Deep neck abscess is a rare complication of acute mastoiditis in the era of antibiotics. In preantibiotic era, Bezold’s abscess was the most common cause of the otogenic deep neck abscess. We reported a 19-year-old male with cholesteatoma complicated by acute supplicative mastoiditis, lateral sinus thrombosis, thrombophlebitis of the ipsilateral internal jugular vein and deep neck abscess formation. The pathway of the deep neck abscess formation is different from the classic presentations of Bezold’s abscess. Computed tomography, magnetic resonance imaging and ultrasonography were complementary for the detailed demonstration of the origin and the extent of the disease. Segmental mural lysis of the infected internal jugular vein was considered to be responsible for the development of deep neck abscess formation.

**Key words:** mastoiditis; Bezold’s abscess; neck, abscess; sinus, thrombosis

Otitis media is a common disease of childhood. However, it has been estimated that 1.5% of all adults suffered from active chronic otitis media [1]. Complications of otitis media can be categorized into two groups: intracranial and extracranial. Intracranial complications occur in 0.24% of the patients and include meningitis, encephalitis, brain abscess, epidural abscess and lateral sinus thrombosis. Extracranial complications occur in 0.45% of the patients and include facial nerve paralysis, labyrinthitis, perichondritis, coalescent mastoiditis and subperiosteal abscess [2]. Deep neck abscess is extremely rare in the era of antibiotics. Herein, we present a rare transcranial complication of cholesteatoma and acute mastoiditis with resultant deep neck abscess formation. Imaging studies with different modalities are valuable for tracing the pathway of disease extension.

**CASE REPORT**

A 19-year-old male came to our emergent unit with pale and ill looking. He suffered from progressive vertigo, vomiting, nystagmus, supernovened chillness and fluctuating fever (the spike up to 40.3°C) for five days. On physical examination, swelling, erythema, heatness and tenderness on his left retroauricular region were noticed. Otoscope displayed perforation of the left tympanic membrane with purulent discharge. Laboratory data showed elevated white blood cell count (23500/mm³) and C-reactive protein (180 mg/dl). Due to the past history of chronic otitis media, he was clinically impressed as acute mastoiditis complicated with Bezold’s abscess formation. High-resolution computed tomography (HRCT) of temporal bone showed soft tissue density filling the left tympanic cavity and cloudiness of the left mastoid air cells with bony erosion at the tympanic tegmen (Figure 1a). Magnetic resonance images (MRI) further displayed swelling of the left temporalis muscle, thrombosis of the sigmoid sinus, jugular bulb and upper internal jugular vein.

Reprint requests to: Dr. Yao-Liang Chen
Address: First Department of Diagnostic Radiology, Chang Gung Memorial Hospital,
No. 5 Fu Hsing Street, Kwei Shan, Taoyuan 333, Taiwan, R.O.C.
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(IJV) (Figure 1b, 2a). T1-based enhanced MR angiogram of the neck confirmed IJV thrombosis (Figure 2b). Enhanced CT of the neck disclosed deep abscess formation that was contiguous to the thrombotic left IJV and deep to the sternocleidomastoid muscle (SCM)(Figure 3a). High-resolution ultrasonography (HRUS) clearly depicted focal lysis at the wall of the infected left IJV (Figure 3b). The patient underwent left canal-down radical mastoidectomy, tympanoplasty, meatoplasty, incision and drainage of the left neck abscess. Left suppurative mastoiditis complicated by attic cholesteatoma was explored. However, there was no continuation between the mastoiditis and deep neck abscess either by imaging findings or by surgical exploration. Pus culture showed moderate growth of anaerobic bacteria including Peptostreptococcus anaerobius and Peptostreptococcus asaccharolyticus. Treated by thorough open drainage of abscess and intravenous antibiotic administration, the patient was discharged three weeks later with the sequel of left hearing loss (over 100 decibel).

DISCUSSION

Acute mastoiditis can be the complication of pre-existing secretory otitis media or other chronic disease of middle ear, including cholesteatoma. Before the use of antibiotics it was common and associated in some instances with serious complications. The mucosa of the tympanic cavity and its extension into the mastoid cells has an inherent ability to overcome acute infection. As a result, acute otitis media and mastoiditis may be self-limited. However, severe suppurative and necrotizing infections of the middle ear can cause systemic reaction [3].

The incidence of complications resulting from suppurative otitis media has significantly decreased in the era of antibiotics. In the beginning of the 20th century, 50% of all cases of otitis media developed coalescent mastoiditis. Recent studies suggested a current incidence of only 0.24% [2]. Complications of otitis media can be grouped into two broad categories: intracranial and extracranial. Intracranial complications include meningitis, encephalitis and lateral sinus thrombosis. Prior to the widespread use of antibiotics, 2.3% of patients with otitis media developed intracranial complications. Nowadays, the rate has fallen nearly 10-fold to 0.24%. The contemporary risk for developing extracranial complications of otitis media is approximately twice of that for intracranial complications, with 0.45% of patients experiencing problems such as facial nerve paralysis, labyrinthitis, perichondritis, coalescent mastoiditis, subperiosteal abscess or Bezold’s abscess [1].

Bezold’s abscess was considered clinically when mastoiditis coexisted with deep neck abscess. In 1908,
Bezold was the first to describe abscess in the neck arising from mastoiditis. Inflammation and infection may result in necrosis of the mastoid tip, allowing the pus to track from the medial side of the mastoid process through the incisura digastrica (digastric groove). The pus is prevented from reaching the surface by neck musculature, but can track along the fascial planes of the digastic muscle or SCM [1]. Bezold’s abscess usually spreads into the substance of the SCM and confines to the posterior cervical and perivertebral spaces by the pharyngobasilar fascia and the deep layer of deep cervical fascia [4,5]. It may extend into the carotid, prevertebral, danger, and retropharyngeal spaces. By gaining the access into the danger space, an abscess may extend into the mediastinum or into the base of the skull [4]. Due to the depth of their location, Bezold’s abscess may be difficult to be palpated. Pneumatization of the mastoid process leads to thinning of the bone and is considered an important factor in the development of Bezold’s abscess. Therefore, Bezold’s abscesses are more common in adults than in children because well pneumatization of mastoid tip often occurs in adults.

