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Olecranon bursitis: a systematic overview

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Abstract

Background: Olecranon bursitis is a common condition where the bursal cavity, superficial to the olecranon, becomes inflamed. This can occur either with or without infection and has been given pseudonyms relating to the repeated minor trauma from external pressure that often predisposes. As a result of the multiple aetiologies, olecranon bursitis can present to any medical specialty with reasonable frequency and, although many therapies are described, a single, evidence-based and standardized treatment pathway is not well described.

Methods: We summarize the key points within the literature and subsequently propose an evidence-based treatment pathway.

Results: Relevant evidence is presented from appropriate publications to add rational to existing decision-making processes, together with personal experience and suggested operative bursectomy techniques from an established upper limb surgeon. The common and significant aetiologies are summarized and, in particular, red flag symptoms are highlighted by way of warning to the unsuspecting investigator.

Conclusions: The conclusion is provided in diagrammatic form, providing a suggested treatment pathway from history and examination through to operative intervention.

Keywords

Bursitis, bursectomy, inflammation, olecranon, orthopaedics, trauma

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Introduction

Olecranon bursitis is characterized by an abnormal increase in the volume of fluid within the bursal cavity. The bursal lining is a poorly vascularized synovial membrane that has a low coefficient of friction, thereby allowing the bony olecranon to glide under the skin during flexion and extension of the elbow (Figure 1). This superficial position and limited vascularity makes the olecranon bursa particularly vulnerable to injury and inflammation. It is this limited vascularity that is the proposed reason for infection via a transcutaneous route, rather than via haematogenous spread, even when no obvious wound is present.[1] *Staphylococcus aureus* predominates as the causative bacteria, with β-haemolytic strep also being common. Of the 150 human bursa, the olecranon is the most commonly affected by an inflammatory process.[2]

Despite the frequent presentation of this condition to both primary and secondary care, there is no randomized control data available and, with multiple small number studies often providing conflicting findings, there is currently no consensus on treatment.

We review the literature, summarize the key features and suggest appropriate treatment pathways, at the same time as emphasizing the importance of recognizing or excluding alternative and sometimes more sinister diagnoses. Accordingly, we present our practice and observational outcomes.

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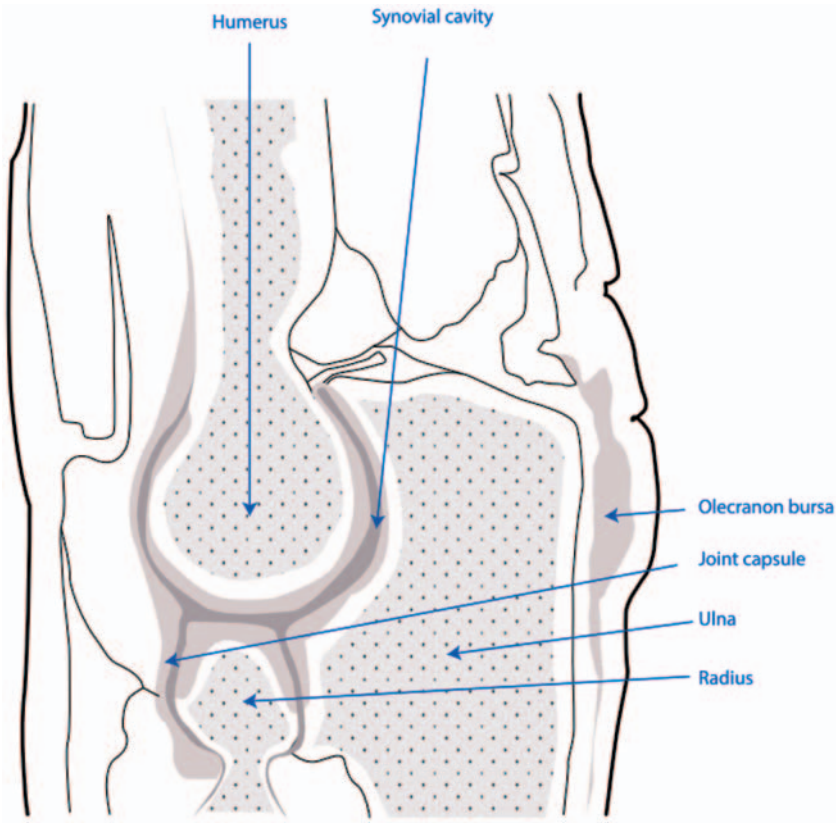

Figure 1. Cross-sectional anatomy of elbow joint.

