Egyptian Journal of Anaesthesia (2016) 32, 397–402

HOSTED BY

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Egyptian Society of Anesthesiologists

Egyptian Journal of Anaesthesia

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www.sciencedirect.com

Research Article

Excessive sweating following intrathecal μ agonists: Effective atropine management

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Received 10 January 2016; revised 26 March 2016; accepted 3 April 2016
Available online 20 May 2016

KEYWORDS

Profuse sweating;
Intrathecal morphine;
μ agonist;
Atropine;
Temperature

Abstract Many thermal distortions may accompany the profound extended analgesia of intrathecal μ agonists. Hypothermia is the commonest, but a syndrome including a profuse sweating was also reported. Conservative management for this sweating extends patient suffering more than 6 h. Other treatment options showed variable success rates. In a case series, atropine sulfate showed effective sweat suppression following intrathecal morphine 0.3 mg or fentanyl 25 mcg. Treatment options and possible mechanisms of sweating are reviewed in relation to opioids, regional block and general patient factors.

1. Introduction

Hypothermia, commonly associates the analgesia of intrathecal (IT) μ agonist opioids [1]. In addition, a profuse sweating sometimes appears within a syndrome that starts about three hours of injection, also comprising, feeling hotness, lethargy, itching, nausea, vomiting, hypothermia without shivering and sometimes dysphoria [2–8]. However, patients are hemodynamically stable with normal electrolytes, complete blood count, ECG, troponin, serum cortisol and thyroid stimulating hormone and not responding to aggressive warming measures [3,7].

The mechanism of this syndrome is not clear. Active treatment options included naloxone [3] and lorazepam [4,6,7]. Atropine is suggested as an option in this study.

1.1. Thermal effects of anesthesia

General [9] and regional anesthesia [10], as well as IT opioids [11] highly increased the thermostatic inter-threshold range thus intensifying hypothermia [12]. Opioids inhibit the warm-sensitive neurons in the medial preoptic area of the hypothalamus which is the principle center for thermoregulation [13]. IT morphine (Mo) can reach the opioid receptors in the hypothalamus through cephalad spread within CSF causing new setting of thermoregulatory center that the inter-threshold range was above this new upper temperature threshold generating sweating [5]. Nevertheless, opioids exert a range of thermoregulatory effects, depending upon various factors, such as animal species, age, circadian rhythms, route of administration and dose [14].

2. Case series and results

The first case involved in this study (Table 1, case1) managed conservatively only, due to lack of experience with this phenomenon. Knowledge regards this syndrome stimulated

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Peer review under responsibility of Egyptian Society of Anesthesiologists.

http://dx.doi.org/10.1016/j.egja.2016.04.003

Table 1 Clinical, surgical and anesthetic data of case1 – conservatively treated.

Patient Age/sex	Medical problems	Surgery/duration	IT drugs	Symptoms & signs	Onset after spinal	Treatment	Treatment efficacy
Case 1 59 y/f	DM, HTN, IHD, LBBB AF	Total knee arthroplasty/3.5 h	20 mg Bup +0.3 mg Mo	Hypotension	10 min	Ephedrine (total 20 mg)	(+)
				Itching	30 min	Diphenhydamine 45.5 mg	(−)
				Sweating, hotness, weakness, BP 130/80, HR 70/min 35.8 °C	2.5 h	Conservative	(−) (Sweating for 12 h)
				Hypoglycemia (RBS 75 mg)	2.5 h	5% dextrose (Still sweating)	(+) (RBS 175 mg)
				N&V	2.5 h	Metoclopramide, granisetron	(±)

F: female, y: years, DM: diabetes mellitus, HTN: hypertension, IHD: ischemic heart disease, LBBB: left bundle branch block, AF: atrial fibrillation, h: hours, IT: intratheal, Bup: Bupivacaine, Mo: Morphine, RBS: random blood sugar, N&V: Nausea & vomiting. Treatment Efficacy for controlling the symptoms: (+) Effective, (−) not effective, (±) partial control.

prospective search about further cases for trial evaluation of atropine as a treatment for this sweating. Over two years (2014–2015) observation attained more three cases (Table 2, cases 2, 3, 4) among 217 consecutive patients underwent lower limb orthopedic surgery under spinal anesthesia using IT: 15–20 mg 0.5% heavy bupivacaine plus Mo 0.2–0.3 mg in 32% of patients, or fentanyl 25 micrograms (mcg) in 68% of patients. Mean age was 34 years (range: 17–72). Results revealed that the incidence of the profuse sweating syndrome in these patients was 1.8%. Sweating started about three hours after spinal injection. This syndrome was detected intra operatively in all cases except case 4 (Table 2) that was consulted six hours postoperatively due to profuse sweating, vomiting and urine retention. Atropine was tried in the last 3 cases as 0.5 mg increments. Atropine 1 mg was promptly effective within minutes in discontinuing this sweating without recurrence. Subsequently temperature was rising slowly. Tables 1 and 2 display the clinical, surgical and anesthetic data of these cases.

3. Discussion

This study showed that atropine controlled the sweating following IT Mo or fentanyl. The incidence of the excessive sweating in this study was 1.8%. Erdine et al. reported the same rate of sweating after IT morphine [15]. Hess et al. reported an incidence about 7% [4], the same as Paice et al. [16]. This rate may be up to 9% with both transdermal fentanyl and oral Mo [17] and up to 40% in patients on long-term methadone treatment [18]. The treatment options and possible etiologies will be discussed.

3.1. First: Treatment options

3.1.1. Atropine

Atropine effectively controlled the profuse sweating following IT Mo or fentanyl in this study. The nerve supply of sweat glands is mainly cholinergic [19] with a few adrenergic terminals [20]. Acetylcholine is the main pre and postganglionic transmitter of the sympathetic nervous supply of the sweat glands [21], and hence the rationale of using atropine as an anticholinergic greatly attenuates or abolishes sweating [22]. Atropine suppressed thermal and non-thermal sweating where both types are cholinergic [23]. The cholinergic system is also involved in Mo-induced hyperthermia [24].

Similarly, the anticholinergic hyoscine controlled the opioids – induced sweating [25]. On the contrary, IT neostigmine 100 mcg, produced profuse sweating, agitation, nausea and vomiting [26].

Atropine has the advantage of high end-organ efficacy whatever the mechanism of sweating. The tachycardia combining atropine may be beneficial against the high degree of cardiac vagal activity associated spinal anesthesia [27] or the parasympathetic predominance associated Mo as confirmed by heart rate variability [28].

3.1.2. Naloxone

Thermal changes of μ agonists are opioid receptor effects. Naloxone antagonized sweating, hypothermia, sedation, pruritus, nausea and vomiting [29,30]. On the other hand, naloxone