A 22-year old male patient from rural background presented to our tertiary care orthopedics outpatient department after being referred from the primary health care center. The patient had a gradually increasing, non-radiating, dull pain in the left knee for the past 3 months. The pain worsened after exercise and walking, and was relieved on taking non-steroidal anti-inflammatory drugs. There was no history of swelling, fever, loss of appetite, trauma, malaise, weight loss, pain, or swelling at any other site. On examination, the general condition of the patient was fair except for mild pallor and slight limp on walking. On local examination, the patient had tenderness in the left knee joint, centered in the region of tibial condyle, without any other sign of inflammation or adjacent lymphadenopathy. The active range of motion was limited and the passive range of motion was full in all directions but painful. The rest of the orthopedic examination was normal. The patient was advised plain radiographs of the knee (Figure 1) followed by contrast-enhanced magnetic resonance imaging (MRI) (Figures 2 and 3).

**What is your diagnosis?**

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Figure 2. (A) T1W sagittal MRI sequence of the left knee of the patient showing a well-defined cystic lesion in tibial metaphysis, with a central hypointense core, surrounded by a hyperintense rim of granulation tissue ("penumbra sign"). (B) On DESS sequences, the lesion appears hyperintense with an intralesional hypointense debris; also noted is effusion within the patellofemoral joint (white arrow). (C) Axial T2W sequences showing hyperintensity within the lesion. (D) MEDIC sequences showing no evidence of intralesional hemorrhage or calcification.

Figure 3. Postgadolinium fat–saturated T1W sequences, showing peripheral rim enhancement of the lesion and the overlying inflamed subcutaneous tissues.
Brodie abscess is known since its first description by Sir Benjamin Brodie, a surgeon in St. George’s Hospital, London, United Kingdom, in 1832. Although unaided by radiography or bacteriology, the clinical and surgical description of bone abscesses given by him was accurate and precise. He reported 8 cases having disease in tibia, which was characterized by mild-to-moderate local symptoms. All cases were treated with trephine osteotomy, and pus-filled cavities were drained. The radiologic description of the brodie abscess began in 1906 with Thomson series and was further detailed by Brickner in 1917 and Brailsford in 1938.

Radiologically, it is characterized by small metaphyseal lytic lesions with well-defined margins, most often in tibia, usually in young patients prior to epiphyseal closure. It has a tendency to cross the growth plate and may extend into diaphysis in a snakelike fashion, resulting in the so-called “serpentine sign.” An area of sclerosis, a sequestrum, may be seen in the center of the lytic lesion. MRI is the most sensitive investigation to exactly localize and diagnose a brodie abscess. The central part of the lesion appears hypointense on T1W sequence and hyperintense on T2W sequence. On T1W sequence, the central hypointensity is surrounded by a rim of hyperintensity, known as penumbra sign, which is characteristic but not pathognomonic for a brodie abscess. This hyperintensity is due to the protein-rich granulation tissue, which is further surrounded by a hypointense rim on all sequences, corresponding to a sclerotic bone. The lesion is surrounded by edema, which appears hyperintense on T2W sequence and hypointense on T1W sequence. On post-contrast sequences, the lesion shows a typical peripheral enhancement.

A Brodie abscess is a great mimicker of a number of diseases. Metaphyseal eccentric lesions need to be differentiated from nonossifying fibroma or chondromyxoid fibroma, metastatic neuroblastoma, or stress fractures. An epiphyseal lesion may simulate chondroblastoma, clear cell sarcoma, fungal osteomyelitis, tuberculous osteomyelitis, aneurysmal bone cyst, pigmented villonodular synovitis, degenerative changes, degenerative erosions, intraosseous ganglion, giant cell tumor, or gout.

If the diagnosis is clear on the basis of characteristic clinical and imaging findings and laboratory results, treatment with antibiotics alone may be undertaken without biopsy. The failure of symptoms to resolve after a 6-week course of antibiotics or the worsening of the condition during treatment should prompt the clinician for definite tissue and/or bacteriologic diagnosis, followed by surgical treatment and use of appropriate antibiotics. Other indications for surgery are impending sinus formation or drainage into a synovial joint.

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