Aetiology

Initially, it is important to recognize those red flag signs suggesting a neoplastic pathology mimicking a simple olecranon bursitis. Such signs include a rapidly expanding growth, failure of initial treatment, weight loss and prior history of neoplasia. Under these circumstances, appropriate referral, investigation, biopsy and treatment should be undertaken. Figure 2 shows a clinical image of a recurrent sarcoma that was initially resected as a benign olecranon bursitis.

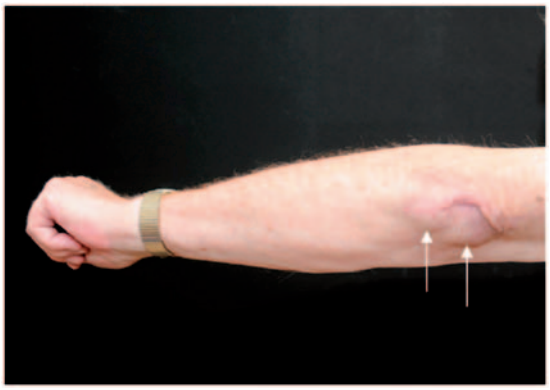

Figure 2. Recurrence of sarcoma indicated by arrows following initial debridement.

Trauma

Most commonly, olecranon bursitis is a non-infective, post-traumatic, inflammatory response to repetitive, minor trauma.[3] Historically, this has prompted the introduction of a variety of pseudonyms, including 'students elbow' and 'plumbers elbow'. An isolated traumatic event can also initiate the inflammatory cascade but, under these circumstances, an underlying fracture must be excluded. In the absence of blunt trauma, a penetrating foreign body must also be considered as a potential cause of a traumatic bursitis.

Medical conditions

Olecranon bursitis is known to be associated with common medical conditions, either directly or as a consequence of immunosuppression secondary to therapeutic intervention. Relatively common conditions with a direct association include diabetes mellitus,

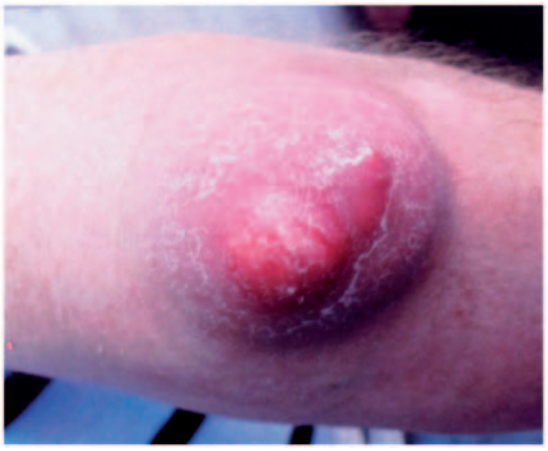

Figure 3. Infected olecranon bursitis in the presence of gouty tophi.

gout (Fig. 3), rheumatoid arthritis, alcoholism and HIV.

Some conditions, which include inflammatory bowel disease, respiratory disease and polymyalgia rheumatica/giant cell arteritis, are often treated with immunosuppressant therapy, and this will increase the risk of developing infective bursitis.[4]

Diagnostics

The list of known causes and associated risk factors summarized above is neither summative, nor exhaustive, although early discrimination of septic from aseptic bursitis has been demonstrated to impact upon the duration of treatment required.[5]

Unfortunately, there is accepted difficulty in confirming an infective cause on history and examination alone.[1,5] Confirmation with aspiration and urgent gram stain and culture can be considered as the gold standard because false positive rates are low provided a suitable aseptic technique is followed.[5]

Other proposed observations and tests have provided a wealth of conflicting evidence, mirroring the frequent overlap between septic and aseptic causes. Swelling, erythema and tenderness, with preserved elbow movement, are universal common features. Fever is present in up to 77% of septic cases[1] and erythema present in 63–100%;[6] therefore, both are only moderately sensitive for infection.

