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Ultrasound-guided administration of lidocaine into the sciatic nerve in a porcine model: Correlation between the ultrasonographic evolution of the lesions, locomotor function and histological findings

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ABSTRACT

Intraneural puncture of local anaesthetics has been associated with permanent or transitory nerve injury. The use of ultrasound (US)-guided techniques for the blockade of peripheral nerves has revealed that intraneural puncture is a relatively common complication, which is not frequently associated with neurological deficits. In this study, 2.5 mL of lidocaine were administered using US-guidance into the sciatic nerve (ScN) of 12 piglets. The punctured nerves were sequentially evaluated by US (cross sectional area and relative echogenicity) before and immediately after the injections, and then at 1, 2, 4, 7 and 14 days. At these times, animals were euthanased two by two at each time point, and ScN samples were removed for histological examination.

Cross sectional area and relative echogenicity values were statistically different immediately after the injections, returning to pre-puncture values within 4 days. The inflammatory process observed by histopathology showed a similar trend indicating that the integrity of the perineurium was maintained. Locomotor deficits were not observed. The increase in size of the ScN produced by the injection of lidocaine intraneurally did not induce motor deficits in piglets in the current study.

Introduction

Regional anaesthesia and nerve blocks are widely used in human anaesthesia to avoid the need for general anaesthesia and its associated risks. The use of local anaesthetics improves intraoperative analgesia and patient comfort during the post-operative period (Roberts, 2006). Similar approaches are also used in the anaesthesia of small animals (Campoy et al., 2008; Echeverry et al., 2009, 2012; Haro et al., 2012).

Nerve damage during the blockade of peripheral nerves (PNB) is considered a major possible complication of these techniques, as permanent injury can be induced (Borgeat et al., 2001) and intraneural injection of local anaesthetic has been recognized as a cause of nerve damage (Kapur et al., 2007). Sharp pain and excessive pressure during the injection have been suggested as possible signs of intraneural puncture (Fredrickson, 2009; Altermatt et al., 2010) although these signs are obviously not reliable indicators when the patients are under general anaesthesia.

Nerve stimulation is a method of nerve location widely employed to perform peripheral nerve blocks (Sala-Blanch et al., 2009a,b). However, it has been reported that evoked motor response may not always be present, even when the needle appears to be in physical contact with the nerve (Bollini et al., 2003). More recently, the use of ultrasound (US)-guided nerve block techniques has allowed the positions of the needle and the target neural structures to be visualized (Marhofer et al., 2005). This technology represents an advance in the accuracy of the nerve location, and also in the safety of the procedure, as it is possible to place the needle at a close but safe distance from the target nerves (Altermatt et al., 2010).

The use of US-guided techniques has demonstrated that nerves are frequently punctured and injected with local anaesthetic. Some reports suggest that nerve puncture or intraneural injection does not inevitably lead to neurological injury (Bigelaisen, 2006; Kapur et al., 2007; Altermatt et al., 2010). Histological studies have documented that subepineural administration of local anaesthetics

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does not affect the integrity of the nerve fascicles, surrounded by the perineurium (Chan et al., 2007; Lupu et al., 2010). However, the capacity for a nerve to be distended is related to the extent of its connective tissue, which is highly variable between different nerves (Sala-Blanch et al., 2009a).

The objective of the present study was to assess the potential adverse effects of an US-guided intraneural administration of a low volume of lidocaine into the sciatic nerve (ScN) of piglets, correlating the ultrasonographic evolution of the nerve distension with the proprioceptive function and histological examinations.

Materials and methods

Animals

This study was approved by the University of Murcia Ethics Committee (15291/10). Fourteen purpose-bred piglets with a mean age of 2.6 ± 0.2 months and a bodyweight of 17.5 ± 1.9 kg were employed. The animals were bred in the Teaching Farm of the Veterinary Faculty of the University of Murcia.

The animals were individually housed indoors on the day before the study began. They had free access to food and water but food was withheld for 12 h before anaesthesia. On physical examination, all animals were found to be healthy and free of proprioceptive and motor deficits.

