specific disease excluding Addison's disease.[5] Obviously, the features of our patient were consistent with this subcategory. The PAS 3 is also known to be associated with other organ-specific and non-specific disorders such as sarcoidosis, celiac disease, myasthenia gravis, rheumatoid arthritis, and Sjogren's syndrome. An associated gastric carcinoid syndrome was also reported. Autoimmunity, environmental, and genetic factors are the 3 major components of the pathophysiology of PAS. The presence of circulating organ-specific and cellular autoimmunity in patients with PAS 3 provides the strong evidence for the autoimmune pathogenesis of the disorder. Environmental precipitator/s such as viral infection, for example may exaggerate an ongoing immune response and precipitate glandular failure. In this regard, the links between congenital rubella infection and type 1 diabetes mellitus, and hypothyroidism is well known. Polyglandular autoimmune syndrome is often observed in subjects of the same family and suggested an autosomal dominant trait with variable penetrance. Certain genetic markers have been found to confer susceptibility to PAS 3, and the frequently described haplotypes include DR-B*04/ DQA 1*0301/DQB1*0302, HLA-DR B1*13, DRB1*1104, and DRB1*0401.[5] A significantly higher frequency of DR3 and DR4 antigens were also detected in patients with PAS 2 and 3 compared with controls in a recent study. Our patient exhibited A26, B8 and DR3 on tissue typing. It is of interest to note that the combination of A26 and B8 has recently been found to be an autoimmune favoring haplotype in Indians.

Evidently, this is an interesting combination of glandular hypo and hyper-function producing constellation of features of PAS type 3, and PHPT in the same patient. Although, the latter was the only apparent manifestation of the endocrine hyper-function, the patient was adequately investigated for a possibility of an associating or evolving MEN syndrome. However, a regular monitoring of the initially elevated serum gastrin would certainly remain imperative in the future management. Unfortunately, the patient lost to follow-up in the last 6 months, and relatives refused to be evaluated. Finally, the association of MEN and PAS is rarely reported in the English literature. We only managed to trace a single case report in which PAS was characterized by mucocutaneous candidiasis, vitiligo and macroglossia, and occurred together with Cushing's disease and PHPT (MEN type I).

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From the Department of Internal Medicine, Mafraq Hospital, Abu Dhabi, United Arab Emirates. Address correspondence and reprint requests to Dr. Haider M. Al-Attia, Department of Internal Medicine, Mafraq Hospital, Abu Dhabi 2951, United Arab Emirates. Tel. +971 (50) 6137795. E-mail: haideralattia@hotmail.com

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Actinomyces meyeri isolation from synovial fluid of a patient with metastatic squamous cell lung carcinoma

Emel S. Cetin, Selcuk Kaya, Mustafa Demirci, Buket C. Aridogan.

The genus actinomycetes consists of several species of gram-positive, non-spore forming bacteria, which grow as obligate or facultative anaerobes. *Actinomyces* organisms are important constituents of the normal flora of mucous membranes. These organisms may produce infection after local trauma, surgery, or aspiration. The main forms of actinomycosis are cervicofacial, thoracic, and abdominal; most cases are due to *Actinomyces israelii,* whereas Garduno et al[1] report occasional implication of other *Actinomyces species*.

We rarely isolate *Actinomyces meyeri* (*A. meyeri*) in cases of actinomycosis. However, an increasing number of cases recognize its potential pathogenicity. In contrast to other species of *Actinomyces, A. meyeri* often causes pulmonary infection and shows a tendency for hematogenous dissemination. Involvement can include any organ of the human body so that a wide range of symptoms may be present. Although, multiple organs are involved, the outcome for these patients is excellent when we administer penicillin for several months and perform surgical procedures when necessary. As actinomycetes are rarely opportunistic agents in immunocompromised patients, the disease deserves special attention in those patients.[2] Here, we report a case of actinomycosis with an uncommon localization that was due to *A. meyeri* in a patient with metastatic squamous cell lung carcinoma.