Analysis of bursal aspirate is suggested by many and common tests include differential white cell count, comparative glucose concentration and protein levels. When serum glucose is compared with aspirate glucose concentration, a >50% disparity is diagnostic for infection.[7] This test in isolation, however, has been partially discredited, with a false negative rate of 9% being reported.[1] Protein and complement levels showed no statistically significant difference and have low predictive value.[7]

Leukocytosis greater than 10,000 mm^3 is likely diagnostic for sepsis, however, septic aspirate cell counts have been reported from 690 cells/mm^3 to 79,400 cells/mm^3[1] and non-infective cases were found to range from 50 cells/mm^3 to 3450 cells/mm^3.[8] This has resulted in false negative reports in up to 12.5% of cases in one study[1] and 31% in another.[9]

A predominance of polymorphs within the aspirate of greater than 50% has been shown as a reliable feature in identifying infection. Monocytes predominate in non-infective samples, comprising >50% of the cell count.[10]

It is worth noting the findings of Hassell et al. who reported a case of seven patients with rheumatoid associated olecranon bursitis.[11] They found aspirate cell concentrations in keeping with septic bursitis; however, all were culture and Gram-stain negative in the absence of antibiotics. Promising results were demonsrated with the sclerosing action of intrabursal tetracycline for rheumatoid bursitis, without the skin atrophy, secondary infections and sinus formation that have been reported as a result of steroid injection.[8]

Given the lack of a single highly sensitive and specific test, a detailed history to identify general and specific risk factors focuses on the patient's occupation, hobbies, medical history, medication, family history and recent trauma. Recurrent or nonresolving olecranon bursitis is of particular importance, raising suspicion of retained foreign body, antimicrobial resistance or incorrect diagnosis. Systemic symptoms should be explored, including fevers, anorexia, lethargy, weight-loss and night-sweats, which are more suggestive of an infective (or rarely malignant) origin.

A thorough general examination of the patient should be followed by specific examination of the affected area and contralateral elbow. Quayle and Robinson reported a case series of 11 olecranon process excisions for non-infective bursitis, where the patients had either an olecranon spur or abnormally prominent olecranon.[12] A 100% cure rate was achieved. The examiner focuses on the size of the swelling, its consistency (soft, firm, hard), fluctuancy, associated erythema, skin temperature, any lymphadenopathy and the characteristics of movement in the elbow joint: specifically range and pain. It is worth noting that our experience mirrors the documented consensus[13] that elbow movement is preserved in bursitis, as opposed to the restriction associated with septic arthritis.

Investigations are preferably performed prior to antibiotic therapy and should include baseline observations, plain film radiographs of the elbow and basic

blood tests, including full blood count; urea and electrolytes; calcium, uric acid, glucose levels; and inflammatory markers such as C-reactive protein and erythrocyte sedimentation rate. The value of blood cultures has been debated, with culture positive rates of 4%,[2] 19%[1] and 30%.[4] Clearly, it would be sensible to reason that, in the presence of systemic features of infection, a bacteraemia is more likely to be diagnosed from blood culture.

Wherever practicable, prior to the administration of antibiotics, needle aspiration of the bursa should be performed cautiously. An aseptic technique will provide a sample for urgent microscopy, culture and sensitivity, at the same time as providing pain relief by reducing the bursal pressure. Violation of the elbow joint by the needle should be avoided to prevent secondary iatrogenic septic arthritis. Ultrasound guidance may be used to assist accuracy. A differential white cell count and determination of glucose levels is also advocated because these are helpful predictors of infection in the absence of a positive Gram stain or culture, as described above.

Further imaging with magnetic resonance has been described previously[14] (Fig. 4) as a sensitive negative predictor of infective bursitis. The absence of bursal and soft tissue enhancement is reported as a reliable indicator of non-infective bursitis, whereas its presence is nonspecific and can be present in up to 76% of cases of any cause.[14]

Treatment options and complications

Non-infective

Most commonly, the bursitis will be inflammatory and non-infective. Under these circumstances, symptomatic treatment with elevation, splintage, ice and anti-inflammatories is regarded as the option of choice,[15] although we acknowledge that most do not require elevation in a sling.

Routine aspiration and injection of non-infective bursitis with steroid and local anaesthetic has been advocated[15,16] as an appropriate treatment to shorten the natural history; however, Smith et al.[17] oppose this view based on work by Söderquist and Hedström[18] who reported a 10% risk of infection by contamination.

Given the controversy, we have reviewed the two studies that have reported results obtained after steroid injection.

