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ORIGINAL ARTICLES

CASE REPORT

創刊號發刊詞

童綜合醫學雜誌創刊了！

出刊前夕懷著無比興奮的心情，看著這本刊物能呈現在各位眼前。多年來童醫院以戰戰兢兢的心情持續推動醫學研究，提高醫學水準，激勵本院同仁投入研究，從基本病例分析到基礎研究，包含醫、牙、藥、護、行政管理等各個部門，各個領域都能深入並將研究心得撰寫成論文，我們相信只有不斷的努力，雜誌的水準才能提升！

當然童綜合醫學雜誌是對外開放的，誠摯的希望各個領域的專家在這學術的環境中互相切磋，我們有信心辦好這本刊物！雖然校對再三，錯誤難免，希望各界先進不吝指教。

院長 童瑞華

The Prevalence of Gastritis and Delayed Gastric Emptying in Asymptomatic Volunteers

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Background: chronic gastric mucosal inflammation is closely associated with postprandial antral hypomotility. Antral hypomotility producing gastric stasis and pyloric sphincter abnormalities resulting in duodenogastric reflux of biliary-pancreatic secretions. The interactions among mucosal inflammation, alter motor activity and changes in gastric luminal milieu in asymptomatic gastric disease have not been investigated. We investigated 1) the prevalence of chronic antral gastritis in asymptomatic volunteers; 2) the correlation between the severity of antral mucosa damage and the gastric emptying of solids; and 3) ascertained the effects of a prokinetic drug (metoclopramide) on gastric motility.

Methods: In 64 volunteers biopsy specimens were taken from the antrum. The endoscopic biopsy specimens[3-7] were taken from the proximal and distal margins of the antrum. The gastric emptying of solids was analyzed by dual-phase mode consisting of a lag phase and an emptying phase.

Results: Endoscopically evident gastritis was present in 65.6%(42/64), histological gastritis in 78%(46/59). There were significant differences in gastric emptying between normals and those with histological evidence of gastritis (63±9 min vs. 88±6.5 min, $P<0.01$) but not endoscopic gastritis. Histological gastritis correlated the best with delay in gastric emptying 93%(14/15). The lag duration was shortened by metoclopramide in the chronic superficial gastritis group. The emptying rate could be normalized by metoclopramide in the chronic gastritis group(superficial & atrophic).

Conclusions: Gastric emptying correlated strongly with the presence or absence of histological gastritis. The degree of delayed gastric emptying correlated with the degree of gastritis. Metoclopramide may improve the gastric emptying phase and decrease the lag duration in superficial chronic gastritis patients but not in normal subjects.

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Key words: Gastritis, Gastric emptying, metoclopramide

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INTRODUCTION

In surveys, asymptomatic gastritis is present in from 10% of the American population[1] and up to 53% of the Scandinavian population[2]. The incidence among ethnic Chinese is not known. Clinical observations by experienced endoscopists in Taiwan would suggest that it is high. Chronic gastritis is associated with delayed gastric emptying and with carcinoma of the stomach[3,4,5]. In the course of other studies, we found it important to ascertain the incidence of asymptomatic or subclinical gastritis among Chinese in Taiwan.

Helicobacter pylori is the main cause of gastritis and peptic ulcer in adults and children. The infection is commonly acquired in childhood. Although H. pylori infection is usually asymptomatic, the infection is not benign. In asymptomatic gastritis, the gastric mucosal changes is closely associated with the infection. Conversely, these infection is also though to be linked with impairment of gastric function. Persistent gastric function abnormality in asymptomatic condition not only affect the food digestion but also caused the gastric mucosa damage by food stasis and enhancing the infection. Early restoration of gastric function may also attenuate the infection situation.

Although there are some studies on the factors influencing gastric emptying in adult many questions about this function have not been complete resolved. Determining the rate of Gastric emptying in patient with upper gastrointestinal symptoms and no mucosa disease or anatomic abnormalities is therefore essential. Gastric emptying can he evaluated by scintigraphic examination of the clearance of radionuclide food markers and is considered to the standard procedure for investigating gastric emptying.

There is no relevant data on the relations between the depth of gastric mucosal damage and changes of gastric emptying. We conducted a study of the gastric emptying pattern with various type of gastritis in asymptomatic subjects.

The purpose of the present study was to 1) investigate the incidence of chronic antral gastritis in asymptomatic volunteers; 2) note any correlation between the severity of antral mucosal damage and the gastric emptying of solids; and 3) ascertain the effects of a prokinetic drug (metoclopramide) on gastritis motility among normal subjects, those with chronic superficial gastritis group, and those with chronic atrophic gastritis.

METHODS

We studied 64 consecutive military personnel who had been in hospital for minor conditions (e.g.. cellulitis of the leg), or for minor operations not requiring general anesthesia, perioperative antibiotics or any analgesic or narcotic. The most extensive procedure was repair of an inguinal hernia under spinal anesthesia. The procedures are listed in Table I. At the time of discharge, these patients were asked to participate in the study which had been approved by the Human Subjects Committee of the hospital. Subjects were eager to participate as they were relieved of military duty for a longer period.

