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PAROTID HAEMANGIOMA IN AN INFANT MASQUERADING AS ACUTE PAROTID ABSCESS: A DIAGNOSTIC DILEMMA

Paediatrics

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ABSTRACT

Haemangioma is the commonest tumour of infancy and the commonest benign salivary gland tumour of childhood. When a parotid haemangioma ulcerates and becomes secondarily infected it may closely mimic acute suppurative parotitis, posing a diagnostic dilemma. We report an 11-month-old male infant with a rapidly enlarging right parotid swelling bearing ulcerated margins, bluish discolouration of the overlying skin and foul-smelling discharge, treated repeatedly as acute parotitis without benefit. Aspiration performed to exclude a pus collection yielded only blood. Magnetic resonance imaging and contrast-enhanced computed tomography demonstrated a slow-flow vascular lesion of the right parotid gland involving both lobes, extending into the external auditory canal and middle ear cavity and supplied by a branch of the external carotid artery. Oral propranolol produced progressive regression without adverse effect.

KEYWORDS

Infantile Haemangioma, Parotid Gland, Propranolol, Ulceration

INTRODUCTION

Salivary gland tumours are uncommon in children, accounting for under 5% of all salivary gland neoplasms. Haemangioma predominates among them, being both the commonest tumour of infancy and the commonest benign salivary gland tumour of childhood, and constituting roughly half of all paediatric parotid tumours [1]. Females are affected about three times as often as males, and the usual age at presentation is 4 to 5 months, corresponding to the proliferative phase. The natural history is one of early proliferation, a plateau, and slow spontaneous involution, which underpins the traditional recommendation of watchful waiting [1,6].

Parotid lesions may nevertheless proliferate rapidly, ulcerate, become secondarily infected and extend into adjacent structures, whereupon the clinical picture closely resembles acute suppurative parotitis. Ulceration is the commonest complication of infantile haemangioma and is itself an indication for active treatment [5]. We report one such case, repeatedly misdiagnosed and treated as parotitis before the correct diagnosis was established.

Case Report

An 11-month-old male child, born at term by normal vaginal delivery, presented to the paediatric outpatient department with a progressively enlarging swelling of the right parotid region, first noticed at 7 months of age. The overlying skin had developed ulcerated margins with bluish discolouration and foul-smelling discharge. There was intermittent fever of seven days' duration and a productive cough. There was no history of trauma, no swelling elsewhere, and no contact with tuberculosis or household animals. The parents had sought advice at multiple centres, and the child had received oral antibiotics for one week for a presumed acute parotitis, without improvement.

On admission he was active and conscious, with a pulse rate of 110/min, respiratory rate of 30/min, temperature of 98.6°F and oxygen saturation of 98% in room air. Anthropometric parameters were appropriate for age. The first and second heart sounds were normal with no murmur. Chest expansion and air entry were equal bilaterally, with conducted sounds. The abdomen was soft and non-tender with no organomegaly, and neurological examination was normal. Local examination revealed a right parotid swelling of approximately 4–5 cm, soft, mobile, non-tender and non-pulsatile, with ill-defined ulcerated borders, bluish discolouration of the overlying skin and foul-smelling discharge at the margins [Figure 1]. No bruit, sinus formation or airway compromise was present. Multiple right-sided cervical lymph nodes were palpable.

Investigations revealed a microcytic hypochromic anaemia (haemoglobin 8.8 g/dL, mean corpuscular volume 59.5 fL) with a mild leucocytosis (13,300/mm³) and lymphocyte predominance. C-reactive protein was normal and the erythrocyte sedimentation rate was mildly raised at 32 mm/hr. Coagulation profile, renal function, electrolytes and serum calcium were normal, and viral markers were non-reactive. In view of a chronic swelling with cervical lymphadenopathy in an endemic setting, tuberculosis was evaluated; gastric lavage for cartridge-based nucleic acid amplification testing was negative.

Aspiration of the swelling was undertaken to exclude an underlying septic focus, given the fever, ulceration and purulent discharge. Only blood was aspirated, with no pus obtained, raising a strong suspicion of a vascular lesion.

