






Treating Dog Bites in The Emergency Department

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Abstract

Trauma injuries inflicted by animal bites account for a significant number of attendances at Emergency Departments (EDs) in the UK and frequently these presenting patients are assessed and treated in a nurse-led unit. A significant component of the assessment and treatment requires prescribing appropriate analgesia to permit adequate wound assessment and debridement as well as pain relief, along with assessment of the possible risk posed by infection and the subsequent treatment with antibacterial agents – whether prophylactically, or to treat active infection. This report provides an overview of bite injuries presenting to ED, followed by a discussion and review of the pharmacotherapeutics of co-amoxiclav and lidocaine since these are two of the medications frequently used in the ED for initial treatment and emergency care of a first presentation dog bite to the hand.

Keywords: dog bite, co-amoxiclav, antibiotic, lidocaine, analgesia.

Introduction

Fundamentally, the scientific foundation that underpins clinical practice is understanding the connection between the patient, their disease, and ascertaining the most suitable way of curing, alleviating, or averting mortality [1]. Frequently medications are prescribed to achieve one or more of these objectives. Pharmacodynamics is the process of how the drug affects the body, and pharmacokinetics looks at how the body acts on the drug [2]. An understanding of these processes forms the basis for how, when, and why we administer drugs in any clinical situation. Most drugs work by mimicking or replacing natural chemicals, or by stimulating or depressing normal cell activity. Not all drugs are for curative effect, some are prophylactic, others are chosen for instant pain relief or to allow exploration or procedures. The combined antibiotic amoxicillin and clavulanic acid (co-amoxiclav) and the local anaesthetic lidocaine (previously named lignocaine) are commonly considered in dog bite presentations, generally, for prophylactic use and to aid exploration and debridement, but also in active infection and pain management. For the purpose of this report, the presentation of the dog bite and associated pain and infection pathways will be discussed,

along with a critical appraisal of the use of these drugs in treating a dog bite to the hand that presents to ED.

Presentation of bite trauma injuries to the ED

Within a level 1 trauma setting in the Northern Ireland, minor injuries and ailments which are deemed stable and appropriate, are referred directly from ED triage for treatment in an emergency nurse practitioner (ENP) led area. As well as fractures, strains, sprains and burns, bites are regularly assessed in this area. These are primarily, but not exclusively attributable to human, cat and dog. Dog bites account for approximately 250,000 ED attendances in the UK every year [3]. This figure is perhaps unsurprising when the number of canines in the United Kingdom populous is considered: 24% of families in the UK are dog owners contributing to a dog population of about 8.5 millions canines [4]. Kasbekar, Garfit [5] highlight the cost to the NHS for admissions secondary to dog bites in 2010 was approximately 3.3 million pounds for 6,000 patients.

Whilst cat bites in the ED are historically more synonymous with complicated bite injuries due to their perceived higher risk of infection, and human bites are feared by patients because of their perceived risk of transmissible viruses, it is

the dog bite that accounts for most ED biting offences [6]. Dogs have larger teeth, and are usually more forceful than other perpetrators, resulting in crush injuries and lacerations. Pradyumna Raval, Wasim Khan [7] asserts that dog-related injuries and attacks are usually high energy. Biting is generally associated with a struggle to remove the effected part of the body forcefully from a dog [8], resulting, potentially, in what Morgan and Palmer [3] refer to as “the hole and tear effect”. Whilst many bites are unprovoked and occur in the community, most bites occur in the home or by a known dog, therefore, it can be assumed that petting, feeding, and breaking up fights are generally associated with the attack. This correlates with the typical location of the bite in humans; children are more likely to be face-to-face with the pet, and are associated with facial injury [9]. Adults however, are more likely to be at limb level. The literature supports this, as most adult associated dog bites are to the hand or wrist [10-12]. Most bites are minor, self-treated, and under reported [13]. Nevertheless, the assumption by patients that most bites are shallow leads to a delay in seeking medical advice [14] meaning cases of animal bites can be complicated by wound contamination by oral bacteria caused by crushing of the tissues. This is particularly problematic in the hand as scar tissue formation, secondary to suppuration, can cause severe functional impairment [15]. Furthermore, the infection rate is high for animal bites of the upper limbs compared to other areas [16]. Wieschhoff, Sheehan [17] state that traumatic hand and finger injuries account for a considerable number of ED attendances each year.

