以譫妄為表現之空蝶鞍症併發泛腦垂腺低能症：二例報告

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空蝶鞍症患者一般腦垂腺機能正常而無症狀。在此報告一 48 歲男性，表現譫妄之症狀，生化及血液檢查發現低血鈉症及貧血，內分泌功能檢查顯示泛腦垂腺低能症，腦部核磁共振影像顯示為空蝶鞍症。病患之意識情況及精神症狀，在荷爾蒙補充及低血鈉矯正之後，即慢慢恢復正常。本文回顧過去文獻並分析此疾病病因及臨床表現，使醫師增加對此一疾病罕見表現之認識。

關鍵詞：泛腦垂腺低能症，譫妄症，空蝶鞍症
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前 言

席漢氏症(Sheehan's syndrome)[1,2,3]或女性泛腦垂腺低能症[4,5]併發急性精神病症或譫妄症文獻上有病例報告，但在男人泛腦垂腺低能症併發急性精神病之報告，較爲罕見[6]。空蝶鞍症患者一般腦垂腺機能正常而無症狀[7]，在此報告一罕見病例：一位男性病患有空蝶鞍症併泛腦垂腺低能症，且以急性發作，伴有精神病性行爲障礙之譫妄症狀爲其表現。

病例報告

一位 48 歲男性，因突然出現脫衣服、亂摔東西、扯電話線及推人打人等精神病性之怪異行爲，而被送到本院急診室。

病患在 24 年前，曾發生 3 次車禍，有腿骨折的情況，但當時並未有意識喪失。復原之後，並沒有明顯的後遺症。近十年來，病患自覺臉色蒼白、怕冷及鬍鬚、腋毛減少的現象。在體檢時，也曾被告知血壓偏低。到近幾年來，腋毛及陰毛則完全消失。性慾方面則未有明顯異常。在服用中藥之後，症狀並無改善，病患也不以爲意。

病患在本次就醫前約兩週，出現咳嗽、喉嚨痛及嘔吐的症狀。服用中藥之後，不適逐漸改善。但當天下午在辦公室時，突然出現上述精神病性之怪異行爲而被送至急診室。

理學檢查方面，生命徵象穩定，外表蒼白，眉毛細而稀疏。胸腹正常，但腋毛及陰毛均無，睪丸大小正常但較軟。在急診室之精神狀態：不語、不易會談，有躁動，定向力障礙，注意力不集中，記憶障礙，態度不合作，無法配合檢查。住院後一星期之間精神狀態逐漸改善，但態度仍不合作，無法配合醫療，且言行顯幼稚。住院後第 9 天精神檢查發現意識清楚，偶而語言脫軌，非邏輯性言語，關係意念，誇大意念，多疑，對日期定向有誤，仍偶有躁動行爲。住院後第 13 天，其精神狀態仍偶而有躁動、易怒，偶而出現誇大意念等症狀，且發現仍有防衛性的態度，幼稚的行爲，不切實際的想法。其言語較片斷，態度上較不理他人，白天意識清楚，但晚上對地點定向力障礙，以爲住在自己家裡。依病程所見精神病理表現，其精神科之診斷爲譫妄症。其譫妄症伴有精神病性症狀與行爲障礙。實驗室檢查顯示：WBC 13,170/μl, Hb 9.7 g/dl, MCV 78.9 fl, platelet 215x10^3/μl, BUN 8 mg/dl, creatinine 0.8