Weinstein et al. reported a group of 47 confirmed cases of non-infected olecranon bursitis where all were aspirated but only 25 were infiltrated with corticosteroid.[8] This infiltration was performed 7 days post initial aspiration after sample sterility had been confirmed. A reduction in symptom duration was statistically significant for the steroid group compared to the control group. There were, however, complications associated with the steroid group, with two re-presenting with septic bursitis and five suffering from overlying skin atrophy.

Smith et al. subsequently reported a controlled and blinded, prospective trial where the outcomes of 42 aseptic olecranon bursae were divided between four treatment groups.[17] Group 1 received infiltrated steroid with oral nonsteroidal anti-inflammatory drugs (NSAID). Group 2 received infiltrated steroid but with a placebo oral agent. Group 3 received only NSAID and group 4 received only an oral placebo agent.

The study failed to demonstrate a statistically significant reduction in symptom duration when adding NSAIDs to the treatment regimen. Infiltrated corticosteroid did, however, show a statistically significant reduction in symptom duration. Furthermore, there

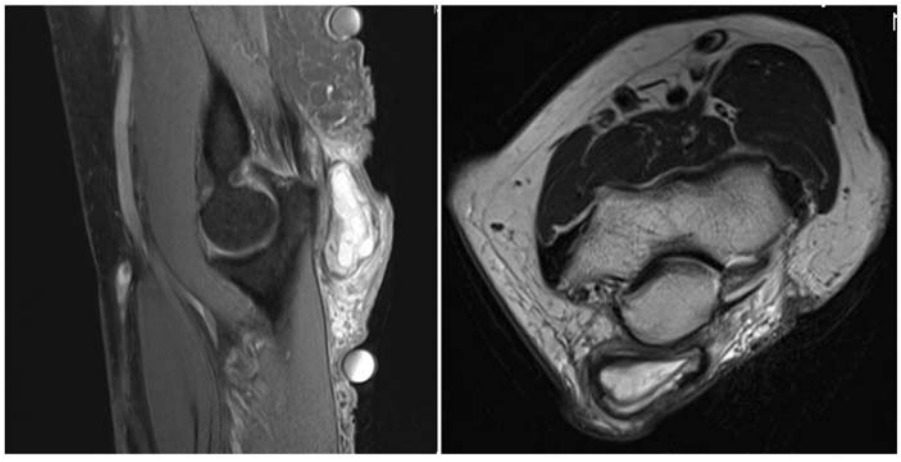

Figure 4. Magnetic resonance images of enhancing olecranon bursitis.

were no cases of secondary septic bursitis or skin atrophy reported.

Given this evidence, it would appear that NSAIDs may only be helpful for symptomatic relief, whereas corticosteroids, although effective, present a risk of secondary infection and should therefore be used with caution.

Where a large and painful bursitis is clinically diagnosed as inflammatory, we will often aspirate in anticipation of beneficial pressure relief. A sample is sent for culture but it is not our practice to inject steroid.

When the aetiology is secondary to a known medical pathology and treatment for both the known pathology and bursitis is combined, there is no published evidence to confirm a reduction in the duration of the associated bursitis. It would appear logical, however, and there might be ethical difficulties related to withholding treatment as part of a controlled study.

Where there is non-infective bursitis in the presence of an olecranon spur, it is popular opinion that operative excision of the spur can significantly reduce the risk of recurrence, with two small studies reporting success.[19,20]

Infective

For those less common episodes of infective olecranon bursitis, many treatment options have been proposed. Ho and Su have provided a classification system where clinical signs denote whether a bursitis is mild, moderate or severe.[5]

- Mild disease: local inflammation with no systemic signs.
- Moderate: significant local inflammation +/− mild systemic signs.
- Severe: intense peri-bursal cellulitis +/− infected wound with systemic signs, including pyrexia or rigors, or a serum leukocytosis >10,000/mm^3.

Importantly, this classification system was devised for observing the duration of antibiotics required to achieve sterility of infected bursal aspirates. The study by Ho et al. specifically excluded patients with 'underlying host defects, such as diabetes mellitus, renal or hepatic disease, underlying malignant disease or rheumatic disease because they are 'more prone to infection and may not respond in the same manner'.[5]

This is supported by Garcia-Porrua et al. who demonstrated a longer antibiotic duration was necessary for immunocompromised patients.[1]

Given the common association between infected bursitis and co-existing medical conditions, a modification to the Ho–Su classification[5] is proposed, where the presence of co-morbidity likely to affect healing or immune response, increases the severity by one level within the Ho–Su classification.

