pain after blunt trauma

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We present a case of liver capsule pain after blunt abdominal trauma. The patient was unable to tolerate patient-controlled i.v. opioids, and epidural infusion of local anaesthetic was considered undesirable because of the potential risk of complications. Pain was managed successfully with paravertebral infusion of local anaesthetic at the right T10 level. Innervation of the liver and possible mechanisms of visceral pain processing are discussed.

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Pain arising from the liver capsule is most commonly encountered in advanced malignancy. The liver capsule is a visceral structure whose afferent input to the central nervous system is probably routed via the sympathetic nervous system. Postoperative pain usually has a dominant somatic component which contributes more to the overall pain experience than input from visceral structures, and is often managed by techniques such as epidural infusion analgesia. However, there are practical problems associated with epidural infusions, such as the requirement for additional training of staff and an appropriate environment for monitoring progress, in addition to potential clinical complications, such as hypotension, urinary retention, and respiratory depression, nausea, sedation and itching if opioids are included in the infusate.

We present a case of blunt abdominal trauma resulting in a fracture in the liver parenchyma whose surgical management was conservative, but complicated by severe right hypochondrial pain, which was treated with an infusion of local anaesthetic into the paravertebral space at the right T10–11 level.

Case report

A 35-yr-old female had attended the January sales with a friend and on return to her home had been unloading her purchases from the passenger side of the stationary vehicle. Her companion inadvertently allowed the car to move backwards and the open door knocked the subject to the ground. The situation was markedly worsened when the driver, horrified at what had happened, attempted to halt the car by pressing on the footbrake, but accidentally depressed the accelerator, causing the vehicle's rear wheel to drive over the subject's abdomen while she lay recumbent on the drive.

She immediately became aware of an excruciating pain in the right side of her abdomen and chest, and found it difficult to draw breath. She was admitted to the nearest hospital, where chest x-ray confirmed that there was no lung injury or fractured ribs, but CT scan of the liver showed a fracture along the main scissura of the liver, and a small amount of free peritoneal fluid. Because there was no active bleeding, the decision was made to treat her conservatively. She was admitted to the intensive care unit and central venous pressure monitoring was commenced via a triple-lumen subclavian catheter. She remained haemodynamically stable through the night, although she continued to experience considerable pain in the right hypochondrium to the extent that respiratory function was compromised. A patient-controlled analgesia device set to deliver an i.v. bolus of morphine on demand was connected to her subclavian line, but the opioid caused troublesome nausea. The retching induced by this worsened, rather than relieved, her pain and the regimen was discontinued. The following morning she was transferred from the district general hospital to the regional hepatobiliary surgical high dependency unit.

It became clear that oral dihydrocodeine and ibuprofen 200 mg three times a day was insufficient to adequately control her hypochondrial pain, and 3 days after the initial injury, because of concerns about her inability to take deep breaths or to expectorate, the advice of the acute pain team was sought. On examination, she was clearly distressed by her pain and unable to generate sufficient respiratory

excursion to allow auscultation. Although there was some chest wall tenderness on the right side, she permitted 'springing' of her rib cage which, together with the normal chest radiograph, confirmed the absence of rib fractures. Palpation of her abdomen revealed some right iliac fossa pain, possibly caused by gravitational collection of intraperitoneal blood. There was exquisite tenderness in her right hypochondrium and epigastrium. As she had previously felt extremely nauseated with opioids, and the primary source of her pain appeared to be from the liver capsule rather than the chest or abdominal wall, the decision was made to attempt passage of a paravertebral catheter at the level of the section of the thoracic sympathetic chain from which arise the greater and lesser splanchnic nerves. This was in preference to an epidural infusion of local anaesthetic and opioid which, in the context of a liver fracture and the possibility of major haemorrhage, was considered undesirable.