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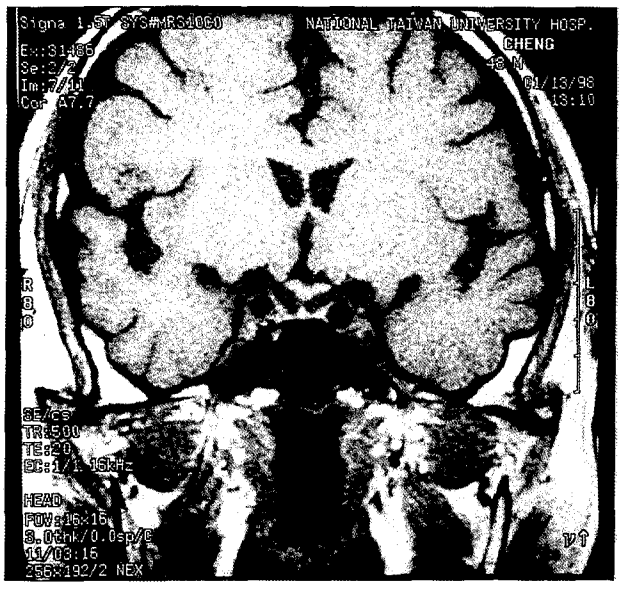
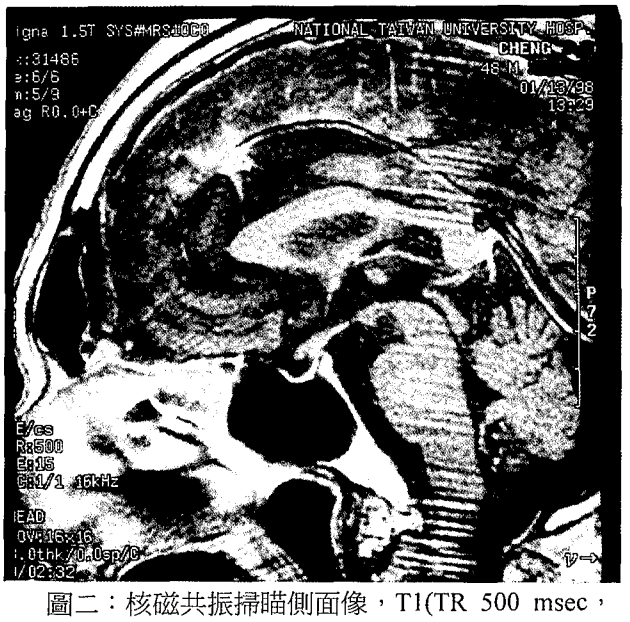


圖一：核磁共振掃瞄正面像，T1(TR 500 msec，TE 20 msec)，顯示空蝶鞍症。



圖二：核磁共振掃瞄側面像，T1(TR 500 msec，TE 15 msec)，Gd-DTPA 顯影後，顯示空蝶鞍症。

mg/dl, Na 122 mM, K 3.7 mM, Ca 2.18 mM, glucose 110 mg/dl。主要異常爲低血鈉及貧血。內分泌功能檢查：T3 67.5 ng/dl，T4 8.69 μg/dl，hsTSH (high-sensitivity thyroid-stimulating hormone) 0.383 μIU/ml，ACTH (adrenocorticotropic hormone) 7.2 (8am) pg/ml，cortisol 3.13 (8am) μg/dl，hGH (human growth hormone) 0.44 μg/l，testosterone 0.12 ng/ml，estradiol <20 pg/ml。在靜脈注射 100μg LHRH (luteinizing hormone releasing hormone)，血清之 FSH(follicle-stimulating hormone)及 LH (luteinizing hormone)在 0, 15, 30, 60, 120 分鐘分別爲 0.3, 0.3, 0.6, 0.9, 1.2 及<0.7, 1.8, 3.8, 3.6, 2.7 mIU/ml。在靜脈注射 400μg TRH(thyrotropin-releasing hormone)，血清之 prolactin 及 hsTSH 在 0, 15, 30, 60, 120 分鐘分別爲 4.7, 7.0, 7.4, 5.7, 5.2 ng/ml 及 0.316, 1.320, 2.920, 3.200, 2.100 μIU/ml。在靜脈注射胰島素 0.5U/kg，血糖值在 0, 15, 30, 60 分鐘爲 81, 64, 49, 83 mg/dl，hGH 在 0, 15, 30, 60, 120 分鐘爲 0.25, 0.16, 0.12, 0.16, 0.35 μg/l；ACTH 爲 3.5, 10.9, 12.1, 10.1, 2.1 pg/ml；cortisol 爲 2.82, 1.82, 2.38, 1.93, 3.88 μg/dl；prolactin 爲 5.1, 4.9, 4.8, 4.8, 5.4 ng/ml。以上結果顯示其腦垂腺、甲狀腺、腎上腺及性腺功能均低下。腦波顯示中度瀰漫性皮質功能異常，尤其在雙側前葉部位。在診斷泛腦垂腺低能症(panhypopituitarism)之後，即給予腎上腺皮質素 hydrocortisone 100mg iv q8h、甲狀腺素 Levothyroxine 100 μg po qd 及 3%食鹽水靜注。腦部核磁共振影像顯示空蝶鞍症(empty sella syndrome)(圖一、圖二)。因此，確定病患即爲空蝶鞍症併泛腦垂腺低能症以併有精神病性症狀之譫妄症爲表現。病患之意識情況及種種精神症狀，在荷爾蒙補充及低血鈉矯正之後，即慢慢恢復正常。腦波追蹤也有逐漸改善。