Proposed treatment options are subclassified as to whether they are performed acutely, or as a delayed procedure following antibiotic therapy. Significant swelling with pointing is considered an indication for incision and drainage, only once attempted aspiration has failed because of loculation.[9]

A large retrospective series reviewing the recurrence rates in 237 episodes of infected olecranon bursitis, was performed by Perez et al.[2] They found single stage acute bursectomy to be associated with increased recurrence rates compared to multistage open procedures with delayed primary closure. Their study included a 91% acute bursectomy rate (olecranon and prepatella), 41% of which were single stage; however, no information was provided on wound healing duration or complications.

Unfortunately, there are no randomized comparative studies comparing outcomes after acute multistage bursectomy with delayed single stage procedures.

Degree et al. reported a retrospective review of 37 cases of open bursectomy for chronic bursitis.[15] Patients with a gouty or rheumatoid cause were excluded from the study. Some 43% healed without complication, 27% had delayed healing with excessive exudate and 22% suffered recurrence, 50% of whom required further intervention.

Antibiotic duration and administration sparks controversy. Some have advocated outpatient treatment with prolonged oral antibiotics, with or without percutaneous needle aspiration,[5,21–23] whereas others[2,24] have described immobilization and antibiotics, or hospitalization with surgical drainage[18] or suction irrigation.[25] Furthermore, there is considerable variation between suggested antibiotic protocols, with some advocating up to 4 weeks of intravenous antibiotics in non-operative management.[18,22,23,25] Where operative intervention is employed, adjuvant therapy regimens describe short intravenous courses, followed by oral treatment for up to 2 weeks.[18,21]

In mild cases of infective bursitis, several studies advocate needle aspiration before commencing antibiotics as an outpatient; however, differentiating between mild and more severe cases is open to error, despite no cases of inter-observer error in the original study.[5] Treatment failure rates of between 9% and 32% for mild bursitis, and 48% to 51% for severe bursitis, are reported.[9,21,23]

It has been suggested[2] that failure to intervene surgically is the most potent independent risk factor for recurrence (14.6% versus 80%), although, without the evidence provided by a large number randomized

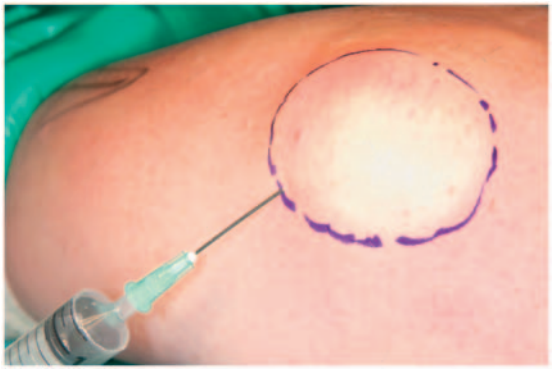

Figure 5. Infiltration of 5 mL of normal saline to identify and delineate bursal sac.

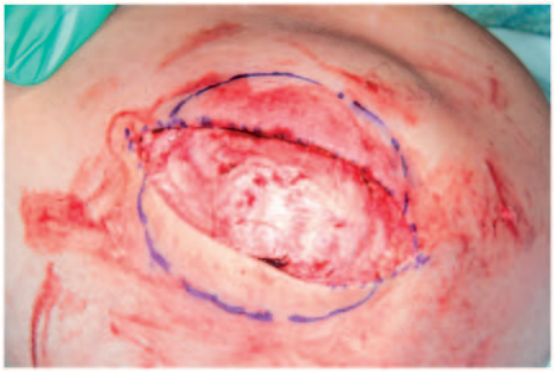

Figure 7. Skin excised with bursa visible in floor of wound.

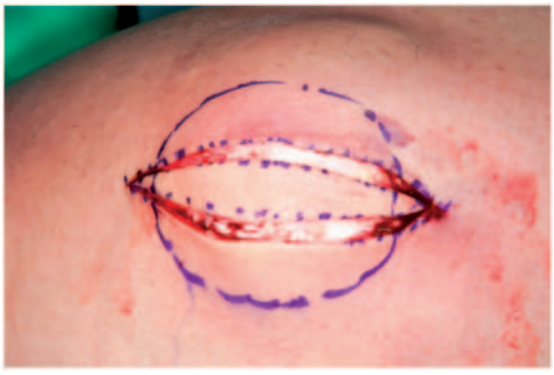

Figure 6. Elliptical skin incision.