In April 2004, a 44-year-old woman complained of severe motion-dependent pain on her right hip. There were no signs of infection, and her complaints were considered to be due to lumbal disc herniation. In July 2004, she had to be hospitalized due to progressive pain by walking and proliferative plasma cell infiltration was detected from the biopsy of right caput femoris. A pathological fracture occurred after radiotherapy was applied to the femur. For this reason, Thompson prosthesis was applied. In November 2004, diffuse swelling on her left knee, on arcus mandibulae, and left frontal bone existed. Pathological examination of the biopsy from the left knee revealed metastatic squamous cell carcinoma, and she was transferred to the oncology service for chemotherapy planning and the investigation of the origin of the malignancy. The thoraco-abdominal CT scan revealed a soft tissue lesion of 65.36 mm size at the inferior lobe of left lung. On the posterior side of right sacroiliac joint, a metastatic soft tissue lesion of 70.42 mm size, causing destruction on the bone was detected. Furthermore, certain lytic and sclerotic lesions we observed on corpus vertebrae and posterior components of it. According to these findings, the diagnosis was determined as squamous cell lung carcinoma with metastatic bone lesions. She underwent a chemotherapy protocol consisting of cisplatin and etoposid. In addition, ampicillin/ sulbactam we administered as empirical therapy. Meanwhile, synovial fluid obtained during biopsy was sent to our laboratory for microbiological examination. The obtained material was cultured aerobically and anaerobically; no microorganism were seen on gram stain. After 72 hours of incubation at 37°, anaerobic blood agar plates (Brain heart infusion base supplemented with 5% sheep blood, 0.5% yeast extract, hemin, L-cysteine and vitamin K_1) yielded colonies of gram-positive, catalase negative bacilli in pure culture, subsequently identified as *A. meyeri* with use of the Rapid ID32 A system (bioMérieux, France). The isolated strain was found to be sensitive to all drugs tested by using ATB ANA system (bioMérieux, France). (Penicilin, amoxicillin, amoxicillin/klavulanic acid, piperacillin piperacillin/tazobactam, ticarcillin/ klavulanic acid, cefoxitin, cefotetan, imipenem, clindamycin, chloramphenicol, metronidazole). After 10 days of chemotherapy, thrombocytopenia and neutropenia existed. In December 2004, her condition deteriorated and she died due to cardio-pulmonary arrest.

The location of infections due to *Actinomyces species* is the cervico-facial region in 50-65% of the cases. The most frequently encountered germ is *A. israeli*, observed in 85% of the cases. Presenting clinical manifestations of actinomycotic infections are confusing as they often mimic other disease processes or even neoplasms. Diagnosis may be difficult due to this confusing clinical presentation combined with the fastidious nature of the organism in culture. We require a high index of suspicion to make an accurate and timely diagnosis and to institute the appropriate antibiotic therapy.[3] Recently, emphasis is on the etiologic importance of anaerobic microorganisms in bone and joint infections in certain settings. These settings include recovery of *Actinomyces species*, hematogenously acquired infection, the presence of anaerobes in pure culture, and prosthetic-joint infection. Bussiere et al,[4] reported 3 observations of osseous and articular actinomycosis. The associated soft tissue abscess with osseous lesions of the spine and limbs, and one of the patients had septic arthritis due to this bacterium: and obtained *A. meyeri*, from infectious foci in the 3 cases. The authors insisted on the rare occurrence, at that moment, of osteo-articular actinomycosis outside the maxillo-facial area. Here, we report an *A. meyeri* infection, which presented as arthritis. This case is of particular interest due to the extra pulmonary localization, and the rare species isolated. We could not isolate this bacterium from blood and sputum samples of the patient. In this case, we identified no underlying source of infection, so we suppose that an unrecognized disruption of the gastrointestinal mucosa could have been the portal of entry for *A. meyeri*, and then a hematogenous spread of the infection took place. The nature of the organism and its location to a joint are unusual features of this case. As the infection in the patient reported herein occurred only 4 months after hip surgery, we postulate a hematogenous spread from the patient's own bacterial flora.

We did not receive any pathological data, and material for bacteriological culture from the lesions on os mandibulae and frontale existed at the same time with the lesion on the left knee, so we are not able to make a judgment that these lesions were certainly of malign or bacteriological origin. But, we must emphasize the fact that actinomycosis can present in a variety of forms and may mimic other infections or even neoplasms.[5] The diagnosis of severe actinomycosis parallel to neoplasia leads to speculation of a possible fortuitous association. To strengthen the hypothesis that we should suspect underlying conditions such as immuno-suppression in such disease, we report another case of actinomycosis associated with a malignant disease, namely, a squamous cell lung carcinoma. So this article stresses the importance of considering the diagnosis of the disease especially in immunsupressed patients with malignancies.

