Effect of intra-arterial injection of lidocaine and methyl-prednisolone into middle meningeal artery on intractable headaches

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Abstract

The present report describes the effect of intra-arterial injection of a dose of 40 mg lidocaine and 20 mg methylprednisolone into the middle meningeal artery of two patients suffering from severe headaches. The effect of injection of lidocaine and methylprednisolone was short lasting with effect manifesting within 5 min and lasting 5–8 h after injection. Both patients reported improvement in headache intensity after 24 h post-procedure. Intra-arterial injection of lidocaine and methylprednisolone may represent another treatment strategy for headaches not responsive to standard treatment.

Introduction

Approximately 17% of the U.S. population report severe headaches within the preceding year [1] New interventions such as intravenous anesthetic agents alone [2,3] or in combination with steroids [4,5] have been used for treatment of severe headaches. We report the results of intra-arterial injection of lidocaine and methyl prednisolone in the middle meningeal artery, the primary supply to cranial dura mater, through selective catheterization on headache intensity in two patients.

Case 1

A 66-year-old man with history of migraine headaches presented with 1 day history of acute onset right-sided severe headache. Patient had no response to transdermal fentanyl patch (12 µg/h) which typically alleviated his migraine headaches. He also complained of nausea but no visual symptoms. Previous history was significant for chronic hypertension, hyperlipidemia, and active cigarette smoking. Patient had no neurological deficits on examination. A computed tomographic scan demonstrated right temporal lobe intraparenchymal hemorrhage. Initial blood pressure was 183/124 mm Hg. Patient was admitted to Intensive Care Unit and started on intravenous nicardipine infusion to reduce and maintain systolic blood pressure to <160 mm Hg. Patient’s headache continued despite use of transdermal fentanyl patch 12 µg/h every 48 h, oxycodone 10 mg orally every 6 h as required, and intermittent IV injection of morphine sulphate (2–4 mg). A six-vessel cerebral angiogram was performed to evaluate for presence of any arteriovenous malformation or intracranial aneurysm. The right external carotid artery was catheterized for selective angiography to exclude the presence of any dural arteriovenous fistula. A microcatheter was placed into the right middle meningeal artery (see Figure 1). An infusion of lidocaine and methylprednisolone was undertaken as described below. Patient reported improvement in headache from a
self reported intensity of 10 (on Visual Analog scale) to 5 after 5 min of lidocaine injection. At subsequent interview at 2.5 h post-procedure, patient reported that headache was definitely better than pre-procedure headache with intensity rated at 6.5. Patient reported recurrence of headache at 8 h with intensity rated at 8. After 24 h, patient reported an improvement in headache with intensity rated at 5. Patient appeared to be more comfortable and was discharged. On the day of discharge, patient was resting comfortably but denied any persistent headache.

Case 2

A 33-year-old woman presented with ongoing severe left sided headache for the last 4 days. She described the headache as a combination of pressure and throbbing, radiating from posterior aspect of left scalp to left side and periorbital region. She had a history of left intra-parenchymal hemorrhage requiring hospitalization about 17 months ago. Patient had residual headaches since the occurrence of intra-parenchymal hemorrhage requiring hospitalization about 17 months ago. Patient had residual headaches since the occurrence of intra-parenchymal hemorrhage requiring hydrocodone-acetaminophen 5–500 mg orally every 4–6 h as required, morphine 30 mg orally three times daily, and sumatriptan 100 mg orally as required. Patient had drift in both right upper and lower extremities and partial right hemisensory loss which were residual deficits of previous intra-parenchymal hemorrhage. A magnetic resonance imaging performed demonstrated encephalomalacia of posterior frontal lobe consistent with previous hemorrhage. Patient was started on additional oxycodone 5 mg orally every 8 h. A lumbar puncture was performed on the day of admission and cerebrospinal fluid analysis did not demonstrate any evidence of subarachnoid hemorrhage. A six-vessel cerebral angiogram was performed on the day of admission to evaluate for presence of any arteriovenous malformation or intracranial aneurysm. The left external carotid artery was catheterized using a 5F diagnostic catheter. Images were obtained in anteroposterior and lateral planes and absence of any dural arteriovenous fistula or anastomoses between external and internal carotid artery branches was confirmed. A single lumen microcatheter was advanced over a 0.014 in. microwire through the diagnostic catheter into the external carotid artery. Under fluoroscopic guidance, the microcatheter was advanced through the proximal internal maxillary artery and into the middle meningeal artery. A microcatheter injection was performed in both anteroposterior and lateral planes to visualize the dural branches of the middle meningeal artery and absence of any contribution to ophthalmic or intracranial arteries. A dose of 40 mg lidocaine (2 mg/ml dilution in normal saline) injected in 10 mg doses was administered over 5 min into the middle meningeal artery. Subsequently 20 mg methylprednisolone (4 mg/ml dilution in normal saline) were injected over 5 min into the middle meningeal artery. Heart rate and single lead EKG were continuously monitored and blood pressure was monitored using automated cuff every 3 min. An abbreviated neurological examination was performed after first 10 mg injection and at the completion of procedure using the endovascular procedure-specific scheme which evaluates six aspects of neurological function, including language, gaze deviation, visual fields, cranial nerves, and the function of the upper lower extremities (left and right) as described previously [6].

Figure 2. A lateral view of opacification of branches after contrast injection from the microcatheter placed into the left middle meningeal artery.
Discussion

Activation of trigeminal afferents innervating the dura mater has been associated with headache in migraine and post-surgical procedures [7–9]. Activation of perivascular unmyelinated C fibers and catecholaminergic nerve fibers has been implicated in origin of headache [10,11]. There is evidence that dural application of lidocaine and related compounds can reduce the evoked responses of neurons to dural stimulation [12], and cause long-lasting sensory blockade and subsequent amelioration of pain [13]. The perivascular location of involved nerve fibers increases the likelihood of selective dural anesthesia by intra-arterial delivery of anesthetic agents. Middle meningeal artery is the predominant source of blood supply to the dura [13,14]. The middle meningeal artery divides into frontal, parietooccipital, and posterior temporal branches which are 400–800 µm in diameter [14]. Injection of anesthetic agent such as short-acting barbiturates or lidocaine into the middle meningeal artery prior to embolization in an arterial feeder of a meningioma has been routinely performed [15,16]. Transient visual loss can be seen if lidocaine reaches the opthalmic artery through orbital collateral routes including meningo-ophthalmic artery, and superficial recurrent meningeal branch of the lacrimal artery, and the anterior falcine artery, anastomosing directly with the branches of the middle meningeal artery [17]. Temporary paresis of facial nerve can be seen if lidocaine reaches the facial nerve through the petrous branch of the middle meningeal artery [17]. Therefore, lidocaine injection into the middle meningeal artery is expected to result in temporary anesthesia of afferent nerves supplying the dura.

The observation is preliminary and we are unable to exclude a placebo effect in the absence of control patients [18,19]. Furthermore, assessment of pain is highly subjective and the pain scales are of most value when looking at change in pain intensity within individuals [20]. The effect of injection of lidocaine and methylprednisolone was short lasting. Both patients reported improvement but not resolution of headache even after 24 h. It remains unclear whether such an effect was a benefit related to interruption in continuous headaches, effect of methylprednisolone, or a result of spontaneous resolution independent of procedure. It is possible that the benefit of anesthetic injection into the middle meningeal artery maybe more pronounced with longer acting anesthetic agents.

References

18. Williams KA, Harden N. 2011;Managing the placebo effect:
