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Case Report Open Access

Growing Skull Fracture: Case Report after Rottweiler Bite and Review of the Literature

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Abstract

Growing skull fracture remains a rare but clinically significant complication of traumatic skull fractures in children less than 3 years of age. Dog attacks on children commonly cause head and neck injuries. We report the first case of growing skull fracture caused by a Rottweiler bite in a 21 days old neonate. Early diagnosis and surgical repair resulted in excellent outcome.

Keywords: Skull fracture; Dog bite; Surgical repair; Parenchymal injury

Introduction

Growing skull fractures (GSF) are rare complications occurring in 0.05-1.6% of traumatic skull fractures in children, most commonly in those less than 3 years old [1]. They are most common in the parietal region of the head [2]. The pathological requisites, as described by Lende and Erickson, include; skull fracture in infancy/early childhood, dural tear with intact arachnoid at the time of fracture, underlying parenchymal injury and cranial defect resulting from enlargement of the fracture gap because of an underlying expanding force [3]. The etiology of GSF is most commonly a fall resulting in head trauma [4]. Animal attacks resulting in head injuries have also been described and also are associated with delayed presentation due to fear of euthanization of the dog. Dogs are the most common domestic animals known to inflict head and neck injuries in children. Kumar et al. [5] recently described the neurological sequelae of dog bites to the head and neck region in 124 children. However, to the best of our knowledge, a growing skull fracture resulting from a dog bite to the head has not been described. Here, we describe the case of a 21 days old neonate who developed a growing skull fracture 3 weeks after a Rottweiler bite to the head. The treatment of choice for growing skull fracture remains surgery.

Case Presentation

The patient is a 21 days old male who presented to the emergency department for a head laceration following a dog bite. The parents were at a friend's house, with the father holding the patient in his arms, when a 130 pound Rottweiler came up and bit the patient on the head, without provocation. According to records, the Rottweiler was vaccinated. The patient was immediately taken to an outside hospital, where he received a dose of intravenous ceftriaxone and was then transferred to a level 1-trauma center for further care. On examination, the patient had an 8 cm V-shaped scalp laceration in the right parietal region, without evidence of violation of the dura. He was otherwise neurologically intact. CT brain without contrast showed a mildly displaced fracture of the right parietal bone (Figure 1). He was taken to the operating room for washout and repair of the scalp laceration. He was admitted to the pediatric intensive care unit after surgery. He was started on intravenous unasyn, which was later transitioned to oral augmentin for duration of 1 week. The most common organisms from dog bite wounds are pasteurella, streptococcus, staphylococcus, and neisseria, which is why the antibiotic types were chosen. The offending Rottweiler was tested for rabies and found to be negative. The patient was discharged home after a 3 days hospital stay.

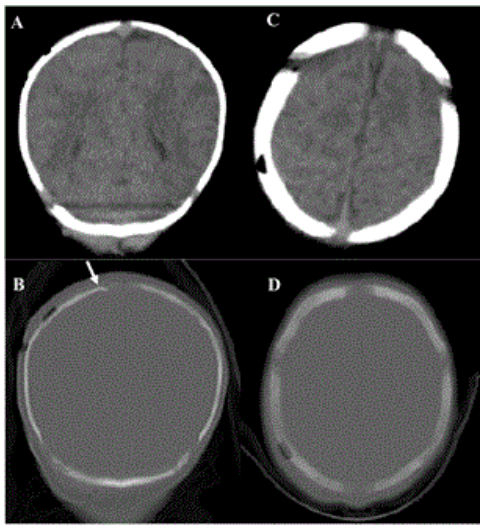

Figure 1: Imaging at initial presentation - CT brain without contrast showing minimally displaced right parietal fracture on coronal plane (B, white arrow) and absence of brain parenchymal injury on coronal and axial plane (A, C).

Three weeks after discharge the patient was re-admitted to the pediatric intensive care unit for increasing swelling in the right parietal region, at the site of the original Rottweiler bite. The swelling was not

associated with signs and symptoms of infection. On examination a 7 cm × 7 cm soft swelling with no drainage and no erythema was found in the right parietal region. The patient was neurologically intact and had no meningismus. Magnetic resonance imaging revealed a small right parietal encephalocele through a dural tear and an osseous defect, with a subgaleal fluid collection. Three-dimensional CT brain imaging demonstrated the osseous defect in the right parietal region (Figure 2), corresponding to the site of the minimally displaced right parietal fracture from the Rottweiler bite. The clinical history, together with these imaging findings, was consistent with progressive growing skull fracture. The option of surgical management was discussed with the parents, and consent was obtained to perform a right parietal craniectomy and autologous duraplasty.

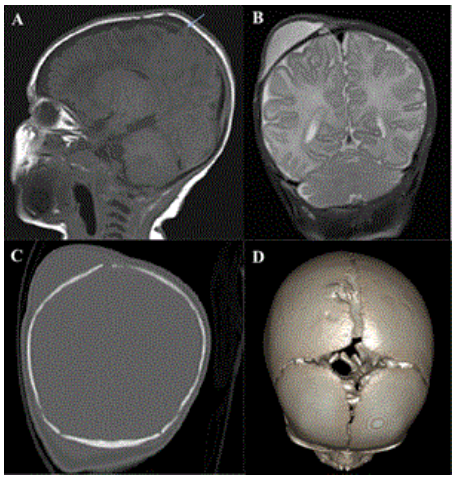

Figure 2: Imaging at 3 weeks after dog bite - Magnetic Resonance Imaging sagittal and coronal plane (A and B) demonstrating dural tear and right parietal encephalocele and subgaleal fluid collection. CT imaging bone window coronal plane (C) and three-dimensional CT imaging (D) show the osseous defect in the right parietal region.

Surgical procedure

Under general anesthesia, the patient was positioned in the left lateral decubitus position and the head supported by a horseshoe. The skin was marked, prepped and draped in a sterile fashion. An incision was made and the scalp dissected to expose a subgaleal cyst of fluid. This corresponded to a leptomeningeal cyst, which was then opened showing xanthochromic fluid that was sent to the laboratory for regular microbiological investigations. Reflection of the scalp flap revealed a bony defect with brain herniating through, corresponding to the encephalocele found on pre-operative images. A small bur hole was drilled and a 2 × 2.5 cm craniectomy made (Figure 3). The craniectomy showed brain herniating through the dural defect, which was cauterized. The dural defect was closed with a pericranial graft. The skin was then closed in layers.

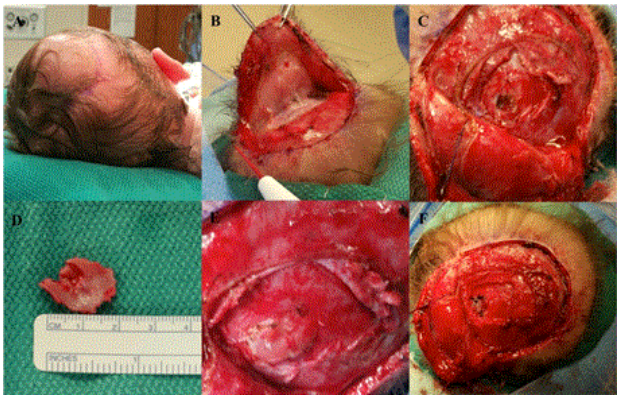

Figure 3: Surgical repair of the growing skull fracture. A. Left lateral decubitus positioning of patient. B. Leptomeningeal cyst is opened, showing brain tissue herniating through osseous defect (asterisk). C and D. Craniectomy is performed. E and F. Water-tight closure of dural defect is secured with pericranium.

Postoperative care and follow up

Postoperatively, the patient was sent to the pediatric intensive care unit. He received perioperative antibiotic prophylaxis for 24 h and a 7 day course of keppra for seizure prophylaxis. Cultures from the subgaleal fluid collection showed no microbial growth. He was discharged home after a 6 days hospital stay. At the 4 months follow up patient was developing normally, incision was well healed and he had no neurological deficits.

