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Primary hypogammaglobulinemia presenting with prostate abscess and *Fusobacterium mortiferum* bacteremia in a 28-year-old man

KEYWORDS
Humoral immunity;
Primary immunodeficiency;
Common variable immunodeficiency disease;
Invasive pneumococcal infection;
Prostatitis

Dear Editor,

Primary hypogammaglobulinemia is a common primary immunodeficiency, which is often associated with severe bacterial infections such as invasive pneumococcal infection. However, the diagnosis is frequently delayed. Here, we present a case of primary hypogammaglobulinemia with presentations of prostatic abscess.

This 28-year-old man had been in his usual state of health until two months prior to this admission, when intermittent fevers with dysuria and urinary frequency developed. He sought medical attention at another hospital, where digital-rectal examination showed tenderness on palpation of the prostate. A computed tomography (CT) revealed prostatic abscess (Fig. 1A). A CT-guided drainage was performed and the blood culture yielded *Fusobacterium mortiferum*. A three-week course of ciprofloxacin and metronidazole was administered. However, fever recurred one day after discharge and the urinary symptoms persisted. Left hip pain with mild swelling ensued, for which he sought medical attention at this hospital.

He had received a diagnosis of mild mental retardation of undetermined causes since childhood. A diagnosis of severe pneumococcal pneumonia complicated with empyema was made, for which he had undergone decortication of the lung three years previously. He reported no urinary catheterization or sexual exposure. There was no family history of diabetes mellitus, malignancies or known immunodeficiency.

On admission, residual abscess of the prostate was found (Fig. 1B). Intermittent fevers persisted and the left hip pain worsened with limited range of motion and pain and stiffness of the left knee developed despite antibiotic treatment. The soft-tissue ultrasound revealed increased effusion of the left hip joint and normal structure of the left knee. Cultures of a synovial fluid specimen yielded negative results. A diagnosis of probable reactive arthritis was made according to preliminary classification criteria.[1]

In the context of unusual presentation of prostatic abscess and a preceding history of pneumococcal empyema in this adult, immunodeficiency was highly suspected. Investigations revealed undetectable serum immunoglobulin A, G, M (IgA, IgG, IgM) and absence of B-cells. He was discharged with a 2-month course of oral levofloxacin. A monthly infusion of intravenous immunoglobulin was administered, and he remained well during the follow-up as an outpatient for 6 months.

Abbreviations: IVIG, intravenous immunoglobulin; CT, computed tomography; IgA, immunoglobulin A; IgM, immunoglobulin M; IgG, immunoglobulin G; CVID, common variable immunodeficiency disease.

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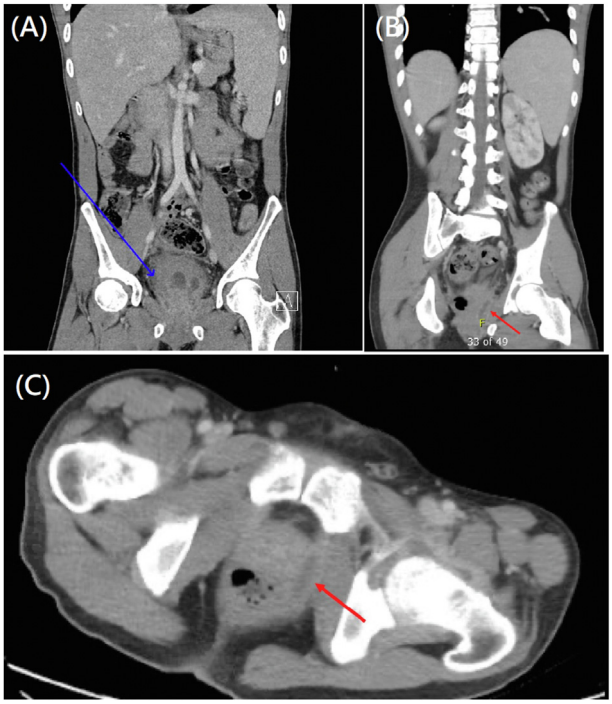

Figure 1. A: Computed tomography of the abdomen and pelvis revealing an abscess in the prostate at another hospital (arrow). B and C: Computed tomography of the residual abscess of the prostate on admission to this hospital (arrow).