Pathophysiology associated with bite trauma injuries

The hand is an intricate anatomical structure. Aside from five metacarpals and associated phalanges, there are also two congruent rows of carpal bones and an underlying complex construction of tendons, muscles, blood vessels and nerves. The intrinsic and extrinsic muscles also house an elaborate and structured organisation of tendons which allow complex hand movements and facilitate dexterity. Due to numerous small compartments and fine tissues covering the bones and joints, there is a higher risk of infection to the hand from dog bite wounds [18]. Naito, Sugiyama ¹⁴ highlights that the prognosis for patients with bite injuries involving joints is poor, and is associated with complications.

According to Tabaka, Quinn [19], the primary morbidity associated with dog bites is infection. The primary presentation, however, is likely to be pain. Not all dog bites which present to the ED are infected. Most presentations are early therefore symptoms of infection have not yet emerged, or are simply low risk. Hand bites pose a higher risk for infection. According to Linton, Potgieter [20], each presentation should be considered in a systematic manner and they reiterate that caution must be taken in a joint injury. Hands present the highest risk of secondary presentation, which can subsequently lead to infectious

complications [21]. Initial presentation does not always merit antibiotics. In the UK, NICE Guidelines [22] advise that a thorough assessment, including a full tendon check, is carried out.

Pre-existing comorbidities, such as history of a splenectomy, cirrhosis, and diabetes, as well as all bites to the hand and all animal related puncture wounds, predetermine if prophylactic antibiotics are required in a dog bite that otherwise appears clean and without signs of infection [22,23].

Pain commonly accompanies infection caused by pathogens since nociceptor neurons are directly activated [24]. Pain is also one of the main reasons that people seek medical care in the ED [25]. A dense network of sensory nerves innervates peripheral tissues which, in the case of a breakage to the skin, can be exposed to bacterial pathogens [24]. While it may indicate infection, it may also simply be the response to trauma. Pain secondary to inflammation is thought to be a nociceptive response activated by proteins and lipids such as cytokinase, and prostaglandins, as well as amines and protons [26]. Pain is a complex phenomenon; it can be considered a defence mechanism which warns the brain of potential damage about to occur. Nociception is defined as the perception of pain by sensory receptors (called nociceptors) which are part of the peripheral nervous system (PNS) [27]. Nociceptive pain can be visceral or somatic. The disruption of somatic pain receptors in the skin and superficial fascia is in keeping with the anatomy of a dog bite [28]. Sorokin [27] and D'Arcy [29] further report that in a wound the pain stimulus is first perceived in the nerves closest to the injury. A-fibres are large nerve fibres coated in myelin which serve to rapidly transmit nerve impulses, and are associated with localised pain. C-fibres are less myelinated, therefore the pain impulse is conducted far slower [30]. This pain transmission is thought to take part in 4 stages: transduction, transmission, perception and modulation [27,28,31]. This can be summarised as a noxious stimulus converts energy into a nerve impulse which is detected by nociceptors (transduction). The neural pain signal is transmitted from the PNS to the central nervous system (CNS). This pain impulse is further transmitted to the dorsal horn of the spinal cord where it is identified as pain, and here, the brain modulated or influences transmission at the level of the spinal cord.

Rationale for prescribing lidocaine and co-amoxiclav when treating bite injuries in the ED

Lidocaine for local analgesia

Pradyumna Raval, Wasim Khan [7] highlight that the injuries sustained from a dog bite can be very deceptive. The biting force of canine jaws varies by breed, and can result in large wounds and major devitalisation of tissues [3]. Oral pain relief, initially paracetamol based, is likely to be offered, this will lower prostaglandin production and has fewer drug interactions and gastrointestinal complications than other