Discussion

Growing skull fracture remains a rare but clinically significant complication of traumatic skull fractures in children less than 3 years of age. In many case series reported, the most common cause has been skull fracture resulting from a fall. The current case is the first reported case of growing skull fracture caused by a dog bite. Dogs are pets that are in frequent contact with children and the potential for attacks should always be recognized and guarded against. Kumar et al. noted that large breed dogs are more likely to cause head and neck trauma in children [5]. Of the breeds of dogs involved in fatal human attacks, Sacks et al. determined that the Pit Bull and Rottweiler are the breeds most commonly implicated in fatal attacks [6]. Not surprisingly, the current case was caused by an unprovoked attack by a 130 pound Rottweiler. The diagnosis of GSF is based on clinical signs and symptoms, most commonly scalp swelling [7], combined with radiological demonstration of skull fracture, dural tear, and herniation of brain parenchyma. CT brain can demonstrate the osseous defect, while dural tears and brain herniation are best revealed by magnetic resonance imaging [8,9].

The pathogenesis of GSF involves a skull fracture with a dural tear and entrapment of arachnoid membrane or brain tissue within the fracture margins with or without the development of neurological deficits [10]. Various classification schemes have been described. Ur-Rahman et al. [11] described three types of GSF based on the predominant factor for fracture growth, in order of increasing severity, as follows: Type I as a GSF with leptomeningeal cyst; Type II as GSF with underlying gliotic brain; and Type III as GSF with porencephalic

cyst [11]. Liu et al. [1] classified GSF into 3 stages of progression based on a time scale. Stage 1 (prephase of GSF) is described as occurring from the time of injury to the time just before enlargement of the fracture and is characterized by skull fracture with dural tear and herniation of brain tissue or arachnoid membrane through the fracture. Stage 2 (early phase of GSF) occurs from the time of the initial fracture enlargement to 2 months after the beginning of enlargement. Stage 3 (late phase of GSF) begins at 2 months after initial enlargement and is characterized by gross enlargement of the bone defect, skull deformity, and neurological deficits. Liu et al. [1] found that patients treated in stages 1 and 2 have better outcomes compared to patients treated in stage 3. Our patient was diagnosed of Stage 2 GSF at 3 weeks after the Rottweiler bite, with mild enlargement of the bone defect, with no neurological deficits.

The treatment of choice for GSF remains surgical repair. Depending on the size of the initial defect and stage of progression, surgical repair may include craniectomy, resection of gliotic/herniated brain parenchyma, duraplasty and cranioplasty [1,7,12]. In patients who develop seizures preoperatively, electrophysiological monitoring has been recommended for localization of seizure foci during resection of the herniated brain parenchyma [1]. The choice of material for duraplasty may be autologous (pericranium or fascia lata) or synthetic. Autologous duraplasty has the advantages of being more biocompatible, inexpensive, and is associated with lower risk of infection. Cranioplasty is recommended for defects larger than 3 cm and can be achieved with autologous bone (either split calvarial graft or rib autograft), titanium mesh or polymethyl methacrylate. In our patient, duraplasty was performed with a piece of pericranium [7]. We did not perform cranioplasty in our patient because the size of the bony defect and the craniectomy were small (less than 3 cm).

The outcomes of surgery for GSF have generally been reported to be good. Possible complications of GSF described in the literature include cerebrospinal fluid leak, hydrocephalus, bone flap infection, seizures, recurrence, and death. Mortality is in the range of 0 to 8% [7]. Prasad et al. identified the following as poor prognostic factors: age >8 years, female gender, large defects (>7 cm), severe head injury at initial trauma, defects crossing the midline and delayed repair (>8 months) [7]. Our patient sustained a dog bite to the head at a very early age of 21 days and the diagnosis of GSF was made relatively early at 3 weeks. Surgical repair resulted in excellent outcomes, similar to other cases reported in the literature.

Conclusion

Dog bites in the head and neck region in children can cause serious neurological sequelae, including growing skull fracture. A high index of suspicion can result in early diagnosis, treatment, and good outcomes.

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