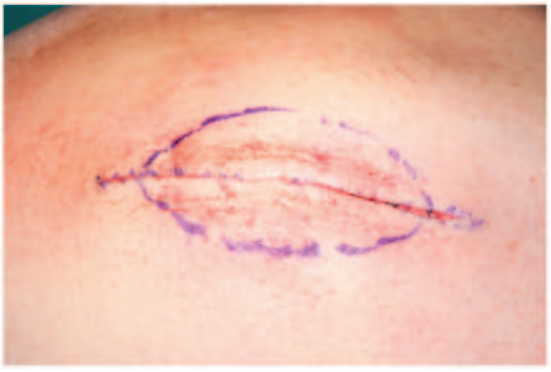

Figure 8. Skin closure demonstrating elliptical conversion of spherical skin marking.

control trial, it is inappropriate to advocate surgery for all.

This suggestion was proposed with adjuvant treatment with either a short or long course of antibiotics. Following a retrospective analysis of 343 episodes of severe infective bursitis requiring hospitalization (237 olecranon, 106 patellae), the following conclusions were drawn;

- One-stage bursectomy and closure reduced in-hospital stay by 4 days.
- Intravenous antibiotics were not required in patients with normal gut function because there was no significant reduction in infection duration with intravenous adjuvant antibiotics.
- Recurrence rates were not improved with a more than 7-day antibiotic course.
- Bacteraemia was identified in only 4% of patients.
- The only independent risk factor for recurrence in postoperative patients was immunosuppression.
- Recurrence rates secondary to immunosuppression were unaffected by treatment modification.

Within the umbrella of 'surgical excision', we include the techniques of arthroscopic versus traditional open procedures, with arthroscopy gaining merit based on reduced theoretical risk of wound complications. We acknowledge that the arthroscopic approach is quite uncommon and, once again, there is no controlled-trial data to support this, although multiple, small number case series report few complications for bursectomy at the olecranon and patella.[26,27] The feared complications in open excision are wound dehiscence, chronic sinus and skin necrosis as a result of the watershed midline blood supply. Arthroscopic techniques aim to avoid this with port sites located distant from the midline.

Our practice

It is our practice to take a focused history and a careful examination at presentation, initially not only to exclude red flags, but also to differentiate between infective and non-infective causes, with appropriate investigations including inflammatory markers and radiographs. The radiograph intended to demonstrate