Hypogammaglobulinemia can be primary or secondary, and the diagnosis of primary hypogammaglobulinemia can be made only after secondary causes of hypogammaglobulinemia such as drugs, multiple myeloma, lymphomas, and immunoglobulin loss[2] are excluded. Common variable immunodeficiency disease (CVID) and X-linked agammaglobulinemia caused by Bruton's tyrosine kinase gene mutation are the two most common causes of primary hypogammaglobulinemia. The clinical presentation of humoral immunodeficiency includes recurrent otitis media, sinusitis, and pneumonia.[3] While patients with humoral immunodeficiency are vulnerable to infections caused by encapsulated bacteria with polysaccharides, including *Streptococcus pneumoniae*, *Haemophilus influenzae*, *Neisseria meningitidis*, and *Klebsiella pneumoniae*, *F. mortiferum*, a common flora in oral cavity, gastrointestinal tract, and genitourinary tract,[4] has not been previously reported in such patients. Our case is the first case of primary hypogammaglobulinemia associated with *F. mortiferum* bacteremia.

Our case highlights that timely diagnosis of immunodeficiency relies on physicians' awareness of and alertness to the unusual presentations, pathogens and severity of any infectious disease.[5]

Declaration of competing interest

None to declare.

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Chien-Hung Lu
Department of Internal Medicine, National Taiwan University Hospital and National Taiwan University College of Medicine, Taipei, Taiwan
E-mail address: b97401059@ntu.edu.tw

Wang-Da Liu
Department of Internal Medicine, National Taiwan University Hospital and National Taiwan University College of Medicine, Taipei, Taiwan
Department of Medicine, National Taiwan University Cancer Center, Taipei, Taiwan
E-mail address: b95401043@ntu.edu.tw

Chun-Fu Huang
Department of Internal Medicine, National Taiwan University Hospital and National Taiwan University College of Medicine, Taipei, Taiwan
E-mail address: dkseneren@gmail.com

Chun-Hua Liao
Department of Pediatrics, National Taiwan University Hospital Biomedical Park Hospital, Hsinchu, Taiwan
E-mail address: shiva90345@gmail.com

Ting-Yuan Lan
Department of Internal Medicine, National Taiwan University Hospital and National Taiwan University College of Medicine, Taipei, Taiwan
E-mail address: d20023579@gmail.com

Chun-Min Kang
Department of Laboratory Medicine, National Taiwan University Hospital and National Taiwan University College of Medicine, Taipei, Taiwan
E-mail address: jimmyscorpio9@gmail.com

Chien-Chin Lin
Department of Internal Medicine, National Taiwan University Hospital and National Taiwan University College of Medicine, Taipei, Taiwan
Department of Laboratory Medicine, National Taiwan University Hospital and National Taiwan University College of Medicine, Taipei, Taiwan
E-mail address: jake9665@gmail.com

Yao-Hsu Yang
Department of Pediatrics, National Taiwan University Hospital, Taipei, Taiwan
Department of Pediatrics, National Taiwan University Hospital Hsin-Chu Branch, Hsin-Chu, Taiwan
E-mail address: yan0126@ms15.hinet.net

Chien-Ching Hung*
Department of Internal Medicine, National Taiwan University Hospital and National Taiwan University College of Medicine, Taipei, Taiwan
Department of Tropical Medicine and Parasitology, National Taiwan University College of Medicine, Taipei, Taiwan
Department of Medical Research, China Medical University Hospital, Taichung, Taiwan
China Medical University, Taichung, Taiwan

*Corresponding author. Department of Internal Medicine, National Taiwan University Hospital, 7 Chung-Shan South Rd., Taipei City, 10002, Taiwan.
E-mail address: hcc0401@ntu.edu.tw (C.-C. Hung)

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