Rylova, et al.: Intravenous lidocaine in COVID‑19 infection

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...for our approach whereby the optimum effect of lidocaine on pain occurs with 24–48 h of an infusion.

With concerns about potential lidocaine toxicity in this critically ill patient, and as we were not able to readily follow serum lidocaine levels at our institution, we chose an intermittent instead of a continuous infusion. We did not observe any clinical signs of lidocaine toxicity. We noted an abrupt decline in the inflammatory markers between day 6 and day 8 after admission to the ICU, and we discontinued the infusion after a combined 48 h of lidocaine treatment. Han et al. did not describe such an acute decline in CRP levels following cytokine profiles in 14 critically ill patients with COVID‑19 disease during their hospitalization.

It is conceivable that an association between recovery of inflammatory markers and lidocaine treatment may exist. While the reasons for our observation are likely multifactorial, it merits further exploration.

Auto‑destructive inflammation is a key feature during severe COVID‑19 infection. Lidocaine with its anti‑inflammatory properties may mitigate the inflammatory response and possibly have additional antiarrhythmic, analgesic, antithrombotic, and sedative benefits. We describe a case with intermittent intravenous lidocaine administration for severe COVID‑19 disease with successful patient outcome.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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Letters to Editor

Ultrasound‑guided glossopharyngeal nerve block: Description of a new technique

Dear Editor,

Eagle syndrome is frequently treated with a combination of drugs (nonsteroidal anti‑inflammatory drugs, anticonvulsants, anti‑depressants) and glossopharyngeal nerve block (GPNB) which is usually performed by landmark technique.[1]

Recently ultrasound‑guided nerve blocks in comparison to landmark techniques have gained popularity due to various advantages like visualization of passage of block needle in real time, visualization of vessels, and requirement of lesser volume of local anesthetics. Glossopharyngeal nerve is a very small nerve and is not visible on ultrasound and thus ultrasound‑guided GPNB (UGPNB) has not been explored. A previous cadaver study blocked the nerve distally in its course; however, this has not been validated in human patients.[2]

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GPN lies posterior to internal carotid artery (ICA) at submandibular region.[3] As pulsations of ICA would be easily
identifiable on ultrasound, we are proposing a new technique of UGPNB wherein LA can be deposited posterior to ICA at submandibular region for eagle syndrome. Here we describe the technique in ten patients of eagle syndrome.

All non-obese patients with NRS ≥5 due to eagle’s syndrome were placed in supine position. Neck was turned to opposite side and ultrasound neck scan (FUJIFILM SonoSite Edge, Linear probe 13–6 MHz) was done from base to identify common carotid artery and IJV which was confirmed with color Doppler. Common carotid artery was traced upwards till bifurcation to ICA and external carotid artery (ECA). ICA was then traced till submandibular region of neck (ICA lies posterior and deeper, ECA anterior and superficial). If delineation of vessels was difficult with linear probe, a curvilinear probe (2-5 MHz) was used. Area posterior to ICA and probable path of needle trajectory (in-plane or out of plane) was then scanned for vessels. Shortest avascular path was chosen as the final needle path for the block [Figure 1]. Block was performed without prior local anesthesia with a 26-gauze hypodermic needle of length 3.5 cm with 10 cm extension flushed with saline.

If posterior ICA or both needle trajectories were vascular, hydrodissection was used to push the vessels away. If not successful, needle tip was placed either anterior or above ICA, whichever was avascular. Block was performed after negative blood aspiration with 2 ml of 0.5% bupivacaine 7.5 mg with 20 mg depomedrol (methylprednisolone injectable suspension) and flushing with saline. Drug spread was confirmed.

In seven patients’ drug was deposited at desired location. Drug was deposited anterior to ICA in one patient and above ICA in two patients. NRS decreased to below 2 in all patients from a baseline of 7 after 30 minutes of block and remained less than 3 in all patients for one week [Table 1].

In seven patients, a curvilinear probe was used and in six patients out of plane (OOP) needle trajectory. All blocks were performed by consultant JP who is well versed in ultrasound-guided blocks. For a novice pain physician, OOP needle trajectory would be challenging and should be undertaken with caution. Hoarseness and facial palsy were mild, non-distressing and self-remitting [Table 1].