討 論

空蝶鞍症的原因很多，包括腫瘤、炎症、血管病變、外傷性等[7]。本病例在 24 年前有外傷之病史，約十年之後發生腦垂腺低能之症狀，在時序上有此可能性。依回顧文獻，頭部外傷引起之腦垂腺低能症，多發生於傷後一年內(71%)，但也有延至 20 年後才表現者。其外傷的程度，55%有顱骨骨折，93.4%有昏迷；但也有無任何意識異常者[8]。本病例若是因外傷引起腦垂腺低能症，其病程是符合的。

空蝶鞍症通常不會有腦垂腺低能症表現[9]，這也是本病例不尋常處。

泛腦垂腺低能症的另一鑑別診斷爲淋巴球性腦垂腺炎(lymphocytic hypophysitis)。此病在1962 年首先被提出[10]，主要發生在懷孕或產後，很少在停經後或男性。其機轉爲自體免疫性的腦垂腺破壞，在核磁共振影像可見腦垂腺腫塊病變，切片可見淋巴球浸潤。曾有病例報告顯示病患在五年後變爲空蝶鞍症[11]。至 1997 年爲止，文獻上只有七例男性淋巴球性腦垂腺炎[12]，之後並無個案報告。本病例是否爲此病症，因無法測定抗腦垂腺抗體，故無法定論。

在泛腦垂腺低能症病患發生低血鈉的原因有三[13,14]：(1)醛固酮(aldosterone)分泌降低造成尿鈉排泄增加；(2)甲狀腺素分泌減低，引起利尿作用異常；(3)因糖皮質素(glucocorticoid)及鹽皮質素(mineralocorticoid) 減少引起之腎小管通透性及功能異常。在貧血方面，Ishikawa 等報告一病例[15]顯示一位席漢氏症及空蝶鞍症病患有嚴重貧血(Hb 7.2 g/dl)，其血清之紅血球生成素(EPO)較低。經甲狀腺素及腎上腺皮質素補充之後，其紅血球生成素及血紅素均有上升。所以貧血可由激素之缺乏解釋，但是其詳細機轉仍不明瞭。

依文獻回顧，腦垂腺低能症造成之精神異常包括精神病性疾病[16,17]及譫妄[14,18]等，而又以後者爲多。要診斷由腦垂腺低能症引起之精神異常，必須要有前後時序的關聯。但腦垂腺低能症的表現常是輕微且不明顯，所以在臨床上往往較爲困難。就如同女性的席漢氏症，其腦垂腺低能症的出現可能在產後出血的數十年。

精神病性疾病或譫妄之發生與腦垂腺低能症的關係並不十分清楚。精神症狀可能由激素缺乏本身引起，或次發於激素引起的電解質或血糖異常。例如在艾迪森氏(Addison's)病及甲狀腺低能症皆可發生精神症狀[19]。腦垂腺低能症包含上述疾病之激素缺乏，故也可能產生精神症狀。此外，若腦垂腺低能症是因腦垂腺腫瘤引起者，也可能因腫瘤向上壓迫至額葉、顳葉或第三腦室，而產生精神症狀[16]。