Non-contrast magnetic resonance imaging on a 1.5-Tesla scanner demonstrated an enlarged right parotid gland containing a large, thick-walled, fluid-signal-intensity lesion involving both the superficial and deep lobes, hypointense on T1-weighted and hyperintense on STIR sequences, with patchy restriction on diffusion-weighted imaging. Flow voids and internal septations were present, and the lesion abutted the carotid sheath posteriorly, measuring 5.3 × 4.4 × 4.1 cm. There was mild oedema of the right pterygoid muscle, partial effacement of the right parapharyngeal fat, thickening of the right mastoid air cells with fluid in the middle ear cavity, and multiple enlarged cervical nodes, the largest 2.1 × 2.0 × 1.2 cm at right level II.

Contrast-enhanced computed tomography showed a well-defined lesion in the right post-auricular region measuring 3.4 × 3.3 cm, extending into the external auditory canal and middle ear cavity, with peripheral nodular enhancement on the arterial phase and centripetal filling on venous and delayed phases. The lesion extended into the petrous and squamous temporal bone with an area of expansion, and appeared to be supplied by a branch of the right external carotid artery. The impression was of a slow-flow vascular malformation, favouring haemangioma, with cervical lymphadenopathy.

Paediatric and plastic surgery opinions recommended conservative management with local wound care comprising povidone-iodine cleaning and topical mupirocin. Intravenous antibiotics were administered for one week for the superadded skin infection, following which the ulcerated margins healed. A baseline electrocardiogram was normal, and oral propranolol was initiated at 1 mg/kg/day in two divided doses, with heart rate, blood pressure and blood glucose monitored for two hours after the first dose and after each escalation. The parents were counselled regarding the natural course of the lesion and the adverse effects of propranolol, including hypoglycaemia, bradycardia, hypotension and bronchospasm. The child was discharged in satisfactory condition with clear swelling margins.

At three-week follow-up the swelling had increased to approximately 2 × 3 cm with clear margins. In view of continued proliferation and good tolerance, propranolol was escalated to a maintenance dose of 2 mg/kg/day in two divided doses. On regular follow-up the swelling decreased progressively in all dimensions, with resolution of the ulcerated surface and a good clinical response, and no adverse effect attributable to the drug [Figure 2].

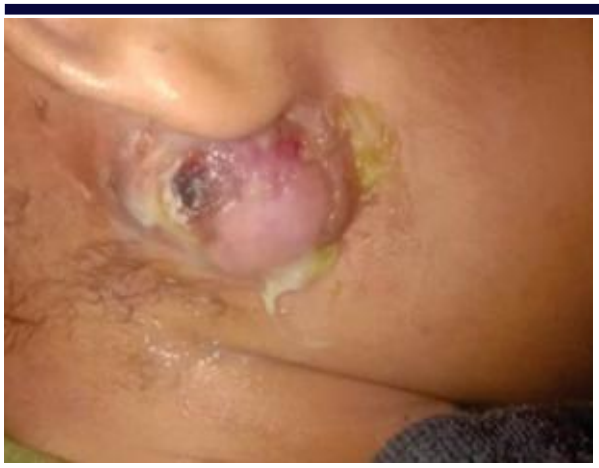

Figure 1: Right Parotid Swelling Showing the Ulcerated Surface with Adherent Crusting and a Central Necrotic Focus, with Bluish Discolouration of the Surrounding Skin.

