Inflammatory swellings of the head and neck

Russell A Cathcart

Abstract

By and large, inflammatory swellings of the head and neck are likely to have one of three aetiologies: infective, autoimmune or allergic, with infective causes being the most commonly encountered. By considering pathologies under these headings for the corresponding site of inflammatory swelling, a clinical diagnosis can usually be obtained particularly when the chronicity of the swelling is taken into consideration. These inflammatory swellings, when acute, can quickly become life-threatening in which case a rapid clinical diagnosis is vital. In this article we revise the pathophysiology of inflammation before considering specific pathologies affecting various anatomical sites of the head and neck including the face, the orbit/forehead, the oral cavity, the neck, the parotid gland and the thyroid gland. The management of these conditions is discussed and other miscellaneous inflammatory swellings are also considered.

Keywords Abscess; allergic; anaphylaxis; autoimmune; infective; inflammatory; oedema; swelling

Introduction

When addressing a topic as diverse and encompassing as inflammatory swellings of the head and neck, one can assume an object-based approach or a process-based approach. The former acknowledges that patients do not present with pathological processes but rather with clinical signs; that is, patients do not present complaining of disease entities, they present complaining of a painful swelling above the eye or over the cheek or within the mouth. As such it would seem sensible for one to learn by rote the diagnoses that result in inflammatory-looking swellings of each anatomical site. Doing so, however, risks exclusivity and omissions and so in many ways it is more sensible to consider the pathophysiological processes which could result in an inflammatory-looking swelling and then more logically list the conditions that those processes may cause at any particular anatomical site.

Pathophysiology of inflammation

With this in mind, it is worth re-familiarizing oneself with what inflammation is and what causes it. Inflammation is the innate response of the tissues of the body to an insult. Put simply, the initial acute phase of inflammation is aimed at maximizing recruitment of the body's defence systems with a view to damage limitation. The defence systems recruited include the clotting cascade, the complement cascade, vascular endothelial activation and the humoral and cellular immune responses. The substances released in this acute phase (bradykinins, leukotrienes, cytokines and histamine) are responsible for the classical signs that are pathognomic of acute inflammation viz. rubor, calor and dolor.

The latent chronic phase of inflammation is aimed at walling-off any foreign material and necrotic tissue until such time as they are sequestered or rendered inactive or non-threatening.

By and large, spontaneous inflammatory swellings of the head and neck are likely to have one of three causes:

- infective
- autoimmune
- allergic.

Other causes of inflammation do of course exist, such as trauma or surgery, but these are invariably apparent at the time of clinical presentation. The underlying diagnosis will, of course, vary according to the actual site of swelling but for any particular anatomical site, it is helpful to consider the cause as being of these three categories.

Infective swellings

The great majority of inflammatory swellings of the head and neck have an infective cause. Although these are often considered under the distinct entities of soft tissue infections or frank abscesses, it is more useful to consider these entities as being at either end of a progressive spectrum.

The head and neck is a region which is rich in lymphoid tissue (pharyngeal tonsils, lingual tonsils and adenoids forming Waldeyer's ring, and the superficial and deep cervical lymph node chains). Given that it is under continuous exposure to environmental pathogens, it is of little wonder that it is so prone to harbouring soft tissue infections. Once an infecting organism has invaded the local tissues at its portal of entry, an inflammatory reaction is triggered in response to antigens on the cell wall of the organism or to the toxins which the organism releases. The perceived virulence of the pathogen or its toxin will determine whether an acute inflammatory reaction or a chronic reaction results.

Acute versus chronic

Infective swellings can be acute, chronic or acute-on-chronic. The delineation between acute and chronic inflammation is not determined chronologically but rather by histological findings. That said, it can generally be accepted that the acute inflammatory reaction to the pathogen will subside after a few days and will either organize and resolve, or, if the pathogen persists, will make way for the containing role of the chronic inflammatory response. The egress of the acute inflammatory soup means that the swelling loses its erythema, tenderness and warmth to become a 'cold' (body temperature), non-tender swelling which may have a dark hue. Frequently the acute reaction is so low-key as to be non-apparent and in this case the swelling will present as a *de novo* chronic swelling.

Bacteria, virus or fungus

The family of the underlying pathogenic organism will determine the dominant cell line recruited to defend against the pathogen and this can provide a diagnostic aid when the underlying organism is not immediately apparent. A bacterial infection will

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primarily see polymorphonuclear white cells being attracted to the area (neutrophils) whereas a viral infection typically sees mononuclear leucocytes. The reason for this is reasonably straight-forward – neutrophils are better adapted to phagocytose bacteria (cellular immunity) whereas lymphocytes concentrate more on generating a humoral response through antibodies to deal with the virus. Larger organisms, such as fungi and parasites, elicit an eosinophilic response as the immune system is recruited in to deal with these organisms.