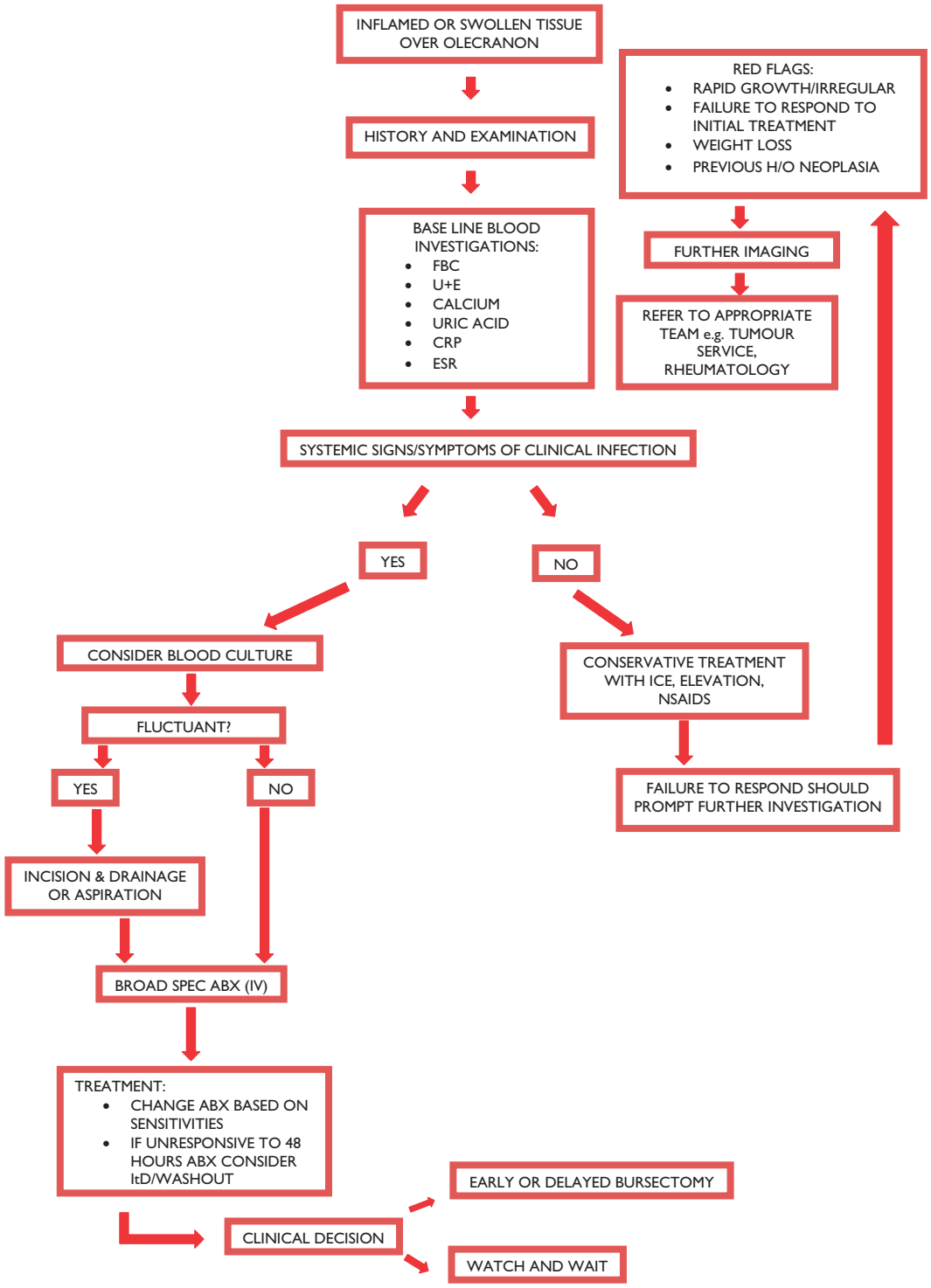

Figure 9. Proposed treatment algorithm.

osteomyelitis if present, an olecranon spur, a fracture, a radio-opaque foreign body and possibly evidence of gout. In those patients with a high index of suspicion for infection and in the presence of fluctuation, aseptic bursal aspiration is performed for urgent microscopy, culture and sensitivity. After aspiration, broad spectrum antibiotics covering a probable *S. aureus* infection (>90% of infected cases)[23] are commenced. If the infection is considered to be mild with either minimal or no collection, oral treatment can be undertaken as an outpatient. The antibiotic regimen will then be modified in response to antimicrobial sensitivities if

available. If more severe (e.g. in the presence of a significant surrounding cellulitis), an obvious collection, systemic evidence of infection or concurrent immunosuppressive disease, then the patient is admitted for intravenous antibiotics and high arm elevation with a Bradford sling. Open drainage is generally undertaken for those cases that (i) have an obvious, fluctuant collection, which is either felt unlikely to or has failed to respond to antibiotics, and (ii) where the patient is clearly systemically unwell secondary to the infective process.

Once the infection has settled, and particularly when a history of recurrent bursitis is present, an interval bursectomy is undertaken.

Occasionally, under these circumstances, the bursa is no longer obvious and, to delineate its boundaries, our practice is to inflate with saline at the start of the procedure, usually 5 mL (Fig. 5). If the skin is of good quality, then a midline incision can be made, although often it is densely scarred to the bursal surface. In attempting to dissect between the layers, the tissue is often compromised, resulting in wound dehiscence, which may require further surgery or prolonged outpatient dressing therapy. In an effort to avoid this and because of the surplus tissue often present, an ellipse of skin (Fig. 6 and 7) can be taken, thereby allowing dissection through higher quality tissue. The wound is then far less likely to fail. Where a bony spur is present, this is usually excised simultaneously. At closure, the elliptical incision comes together to provide a well vascularized midline scar (Fig. 8)

By way of simplifying the treatment pathway, we propose the sequence of investigation and treatment illustrated in Fig. 9, although we caution that no substitution can be made for careful and frequent clinical review

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Declaration of conflicting interests

None declared.

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