Mastoiditis contributes to most of the lateral (sigmoid) sinus thrombosis. In a series of 22 cases of lateral sinus thrombosis, 19 were associated with suppurative otitis media and mastoiditis [6]. Suppuration in the middle ear, mastoid, or both may spread to the adjacent intracranial structures through progressive thrombophlebitis, bony erosion, or direct extension, resulting in meningitis, extradural abscess, subdural empyema, focal encephalitis, brain abscess, lateral sinus thrombosis, and otic hydrocephalus. The clinical pictures of lateral sinus thrombosis are well documented: an ill patient with high swinging pyrexia (‘picket fence’ pattern), headache, neck pain and progressive anemia with, less commonly, occipital edema [6].

The occurrence of deep neck abscess and lateral sinus thrombosis may be coincidental, since both are complications of mastoiditis. There is also a possibility that lateral sinus thrombosis may be arisen by retrograde spread from the infected internal jugular vein which had already been involved by Bezold’s abscess. It is however possible that the neck abscesses may be formed by direct regional spread from the infected internal jugular vein that had autolysed [6], as in our case.

Radiological study is indicated in cases of acute mastoiditis or otitis media when there is clinical suggestion of coalescent mastoiditis, which signifies the

Figure 2. a. Coronal enhanced T1-weighted MRI depicts thrombosis in the left upper internal jugular vein (IJV). A low-signal gap and enhanced extramural tissue (double black arrows) imply thrombophlebitis and possibly segmental mural lysis of the left IJV. As compared with the signal void in right IJV(open large arrow), the thrombosis of left IJV shows slight low signal intensity (double white arrowheads) with thick mural enhancement. b. Venous phase of T1-based MR angiogram shows obvious enhanced flow signal in the right IJV but absence of flow signal in the left IJV, confirming left IJV thrombosis. (RIJV: right internal jugular vein, RCCA: right common carotid artery, RVA: right vertebral artery, LCCA: left common carotid artery, LVA: left vertebral artery)
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transition from mucoperiosteal disease to bone disease and even to intracranial complications. CT can show stages of disease progression when infection spreads by way of soft tissue and bone pathways into dural venous sinuses, meninges, labyrinth, facial nerve, epidural and other intracranial spaces [3]. CT is also valuable in early diagnosis of cholesteatoma in the mastoid cavity and exact delineation of abscess formation [5]. MRI is more useful than CT for evaluation of the complications of acute mastoiditis in some aspects. Septic thrombosis of the lateral sinus and jugular bulb is a highly lethal condition. Enhanced CT may reveal evidence of sinus thrombosis, manifested as a filling defect in the vessel (empty delta sign). However, false-positive diagnosis up to 30% of cases has been reported [7]. In contrast, MRI can distinguish between flowing blood and thrombus. Enhanced MRI with gadolinium-DTPA is a valuable adjunct to achieve the diagnosis and to delineate the extent of the pathology [8], whereas magnetic resonance angiography is the procedure of choice for the confirmation of venous thrombosis. CT and MRI can detect concomitant cerebral infection or infarction as well as infection in the epidural or subdural spaces [7].

For jugular vein thrombosis, HRUS can show an intraluminal mass of low or mild amplitude echoes, loss of respiratory rhythmicity and venous pulsation, absence of venous response to respiratory maneuvers (Valsalva and sniff test) and incompressibility of the thrombotic jugular vein [9]. Because HRUS can depict both transverse and longitudinal planes, it can provide an accurate delineation of luminal compromise by thrombus [10]. For the patient we reported, only HRUS could depict the exact location of segmental autolysis of the jugular wall. Based on the imaging findings of CT, MRI and HRUS, we believe that the deep neck abscess arose from the septic thrombophlebitis of the internal jugular vein rather than direct extension of mastoiditis from the mastoid tip, which is the typical pathway of Bezold’s abscess.

Early surgery is mandatory to treat severe mastoiditis such as coalescent mastoiditis and subperiosteal abscess. The infected neck or abscess formation needs thorough drainage. Given the low incidence and lack of consistent signs and symptoms, Spiegel et al suggested contemporary practitioner must be rely on radiological images to determine the presence and pathway of mastoid abscess [1].

REFERENCE


![Image](image_url)


源起於耳部的深頸部膿瘍-珍珠瘤及急性乳突炎引發的罕見併發症

陳耀亮 吳樹銘 黃浩輝 黃敏政 衛譽遊 萬永亮
林口長庚紀念醫院 放射診斷一科

抗生素發現及使用後，急性乳突炎罕見併發深頸部膿瘍；在此之前，急性乳突炎較常造成Bezold氏膿瘍。我們報導一位19歲男性病人罹患中耳珍珠瘤併發化膿性乳突炎，側竇栓塞，同側內頸靜脈炎及深頸部膿瘍。這種深頸部膿瘍的形成路徑迥異於Bezold氏膿瘍。電腦斷層，磁振造影及超音波對於發現這類感染起源及路徑各擅勝場亦可互補所短,綜合影像判斷可知局部受感染的內頸靜脈壁溶蝕是導致深頸部膿瘍的主因。

關鍵詞：乳突炎，Bezold氏膿瘍，頸部膿瘍，側竇栓塞