Ultrasonographic examinations

A 4–13 MHz linear transducer (MyLab 70, Esaote) was employed for the US examinations. Constant focus, brightness, contrast and gain settings were used during the recording sessions. Prior to ScN image acquisition, images with identical imaging variables to those used for the nerves were obtained from three soft-tissue equivalent ultrasonographic phantoms. The phantoms were made in our laboratory and stored in sterile blood transfusion pockets. Phantom 1 was a mixture at 50 °C of 3% of agar in 138 mL of water and 12 mL of 70% isopropyl alcohol. Phantom 2 consisted of 10% agar added to the same volume of water and alcohol as that employed for Phantom 1. Phantom 3 added 6.4% gelatine to the mixture employed in the Phantom 2. Phantoms were immediately placed in a vacuum apparatus to remove air bubbles and then stored in a refrigerator at 4 °C (Wood et al., 1993).

The digital US images obtained from each nerve at different times, together with the images of the three phantoms were recorded on the same day. All images were evaluated using an image-analysis system (Microm Image Processing software) to determine the values for cross sectional area (CSA) and relative echogenicity (RE) of the ScN.

The same author (MS) performed all measurements, using the image-analysis system. Each ScN was outlined, and a CSA value (mm^2) obtained. The tracing was repeated and a total of three measurements for each sample were made, and the mean was calculated. The RE of each nerve was measured using a scale of 256 grey levels (0 = black; 255 = white). The relative brightness of each nerve in the US image was calculated by dividing its mean echogenicity (ME) by the mean value for the ME for an area of the same size and depth from the three phantom images. The RE measurement was repeated so that three measurements were made at each nerve and the mean was calculated. The coefficient of variation of these measurements was <14%.

Puncture technique

On the morning of the intraneural puncture the animals were chemically restrained by the administration of a mixture of tiletamine/zolazepam (Zoletil 100, Virbac; 4 mg/kg) IM into the epaxial muscles. After 3–5 min, and once the piglets were sedated, the marginal auricular vein was catheterised (Vasocan, BBraun; 24G). The catheter was flushed with saline, and a combination of medetomidine (Domtor, Esteve; 10 μg/kg) and buprenorphine (Buprex, Boehringer Ingelheim; 20 μg/kg) was administered IV. Meloxicam (Metacam, Boehringer Ingelheim; 0.2 mg/kg) IM was also given.

Anaesthesia was induced with propofol (Propofol Lipuro 1%, BBraun) given slowly to effect, until the palpebral reflex and mandibular tone disappeared. Orotracheal intubation was carried out with a cuffed tube of the proper size after desensitising the larynx with lidocaine (Xilonibsa 10%, Inibsa). The piglets were connected to a circle breathing system and anaesthesia maintained with isoflurane (Isoflo, Esteve) in 100% oxygen. A balanced solution of lactated Ringer's fluid (Lactato de Ringer, BBraun; 4 mL/kg/h) was continuously administered during anaesthesia.

The hair was clipped from the sacroiliac region to just below the stifle on the caudal, lateral and medial aspects of the limb. Then the skin was cleaned and coupling gel applied. The piglets were positioned in right lateral position. The transducer was placed in the transverse plane just distal and caudal to the trochanter major and then directed toward the distal aspect, close to its origin to the point where the common fibular and tibial nerves diverge near the stifle. Transverse images were also obtained by positioning the transducer parallel to the dorsal anatomical plane, caudal to the trochanter major of the femur and cranial to the ischiatic tuberosity.

The mark of the transducer in longitudinal and transverse planes was positioned proximal and cranial, respectively. Longitudinal images of the ScN were obtained by rotating the transducer 90° anti-clockwise from the position used to obtain the transverse images. Several acoustic windows were available to approach the ScN nerve along the lateral surface of the thigh. However, it was decided to select a mid-femur approach in order to standardise all the procedures.