Limitations were non-visualization of GPN posterior to ICA at submandibular region however pain relief points to effectiveness of this approach.

To conclude, UGPNB in patients of eagle’s syndrome can be performed in majority patients by a curvilinear probe with out of plane technique by placing LA posterior to ICA at highest submandibular region on affected side.

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Nil.

**Conflicts of interest**
There are no conflicts of interest.

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**Table 1: Characteristics of UGPNB**

| Probe | Needle trajectory | Needle tip | Pre procedure VAS | VAS 30 mts after block | VAS after 1 week | Complications |
|-------|-------------------|------------|-------------------|------------------------|------------------|---------------|
| C     | IP                | Lat ICA    | 7                 | 2                      | 1                | H             |
| C     | IP                | Med IC     | 5                 | 0                      | 1                | H             |
| C     | OOP               | Lat ICA    | 6                 | 1                      | 2                | -             |
| C     | OOP               | Above ICA  | 7                 | 0                      | 2                | FP            |
| C     | OOP               | Above ICA  | 6                 | 0                      | 1                | H             |
| C     | OOP               | Lat ICA    | 7                 | 0                      | 1                | H             |
| C     | OOP               | Lat ICA    | 6                 | 1                      | 1                | -             |
| L     | IP                | Lat ICA    | 7                 | 1                      | 2                | -             |
| L     | OOP               | Lat ICA    | 5                 | 0                      | 2                | -             |
| L     | IP                | Lat ICA    | 5                 | 0                      | 1                | -             |

C: Curvilinear/L: Linear/IP: In Plane/OOP: Out of Plane/ICA: internal carotid artery/IJV: Internal Jugular Vein/H: Hoarseness/FP: Facial Palsy
Dear Editor,

Spontaneous intracranial hypotension (SIH) is also referred to as low CSF pressure headache or post-dural puncture headache. These clinical situations will produce symptoms that include postural headache, neck stiffness, nausea, and vomiting, photophobia, anorexia, vertigo, tinnitus, and diplopia. The headache tends to be localized to the occipital or frontal regions.

Post-dural puncture headaches are mostly an iatrogenic complication from either lumbar puncture or neuraxial anesthesia. Patients with post-dural puncture headache can be treated with a epidural blood patch, though there have been reports of using dextran 40, hetastarch, fibrin glue, gelatin, or cryoprecipitate. This case describes a young male with no significant medical history who developed spontaneous onset of bilateral frontal headache, neck pain, nausea, and vomiting for several weeks. He did not report any previous history of spinal or neuraxial procedures. On presentation to our institution, a computed tomography (CT) angiogram was normal and then a lumbar puncture was performed with a 20‑gauge spinal needle showing increased red blood cell count. The headache became postural in quality and worsened following the lumbar puncture. The patient was reevaluated in the emergency department 9 days later and his physical exam showed generalized hyperreflexia and downward Babinski reflex bilaterally. A CT head with contrast was ordered at this time and showed new bilateral fronto-parietal subdural hematomas. A CT angiogram showed cerebral edema and impending tonsillar herniation. No direct CSF pressures were obtained. Due to persistent symptoms, the patient was scheduled for an epidural blood patch, rare use of this treatment modality for this pathology.

After the risks and benefits were explained in detail including the possibility of a worse neurological outcome, informed consent was obtained. The epidural blood patch was placed at the lumbar L4‑L5 interspace using the standard fluoroscopic technique. Once the procedure was completed, the patient began to verbalize and reported nearly immediate resolution of the headache. A repeat MRI of the brain and cervical spine 4 days after the epidural blood patch showed alleviation of impending herniation.

SIH is thought to be caused by CSF leakage through small dural tears, reduced CSF production, or hyperabsorption of CSF. There is scientific data to support the dural tear theory. Individuals with certain medical conditions are more likely to have SIH; these include Marfan syndrome, Ehlers Danlos syndrome, neurofibromatosis, and disc disease. Management of a patient with SIH includes caffeine, bed rest, abdominal binder, steroids, continuous saline infusion, and epidural blood patch. Spontaneous resolution of this condition can take weeks to months.

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