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From the Department of Microbiology and Clinical Microbiology, Suleyman Demirel University, Isparta, Turkey. Address correspondence and reprint requests to Assistant Professor Emel S. Cetin, Suleyman Demirel Bulvari Salali Apt. No: 60 Daire: 23 32200 Isparta, Turkey. Tel. +902 (46) 2112081. Fax. +9 (246) 237024. E-mail: seslicetin@med.sdu.edu.tr

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Utility of cefoxitin resistance determination by disk diffusion method for routine detection of methicillin resistant *Staphylococcus aureus*

Bodh R. Panhotra, *MD, PhD, MNAMS,*
Anil K. Saxena, *MD, FRCP,*
Abdulrahaman S. Al-Mulhim, *FRCS, FICS.*

M*ethicillin resistant Staphylococcus aureus* (MRSA) cause serious hospital acquired infections leading to high mortality, morbidity, and enhanced cost of patient management due to prolonged hospitalization. These strains are frequently resistant to multiple antibiotics, thus limiting the choice of antimicrobial therapy. Lately, there is an increasing trend of MRSA infections both in industrialized and developing countries. Prompt detection, identification, and confirmation of these strains by the clinical laboratories has a lot of infection control implications of rapid appropriate control measures to prevent its further spread in the hospital.[1] Resistance to oxacillin by the disk diffusion method is the standard criteria of MRSA detection. Recommendations to improve detection of MRSA include, incubation of disk diffusion test plates at lower temperature of 35°C for prolonged period, adding 2% sodium chloride (NaCl**)** to the susceptibility test medium and inoculation on Muller Hinton agar containing 4% NaCl with 6 µg/ml oxacillin.[2] Available confirmatory tests for MRSA such as mecA gene analysis, latex agglutination test detecting PBP2a proteins are expensive and facilities for them are not available with all the clinical laboratories. Recently, the National Committee for Clinical Laboratory Standards (NCCLS) recommended cefoxitin disk diffusion test using standard susceptibility testing conditions for prediction of mecA mediated resistance in MRSA.[3] The present study was carried out to compare the standard oxacillin salt agar confirmatory test to NCCLS cefoxitin disk diffusion test for detection of MRSA strains.

Methicillin sensitive Staphylococcus aureus (MSSA) (n=436), and MRSA (n=109) were isolated from the clinical samples received from the patients admitted during January-December 2004, to the 500 bedded King Fahad Hospital and Tertiary Care Center, Al-Hofuf, Eastern region of Saudi Arabia. These strains were identified by Grams stain, catalase test, tube coagulase test, DNase test, and Staphaurex latex agglutination test (Murex Bio Tech, Dartford, UK). *Methicillin resistant Staphylococcus aureus* strains were identified on the basis of resistance to oxacillin 1 µg disk (Becton Dickinson Co, Maryland, USA) by standard disc diffusion method[2] on Muller Hinton agar (Oxoid Ltd Basingstoke, Hampshire, UK) containing 2% NaCl. The plates were incubated at 35°C for 24 hours, and examined the zone of inhibition under the transmitted light for any colony or the growth film. The zone diameter less than 10 mm was considered as oxacillin resistance, and confirmed the oxacillin resistant strains as MRSA by inoculation on Muller Hinton agar with 4% NaCl, and 6 µg/ml oxacillin. Cefoxitin 30 µg (Becton Dickinson Co, Maryland, USA) disk diffusion confirmatory test was performed using standard test conditions on Muller Hinton agar and the plates incubated at 35°C for 24 hours. Zone of inhibition diameter less than 19 mm was considered as resistant and more than 20 mm as susceptible.[3] The results of salt oxacillin agar confirmatory test for MRSA were compared with the NCCLS cefoxitin disk diffusion test.

All the 436 MSSA were susceptible to cefoxitin (30 µg) with no discrepant results. Of the 108 strains of MRSA confirmed by inoculation on Muller Hinton agar containing 4% NaCl with 6 µg/ml oxacillin, all were resistant to cefoxitin (30 µg) by disk diffusion test. While one strain of MRSA, which had 9 mm zone of inhibition to one µg oxacillin disk but negative by salt agar