She was transferred to the main operating department where, under full monitoring, the procedure of paravertebral catheter placement took place. A full aseptic technique was used. With the patient in the sitting position, the 10th thoracic vertebra was identified, and a point 3 cm lateral to the tip of the spinous process on the right hand side was infiltrated with local anaesthetic. The subcutaneous tissues were infiltrated down to the right transverse process of the 11th thoracic vertebra. A 16-gauge Tuohy needle was placed so that it touched the transverse process and carefully 'walked' superiorly, with a low friction 'loss of resistance' (Portex) device containing normal saline. Approximately 1 cm beyond the point at which bone was first encountered, a soft 'click' was perceived and a reduction in the resistance to injection of saline was noted. This indicated passage of the needle through the costotransverse ligament which forms the posterior limit of the paravertebral space. After careful aspiration to exclude inadvertent intravascular or intrapleural placement of the Tuohy needle, 1% plain lidocaine 10 ml was injected and 5 min allowed to elapse. After this time it was evident that the patient was experiencing less pain and correct positioning of the needle was presumed. A 16-gauge Portex epidural catheter was inserted and secured so that 3 cm of catheter remained within the paravertebral space. A solution of 0.1% bupivacaine was infused at a rate of 10 ml h^{-1}, this being the standard initial rate for our epidural infusions. This rate was subsequently reduced, with no reduction in the quality of analgesia, when it became apparent that the paravertebral space was insufficiently compliant to accommodate this volume and considerable 'back leakage' was occurring along the subcutaneous track of the catheter.

In contrast with her experience of the first 3 days after injury, the patient was able to sleep uninterrupted through a pain-free night and was able to eat and drink freely the following morning. The catheter remained *in situ* for 4 days, after which it was inadvertently dislodged when it caught on a corner of her bed frame while transferring to an armchair. Subsequently, it appeared that migration had occurred so that the whole catheter was intramuscular; a tender swelling of her right paravertebral musculature resulted, following which the catheter was removed intact and without further complications developing. By this time her hypochondrial pain had diminished to a level that could be managed adequately with simple oral analgesics. She was kept in hospital for a further 2 weeks for observation, as the risk of secondary haemorrhage persists for some time after liver injury.

Discussion

Paravertebral block has been most commonly reported in the treatment of post-thoracotomy pain or after breast or gall bladder surgery.[1] This case demonstrates that liver capsule pain can be treated successfully using paravertebral block.

Embryologically, the liver is formed as an endodermal thickening of the foregut.[2] A thin fibrous capsule surrounds the liver and is continuous with Glisson's capsule, which is a sheath of loose connective tissue comprising a fibrillar network of collagen fibres approximately 100 μm thick containing fibroblasts and small blood vessels.[3]

Glisson's capsule envelops the hepatic artery, portal vein and common bile duct in the porta hepatis and extends into hepatic lobules to sheathe the hepatic sinusoids, central and sublobular veins. This collagenous framework appears to support the liver parenchymal cells. The capsule may serve some role as an immunological barrier: HLA class 2 receptors have been found in the capsule[4] and animal studies have isolated mast cells containing heparin, aprotinin and histamine.[5] It also seems to play some part in intrahepatic fluid balance. The interstitial fluid of the liver, in addition to being drained by the lymphatics, may be dispersed across the capsule into the peritoneal cavity.[6]

The liver and capsule are innervated by sympathetic and parasympathetic nerves via the hepatic plexus, whose fibres enter the liver surrounding the hepatic artery, portal vein and bile duct at the porta hepatis and run with the vessels within the liver structure to the portal triads.[7] The sympathetic fibres arise from the thoracic sympathetic chain and reach the liver via the greater and lesser splanchnic nerves and the coeliac plexus. The parasympathetic fibres derive from the right and left vagus nerves.[7] The precise innervation of the hepatic infrastructure is not well documented. However, presumptive sensory nerve endings have been detected surrounding the central veins and bile ducts within the lobules.[7] Studies have demonstrated extensive parenchymal innervation and it has been postulated that adrenergic, cholinergic and other peptidergic neurones register information from osmoreceptors, baroreceptors, and ionic, metabolic and nociceptive receptors.[7]

From the observations of William Harvey and Charles I on the exposed heart of the young Viscount Montgomery in the 17th century, it was known that visceral organs are

insensate to normal stimuli such as touch and pinprick. Although visceral nociceptors have been postulated, there is scant evidence to support their existence. Animal studies suggest that excessive afferent activity after chemical irritation or distension of hollow viscera may be assimilated and interpreted as pain.