腦垂腺低能症是引發精神病症的原因之一，但卻常因其相關症狀不具特異性而被忽略。因此在一新發生精神症狀之病患，以本案爲例，發生併有明顯精神病性症狀與行爲異常之譫妄症，而過去無明顯精神疾病史及沒有家族史者，必須仔細尋找其器質性的原因，並且回顧其病史及實驗室檢查的結果，如有低血鈉及貧血者，則可能有多種激素的異常，必須做進一步的激素測驗。

在治療方面，雖然用抗精神病劑藥可控制其精神病性症狀，但要完全消除症狀，則要給予激素之補充。若不治療內分泌問題而只給予抗精神病劑，如：haloperidol，不但症狀無法完全緩解，更有可能加重其內分泌異常，使症狀反而更加惡化。本病例之症狀也如預期在給予腎上腺皮質素及甲狀腺素之後即漸漸恢復正常，而不須服用任何抗精神藥物。

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Empty Sella Syndrome with Panhypopituitarism Presented as Delirium：Report of Two Cases

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Abstract: Patients with empty sella syndrome usually have normal pituitary functions and show no symptom. We report a man with panhypopituitarism who presented as acute psychotic symptoms. This 48-year-old man had three traffic accidents without loss of consciousness 24 years ago. After recovery, he led an uneventful life. He noted pale appearance, cold intolerance and decreased beard and eyebrow gradually in these ten years. Hypotension was found in health exams. Absence of axillary and pubic hair was also noticed. There was no impairment of libido. Two weeks prior to this admission, he suffered from productive cough, sore throat and vomiting, which was treated by herb drugs. Acute-onset conscious change with queer behavior developed when he was working in the office. He was then taken to the emergency department.

On arrival, he was irritable and disoriented. The BP was 114/64 mmHg, temperature 36.5°C and pulse rate 120/min. Physically, he looked pale. Eyebrow was thin and scanty. The chest, heart and abdomen were normal except diminished pigmentation of areola. There was no axillary hair and pubic hair. The testes were soft. Delirium with acute psychotic state was suspected. Hyponatremia (Na 122 mM) and anemia (Hb 9.7 mg/dl) were disclosed. Hypertonic saline was administered. Brain MRI revealed empty sella. His basal hormone levels were: T3 67.5 ng/dl, T4 8.69 μg/dl, hsTSH 0.383 μIU/ml, ACTH 17.6 (8am), 10.8 (4pm) pg/ml, cortisol 1.73 (8am), 6.50 (4pm) μg/dl, hGH 0.44 μg/l, testosterone 0.12 ng/ml, estradiol <20 pg/ml. After IV 100 μg LHRH, the serum FSH and LH levels at 0, 15, 30, 60 and 120 minutes were 0.3, 0.3, 0.6, 0.9, 1.2 and <0.7, 1.8, 3.8, 3.6, 2.7 mIU/ml, respectively. The serum prolactin and hsTSH levels in response to IV 400 μg TRH at 0, 15, 30, 60 and 120 minutes were 4.7, 7.0, 7.4, 5.7, 5.2 ng/ml and 0.316, 1.320, 2.920, 3.200, 2.100 μIU/ml, respectively. Insulin hypoglycemic test revealed the hGH response at 0, 15, 30, 60 and 120 minutes were 0.25, 0.16, 0.12, 0.16, 0.35 μg/l; ACTH: 3.5, 10.9, 12.1, 10.1, 2.1 pg/ml; cortisol: 2.82, 1.82, 2.38, 1.93, 3.88 μg/dl; prolactin: 5.1, 4.9, 4.8, 4.8, 5.4 ng/ml. Under the impression of empty sella with panhypopituitarism, hormone supplement with IV hydrocortisone 100mg q8h and oral Levothyroxine 100μg qd was administered. His consciousness improved gradually and he was discharged smoothly with oral prednisolone and Levothyroxine. Literatures were reviewed for this rare condition.

Key Words: panhypopituitarism, delirium, empty sella syndrome

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