oral medications available in the ED. Hand injuries require extensive assessment with all presentations of injury, but especially so in traumatic or extensive wounds. Debridement and exploration are necessary in these types of wound, and appropriate pain relief needs to be delivered. Within the ED there is access to multiple variations of local anaesthetic, but the most commonly used, are from the amide group: bupivacaine and lidocaine. The latter is readily available, and commonly accessible under patient group directives (PGDs), which for the non-independent prescriber reduces time spent looking for a concurrent assessment and prescription, and facilitates timely pain relief and treatment for the patient. Lidocaine hydrochloride 1% is the most common preparation. It is available in topical and transdermal preparations, but these are not suitable in conjunction with open wounds. Wells [32] maintains that subcutaneous infiltration of the wound, or via a nerve block, are the fastest acting methods of local anaesthetic administration. Local anaesthetic agents are primarily utilised to prevent pain for varying periods of time and are administered in the PNS [2]. They are very powerful nerve blockers and it is imperative that their effects are limited to a local area only. The drug binds to plasma proteins, and is found to cross both the blood-brain and placental barriers [33]. While administration of some preparations of lidocaine have contributed to severe lidocaine toxicity, there is not much recognition of elevated serum lidocaine levels associated with subcutaneous infiltration [34]. The MHRA [35] acknowledge that the lowest blood levels of the drug occur after subcutaneous administration. Chemically, local anaesthetics are classified as amino-amides and solubility of the local anaesthetic correlates with the potency of the preparation. This lipid solubility assists the passage of drug through lipid membranes of nerve cells. It is thought that as peri-neural tissues are lipid rich, this helps form a depository of the local anaesthetic, thereby enhancing clinical potency. The local anaesthetic causes a temporary interruption of both the production and conduction of the nerve impulses, they intercept sodium ions from entering the nerve, and this in turn stops the nerve from depolarising [2]. As part of the nerve cannot be stimulated nerve impulses that would subsequently be directed towards this section are lost. Binshtok, Gerner [36] acknowledges that by blocking voltage gates and sodium channels in axons, local anaesthetic disrupts action potential generation and prevents transmission of nociceptive information to the CNS. This block causes interruption in sensation which facilitates painless exploration and/or closure of the wound. The literature suggests that thorough debridement of a bite wound aims to discourage infection, and promote maximum healing to the area [6,13,14]. Historically, advice was never to close bite wounds due to an increased infection risk, but this is now strongly refuted by many in the literature [3,11,37]. Depending on the location of a bite on the hand and the size of the wound, either a nerve block or wound edge infiltration can be used [2,32,38]. This method of delivery helps increase efficacy by direct infiltration to the

affected site, furthermore it decreases systemic absorption and associated toxic effects [2,34]. It also has important effects on the CNS and cardiovascular system, since its primary action on the myocardium may cause decreases in excitability, conduction rate and force of contraction [35]. Cardiac arrest due to local anaesthetic toxicity is rare, but evidence shows intravenous infusion of lipid emulsions can reverse the cardiac and neurological effects [39]. It is metabolised in the liver.

Co-amoxiclav for infection

Infection is the major risk associated with dog bites since the wounds are classified as contaminated, and contain a mixture of aerobic and anaerobic organisms [37]. Al-Himdani, Tan [40] maintains that the clinical approach to managing bites in the UK, despite NICE [22] recommendations, is mainly based on consensus opinion rather than evidence. Infection is caused by an invasion and subsequent multiplication of microorganisms in the body. Assessment is generally based on clinical observations within the ED, but in a definite injury such as a bite, visual assessment of the wound is often the initial indicator of the presence of infection.

Much debate surrounds prophylactic antibiotic prescribing in dog bites. Currently in the UK, NICE [22] advises all infected wounds from dog bites should be prescribed per oral antibiotics (co-amoxiclav) for five days, whilst other research advises that all puncture wounds caused by any bite should be treated with prophylactic antibiotics [40]. Despite this, administration of antibiotics in presentations with no signs of infection remain controversial. Medeiros and Saconato [41] says there is no evidence that prophylactic antibiotics will reduced dog bite infection. Tabaka, Quinn [19] further ascertains that antibiotics should only be administered when there is a high risk of infection, but maintains there is no clarity on what constitutes high risk. Smith, Walker [15] argues that prophylaxis is an incorrect term when bites are involved, and administration of antibiotics in these instances should be deemed therapeutic, as no bite can be considered "clean". It is generally accepted that any bite greater than 24 hours old and asymptomatic of infection is unlikely to require antibiotics, as most wounds would exhibit signs of infection by that time. The addendum to this is when delayed closure is required, and antibiotics should be administered in each instance. Even recently, closing any bite remained a contentious issue, and the administration of antibiotics was empirical as opposed to well-defined [11].