Perception of visceral pain may depend on the firing rate of visceral afferent fibres (intensity coding). It may be that several types of sensory receptor are present in varying numbers in different viscera. Specific high or low threshold afferents and intensity-coding afferents may be stimulated differentially depending on the type of stimulus.[8] It has been known for many decades that these fibres appear to travel with the sympathetic efferent neurones, reaching the spinal cord via the sympathetic chain, with identifiable root levels innervating the various viscera.[9]

At the level of the spinal cord, somatic and visceral afferent fibres appear to be arranged differently. In the guinea pig spinal cord, nociceptive somatic afferents terminate in the dorsal horn laminae I and II, forming a concentrated terminal plexus confined to that region. In contrast, terminals of visceral afferents are distributed along several segments of the spinal cord. Visceral fibres bifurcate at the point of entry of the dorsal root into rostral and caudal branches, each of which extends for two or three spinal segments. Collateral branches spread to other areas in the spinal cord, although the bulk (60%) of the terminations occur in lamina I. There appear to be numerous collateral somatosensory projections.[10] This diffuse organization of fibres, together with the many possible connections, may help explain phenomena such as referred pain, somatic motor responses to visceral pain (such as abdominal wall spasm–'guarding') and the often poorly localized nature of visceral pain itself.

In the rat, lamina I of the entire spinal cord projects supraspinally to the parabrachial nucleus at the level of the pons. This is connected to the hypothalamus, amygdala and ventrobasal thalamus. Although many neuropeptides have been shown to influence neuronal activity in both excitatory and inhibitory capacities, the principal mediators of the flow of visceral information to the thalamus appear to be glutaminergic *N*-methyl-D-aspartate receptors.[11] There is a separate pool of adrenergic and GABAergic neurones that control the 'signal-to-background' ratio which can enhance the impact of specific visceral signals.[11] Despite these advances in our knowledge, there is considerable uncertainty regarding the processing of visceral pain messages in humans.

The technique of paravertebral block has been reviewed comprehensively.[1] Placement of a catheter in the paravertebral space at the T10 level on the right hand side would be expected to enable anaesthesia of both the sympathetic chain and the greater and lesser splanchnic nerves. In our case, it appears that theory and practice combined to produce useful analgesia while avoiding the potential pitfalls associated with epidural infusion analgesia and the toxicity associated with systemic opioid administration.

References

1 Richardson J, Lonnqvist A. Thoracic paravertebral block. *Br J Anaesth* 1998; **81**: 230–8

2 Collins P. Embryology and development. In: Bannister L, ed. *Gray's Anatomy*, *38th* Edn. London: Churchill Livingstone, 1995; 187

3 Ohtani O. Three-dimensional organisation of the collagen fibrillar framework of the human and rat livers. *Arch Histol Cytol* 1988; **51**: 473–88

4 Barbatis B, Kelly P, Greveson J, Heryet A, McGee J. Immunocytochemical analysis of HLA class II (DR) antigens in liver disease in man. *J Clin Pathol* 1987; **40**: 879–84

5 Shikimi T, Kabayashi T. Contents of aprotinin, heparin and histamine in mast cells from bovine liver capsule. *Agents Actions* 1986; **18**: 325–8

6 Barrowman JA. Hepatic lymph and lymphatics. In: McIntyre N, Benhamou JP, Bircher J, eds. *Oxford Textbook of Clinical Hepatology*, vol. I, sections 1–13. Oxford: Oxford University Press, 1992; 38–9

7 Hartmann H, Beckh K. Nerve supply and nervous control of liver function. In: McIntyre N, Benhamou JP, Bircher J, eds. *Oxford Textbook of Clinical Hepatology*, vol. I, sections 1–13. Oxford: Oxford University Press, 1992: 93

8 Ness T. Historical and clinical perspectives in visceral pain. In: Gebhart GF, ed. *Visceral Pain*. Seattle: IASP Press, 1995; 3–24

9 Cervero F. Mechanisms of visceral pain: past and present. In: Gebhart GF, ed. *Visceral Pain*. Seattle: IASP Press, 1995; 26–40

10 Sugiura Y, Tonosaki Y. Spinal organisation of unmyelinated visceral afferent fibres in comparison with somatic afferent fibres. In: Gebhart GF, ed. *Visceral Pain*. Seattle: IASP Press, 1995; 41–59

11 Cechetto D. Supraspinal mechanisms of visceral representation. In: Gebhart GF, ed. *Visceral Pain*. Seattle: IASP Press, 1995; 261–281