Superficial versus deep

An understanding of the superficial and deep facial layers of the head and neck is paramount to comprehending how infective swellings evolve and progress within the head and neck and to knowing which infections pose the greatest threat to the patient.

On a fundamental level, the peri-vertebral muscles of the spine are bound to the vertebral column by a layer of dense connective tissue, the pre-vertebral (deep) fascial layer. In front of the spine, the thyroid gland, trachea and oesophagus are bound within the pretracheal (intermediate deep) fascia which allows the pharynx to glide on swallowing. All other structures of the neck superficial to these blocks are bound by a further layer of fibrous tissue – the investing (superficial layer of deep) fascia which extends up onto the lower half of the face, attaching to the zygomatic process on each side and inferiorly onto the clavicles. It is so called because it is the only fascial layer to encompass structures within its substance viz. the sternocleidomastoid and trapezius muscles, and the submandibular and parotid salivary glands.

A final less important layer of fascia – the superficial fascia – circumscribes the entire neck, lying just below the subcutaneous tissues.

Infective swellings superficial to the investing fascia (i.e. subcutaneous) generally present less of a threat as they present early, are easily reached when drainage is required and the likelihood of spread is low. Swellings in the deep compartments of the neck, by contrast, risk lying undetected, are difficult to access once detected and carry the significant risk of rapid spread within the fascial compartment or via the haematogenous route.

Allergic swellings

The head and neck is a common site for type I hypersensitivity reactions to manifest. The inflammatory swellings that result will range from mild tongue or facial swelling to 'full-blown' anaphylaxis with laryngeal swelling and respiratory compromise. Type I hypersensitivity reactions occur when a previously-sensitized individual meets with a specific exogenous antigen. The antigen becomes bound to immunoglobulin E (IgE) and subsequent mast-cell degranulation results in a flood of histamine, eosinophil chemotactic factors and other 'slow-releasing substances of anaphylaxis'.

Autoimmune swellings

The head and neck is also a target for type III hypersensitivity reactions due to autoimmune disease. In type III hypersensitivity reactions, immune complexes form between the antigen and IgG/IgM immunoglobulins and deposition of these complexes within the tissues triggers the complement cascade and neutrophil activation. The resultant swelling is far more indolent and chronic than is encountered in type I reactions.

Site-specific swellings

We can now consider the specific conditions that will present an inflammatory-looking swelling at the main anatomical sites of the head and neck.

Face

Infective facial swelling:

Dental abscess – the majority of inflammatory-looking swellings over the lower face will be a result of dental infection. Although the dental infection itself is usually chronic (and often silent), the facial swelling is usually an acute inflammation in response to the infection finally erupting through the cortex of the maxilla or mandible. The patient will present with a diffuse tender swelling which will be overlying and attached to the maxilla or mandible (Figure 1) and thus will be poorly mobile; this differentiates it from a skin abscess. Management requires antibiotics and surgical, intra-oral drainage with removal of the problematic tooth root.

Maxillary sinusitis – by contrast, is noteworthy that although mid-facial swellings are commonly attributed to sinus pathologies, it is in fact very rare for maxillary sinus infections to erode the anterior sinus wall and present as facial swelling, presumably because there is a path of lesser resistance through its natural ostium. When sinus pathology does result in mid-facial swelling, it is invariably an indication of underlying neoplasia.

Lymph nodes – a discrete cold swelling of the face in a child should raise the suspicion of chronic granulomatous infection, and in particular, mycobacterium. 'Atypical' mycobacteria (scrofula, avium, malmoense) are the usual culprits but it is worth remembering that tuberculosis infection is on the rise again.[1] Being chronic, the swellings are usually non-tender with little associated oedema and it is usual to have a pale blue-purple discolouration to the overlying skin which is pathognomic. Diagnosis is a clinical one as organisms are rarely grown from needle aspirates. The Mantoux test will usually be negative in non-tuberculous infections although false-positives do occur. Management is by surgical excision. Incision and drainage should *not* be attempted as it risks establishing a chronic discharging sinus. More recently, prolonged preoperative oral macrolide antibiotics have proven useful in reducing the size of the inflammatory swelling prior to surgical removal and reducing the need to excise overlying skin.

Allergic facial swelling:

Facial oedema – the main differential diagnosis for acute facial oedema lies between anaphylaxis and angioedema and whilst these two conditions are often considered similar, the terms should not be used interchangeably. Indeed when their presentations are considered, differences are quite apparent. Anaphylaxis is a systemic inflammatory reaction, usually mediated by IgE. Upon contact with an anaphylatoxin to which the individual has previously been sensitized, a type I