The specific characteristics of hand anatomy, coupled with bite mechanics, and organisms found in animal saliva, means that even small wounds can lead to aggressive infections [10]. Dog bites most commonly lead to polymicrobial bacterial infections with a mixture of aerobic and anaerobic organisms [8,42], therefore this makes optimal treatment a difficult challenge [6]. In dog bites, the primary

cause of infection is staphylococcus aureus. These are gram positive, round shaped bacterium which generally live on human skin. In dogs, they are typically found in their upper respiratory tract, but can live on the skin [43]. This bacterium is easily transmitted by biting and general contact with an affected dog. Likewise, staphylococcus aureus may be present on human skin and a laceration or puncture wound causes easy access into human tissues. Pasteurella multocida is a gram negative, motile, penicillin sensitive coccobacillus from the pasteruellae family, and is thought to be the second most common infection in dog bites [3]. This is commonly associated with cat bites, which are believed to cause twice as many incidences compared to dog bites, since cat bites tend to cause puncture wounds as opposed to lacerations [6]. Dog bites tend not to be fatal, however, Linton, Potgieter [20] highlights a third dog-related infection, canimorsus septicaemia (formerly known as dysgonic fermenter type 2 (DF-2)), which although it is uncommon should be anticipated, to limit mortality associated with microorganisms. This is a gram-negative bacillus present in the oral cavities of 22-74% of healthy dogs [44]. It has unique virulence factors that enable it to evade the human immune system and cause life threatening sepsis following a dog bite [45] because it mimics the symptoms of meningococcal infection, and is not usually the initial diagnosis on presentation to hospital. Buttaravoli, Leffler [46] assert that it is a futile exercise to obtain cultures or Gram stains in a dog bite, as results will not correlate well with organisms that will eventually cause infection.

A broad-spectrum antibiotic is required to treat these three bacterial infections, namely co-amoxiclav, orally, in the first instance for five days. Unless there is a visible flexor tendon sheath injury, or septic joint, and the wound requires immediate further specialist intervention, intravenous antibiotics are not initially indicated. Morgan and Palmer [3] advise that erythromycin or flucloxacillin should never be used prophylactically or therapeutically in a dog bite as pasteurella is resistant. Smith, Walker [15] establishes that the added anaerobic coverage offered by a beta lactamase inhibitor is vital.

Co-amoxiclav is a combination penicillin, and belongs to the beta lactamase family which are bactericidal. Amoxicillin is a semisynthetic beta lactam antibiotic that inhibits penicillin binding proteins in the biosynthetic pathway of bacterial peptidoglycan. This is a vital part of the bacterial cell wall which is proceeded by cell lysis and subsequent death [33,47]. As a lone drug, amoxicillin is prone to degradation by beta lactamases produced by resistant bacteria. Clavulanic acid is classified as an adjunct to antibiotic therapy [2]. It is a beta lactam structurally related to penicillin, which functions as a beta lactamase inhibitor, and is produced by Streptomyces clavuligerus. It has a beta-lactam ring which irreversibly binds to beta lactamase thereby deactivating the enzyme and preventing binding to amoxicillin. It has a high affinity for A beta-lactamases, the

bioavailability depends on the dose. Amoxicillin is excreted through the kidneys within 7 hours, while clavulanic acid is partly metabolised, and 40% is eliminated through the kidney [33].

Conclusion

Dog bites represent a large patient group that present to the ED. Initial thorough assessment is key, and helps form a basis to plan appropriate assessment. Whilst global practices on administration of prophylactic or simply therapeutic antibiotics differ, fundamentally, patient comfort is key. Good, rapid acting subcutaneous analgesia such as lidocaine 1% allows for a pain free environment that facilitates assessment, debridement, and treatment where necessary. While specific drug choices for subcutaneous infiltration for initial pain management may differ, there is unanimous use of co-amoxiclav for appropriate dog bite treatment in the non-penicillin allergic patient.

Result

Dog bites are the most common type of biting injury to present at ED. Typically they are treated with an analgesic for pain relief and an antibiotic since the wounds are classed as contaminated.

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RHMS = Study concept and design, acquisition of the data, analysis and interpretation of the data, drafting of the manuscript, critical revision of the manuscript for important intellectual content

MB = drafting of the manuscript, critical revision of the manuscript for important intellectual content

LH = critical revision of the manuscript for important intellectual content

OMN = critical revision of the manuscript for important intellectual content

KMAR = Study concept and design, drafting of the manuscript, critical revision of the manuscript for important intellectual content

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