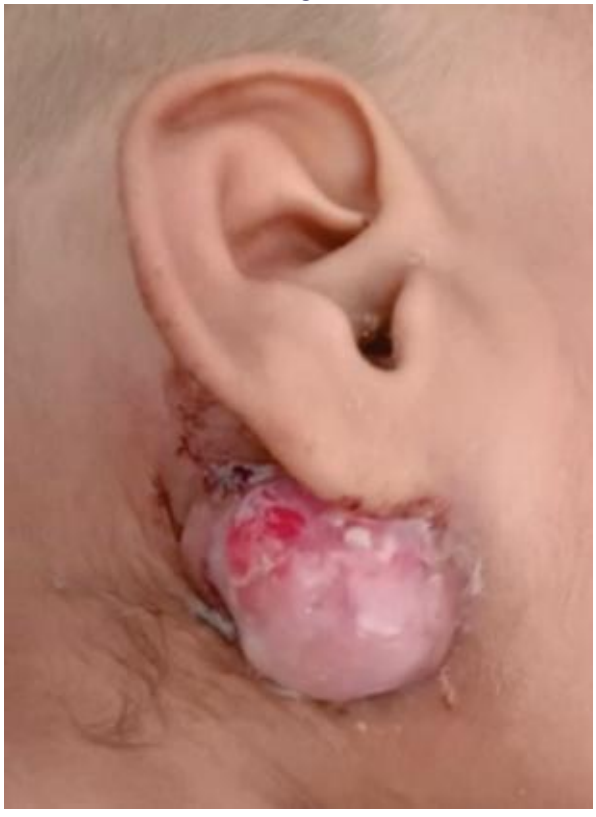

Figure 2: The Same Lesion Following Propranolol Therapy and Local Wound Care, Showing a Clean Granulating Surface with Resolution of the Necrotic Slough and Clear Margins.

DISCUSSION

Infantile haemangioma is a true vascular neoplasm characterised by endothelial proliferation, and must be distinguished from vascular malformations, which are errors of morphogenesis, are present at birth, grow proportionately with the child and do not involute. This distinction, articulated in the biological classification of Mulliken and Glowacki, remains fundamental, since the two entities differ substantially in natural history and in response to therapy [2].

The present case illustrates two issues. First, a parotid haemangioma that has ulcerated and become secondarily infected can convincingly mimic acute suppurative parotitis, particularly when accompanied by fever, foul-smelling discharge and cervical lymphadenopathy. This led to repeated antibiotic courses at multiple centres, and to an aspiration in our own unit, before the vascular nature of the lesion was appreciated. The clues that redirected the diagnosis were the soft, compressible, non-tender consistency of the swelling, the bluish discolouration of the overlying skin and, most tellingly, the aspiration of blood rather than pus. Aspiration or biopsy of a suspected vascular lesion is best avoided owing to the risk of haemorrhage, and imaging should ideally precede any intervention; in this child the strength of the clinical suspicion of an abscess prompted aspiration first, a sequence that earlier cross-sectional imaging would have averted. The associated lymphadenopathy is most plausibly reactive to the infected ulcerated surface, and is not a feature of uncomplicated infantile haemangioma.

Second, the case demonstrates the limitations of a purely expectant approach. Although conservative management suits many uncomplicated lesions, the ulceration, rapid proliferation, deep-lobe involvement and extension into the external auditory canal, middle ear and temporal bone were clear indications for pharmacological therapy [3,5]. Extension towards the external auditory canal threatens conductive hearing loss during a critical period of speech development. Cross-sectional imaging was pivotal: magnetic resonance imaging delineated the true extent of the lesion, which was considerably larger than was clinically apparent, while computed tomography confirmed its vascular nature through arterial-phase peripheral nodular enhancement, centripetal filling and an external carotid feeder. Ultrasonography with colour Doppler is the preferred initial modality in infants, while magnetic resonance imaging remains the investigation of choice for defining deep extension.

Since the serendipitous observation of its efficacy in 2008, propranolol has become the first-line therapy for problematic infantile haemangiomas, largely displacing systemic corticosteroids [4]. Its action is multifactorial, comprising vasoconstriction, inhibition of angiogenesis through downregulation of vascular endothelial and basic fibroblast growth factors, and apoptosis of proliferating endothelial cells. Guidelines advise commencing at 1 mg/kg/day and escalating, once tolerance is established, to 2–3 mg/kg/day, continuing until at least 12 months of age or until adequate regression is achieved [5]. This graded schedule was followed in our patient, with baseline cardiovascular assessment, cautious escalation and structured parental counselling; no bradycardia or hypotension occurred. A multidisciplinary approach involving the paediatrician, paediatric surgeon, plastic surgeon and radiologist is essential, both to establish the diagnosis and to balance the risks of intervention against the natural history of involution.

Thus, this presentation of an infantile parotid haemangioma as an ulcerated, discharging swelling mimicking acute parotitis underscores the importance of thorough clinical examination and timely cross-sectional imaging, so as to avoid misdiagnosis, avoidable invasive procedures and delay in instituting effective therapy.

Declaration of Consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the parents have given their consent for their child's images and other clinical information to be reported in the journal. The parents understand that the child's name and initials will not be published and due efforts will be made to conceal his identity, though anonymity cannot be guaranteed.

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