The tip of an atraumatic insulated peripheral nerve blockade needle (Stimuplex, 0.9 × 150 mm, BBraun) was used for the puncture of the ScN. The insulated needle was inserted in the long axis (in-plane) of the US transducer. The needle was US-guided to ScN, and once the tip was considered to be inside the ScN, a peripheral nerve stimulator (Stimuplex, HNS 11, BBraun) was used to confirm the position. The device was set at a stimulus frequency of 2 Hz and with a pulse width of 100 μs. The current amplitude was started at 0.5 mA and gradually decreased to reach 0.3 mA. At this point, a positive response (extension or flexion of the tarsus) was associated with an intraneural location of the needle tip. Then a total volume of 2.5 mL of lidocaine (Lidocaine 2%, BBraun) was administered and the injection pressure recorded by a pressure manometer (B-Smart, Macosta). An injection pressure ⩾25 psi (172.37 kPa) was considered a sign of intrafascicular injection.

The US examination of the ScN was performed before and after the injection of lidocaine and the CSA and RE recorded. All US scans were made by the same investigator (AA). Locomotor function was assessed daily by observation of the leg position, proprioception, standing and walking patterns. This test was carried out by the same investigator (EB) in all cases. Firstly, by observation of the piglets undisturbed and standing, and later the animals were evaluated at walking after they were gently stimulated. Two piglets were humanely euthanased by the administration of pentobarbital (Eutalender, Normon; 100 mg/kg) and samples of the ScN were immediately (maximum 5 min) taken after euthanasia.

Over the remaining observational days, 1, 2, 4, 7 and 14 days, piglets were anaesthetized two by two by a combination of medetomidine (20 μg/kg) and tiletamine/zolazepam (4 mg/kg). Then, the ScN nerves were evaluated by US and, after the scans had been taken, the animals were euthanased (as previously described) and ScN samples collected.

In order to perform the histological examination, the nerves were carefully dissected, and specimens 3 cm long (1.5 cm on either side of the punctures) were taken from the area of puncture containing the injection sites. The tissues were fixed in 10% neutral buffered formalin for at least 72 h. After fixation, all tissue blocks were processed for paraffin embedding and cross-sections, 8 μm thicknesses, obtained from the injured site and stained using haematoxylin and eosin.

For each sample nerve, at least five step sections separated by 100 μm were examined for histological evidence of nerve inflammation and injury. Evidence of nerve inflammation was defined as the presence of inflammatory cells around vessels and fascicles of nerve tissue (Steinfeld et al., 2010). Evidence of nerve injury was defined as the loss of integrity in the perineurium. The presence of neural inflammation was graded on 3-point nominal score (Lupu et al., 2010; Steinfeld et al., 2010) as follows: 0 = no inflammatory cells; 1 = areas with slight accumulation of inflammatory cells, and 2 = areas with abundant accumulation of inflammatory cells.

Statistical analysis

The statistical tests were performed using SPSS, version 15.0. Data are expressed as means ± standard deviation (X ± SD). Normally distributed data (established by the Kolmogorov–Smirnov test) collected during the three sample points (RE and CSA) were compared using a one-way ANOVA with Tukey post hoc analysis performed to compare the measured parameters. A value of $P \leqslant 0.05$ was considered significant for all the analyses.

Results

The ScN was easily visualized by US in all cases. The ScN was visible between the muscles of the thigh where it lay medial to the biceps femoris and caudal to the femur as a hyperechogenic structure (Fig. 1). In sagittal views, the ScN appeared to be a tubular hypoechogenic structure delimited by two hyperechoic lines. Once the tip of the needle was judged to be inside the ScN, a current of 0.3 mA elicited motor response in all cases. Injection pressure never exceeded 138 kPa (20 psi).

The ScN post-puncture mean CSA values (91.86 ± 34.04 mm^2), measured immediately after the injections, increased significantly when compared with the pre-puncture CSA values (28.08 ± 6.62 mm^2) ($P < 0.001$) (Table 1; Fig. 2). The RE of the ScN measured immediately after puncture (1.22 ± 0.54), significantly decreased in relation to the pre-injection measurements