Approximately one month following discharge the patients underwent the following studies: a) careful history for eructation, bloating, increased flatulence, diarrhea, nausea, vomiting, peptic ulcer disease, smoking, drinking, and aspirin or other drug use; b) gastroscopic examination and antral biopsy and c) gastric emptying studies of a solid meal.

Viscid xylocaine (10 ml) was given by mouth and 10 mg of buscopan intramuscularly immediately before gastroscopy. The stomach and duodenum were carefully inspected and categories of mucosal changes were noted on a standard from. Biopsy specimens[3-7] were taken distal to the incisura from the proximal and distal margins of the antrum. They were fixed in formalin and stained with haematoxylin and eosin. The histological sections of the antral biopsies were independently reviewed and graded by two pathologists blinded to both the endoscopic findings and the gastric emptying studies. The gastritis specimens were classified histologically as chronic superficial gastritis or chronic atrophic gastritis using the criteria of Michael F, Dixon et al[6]. When only intestinal metaplasia was seen (twice), the volunteers were dismissed from the study.

Several[3-7] days later gastric emptying studies were done on 2 consecutive days. In random order, volunteers were given either 50 gm of congee (rice gruel) mixed with 100uci TC99m-DTPA microcapsules with particle size of 1-1.5 mm, or 10 mg of metoclopramide, intramuscularly, followed by the radiolabelled congee meal[7,8]. Gastric emptying data were analyzed by dual-phase mode consisting of a lag phase and an emptying phase[9,10]. The duration of the

lag phase was determined as the interval between ingestion of the meal and first appearance of the detectable amounts of TC99m-DTPA in the proximal small intestine. The post lag solid emptying data were linearized by logistic transformation, and the slope was used as an index of the speed of the gastric emptying in the post lag period[9,10]. From the plotted curves of the observed proportionate emptying data, the half-time (t 1/2) for solid emptying was also calculated.

Data are given as mean value ± SE. Results were analyzed with student's t-test for paired and unpaired data. "Significant" indicates a calculated P value of less than 0.05.

Table 1. The habits,endoscopic and histological finding in asymptomatic volunteers

Subjects	64
Appendectomy	22
Hernioplasty	21
Others(cellulites,lacerations,sprains,etc.)	21
Ages	18-25
Sex	Male
Smoking(0.5 pack a day)	42/64
Drinking(240 ml Beer/day)	24/64
Medications	0/64
Gastritis	
Endoscopically	42/64
Histologically	46/59
A.Atrophic	20/59
B.Non-atrophic	26/59
C.Normal	13/59

Table 2. Smoking among histological normal and gastritis subjects

	Non-Smoker	Smoker	Total
Normal	2	11	13
Gastritis	18	23	41

RESULTS

Endoscopically evident gastritis was present in 65.6% (42/64) of the subjects. Histologically proven gastritis was found in 78% (46/59) (Table 1).

There was a higher incidence of smoking in histologically normal volunteers than volunteers with gastritis (11/13 vs. 23/41) (Table 2).

Gastric emptying was significantly delayed between the normals and volunteers with histological evidence of gastritis, 63.9 min vs. 88.7 min, P<0.01 (Fig. 1). There were no significant differences in gastric emptying between individuals with or without endoscopic evidence of gastritis (80.5 min vs. 80.6 min).

The gastric emptying parameters for individuals with normal antral mucosa, non-atrophic gastritis and

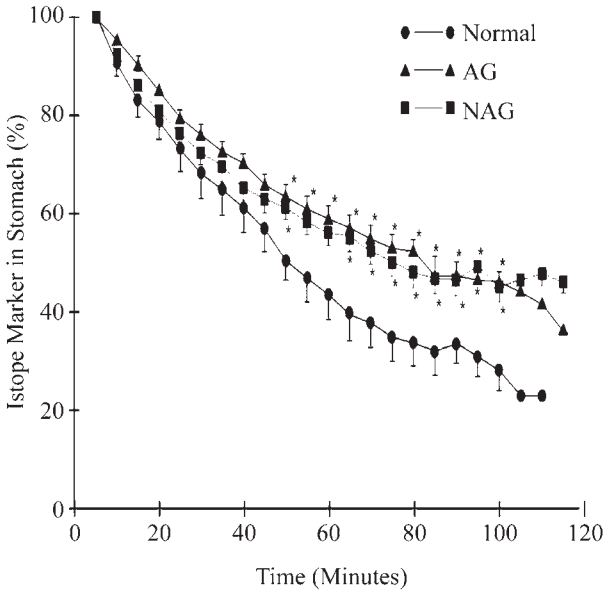

Fig. 1

Table 3. Gastric emptying among histological normal, non-atrophic gastritis (NAG) and Atrophic gastritis gastritis (AG) subjects.

	Normal n = 6		NAG n = 12		AG n = 13	
		+Metoclop ramide		+Metoclop ramide		+Metoclop ramide
Emptying rate (T 1/2) min	59 ± 10	58 ± 15	98 ± 13[a]	58 ± 6[b]	96 ± 10[a]	61 ± 8[b]
Lag Duration min	19 ± 4	17 ± 5	24 ± 3	17 ± 3[b]	26 ± 5	20 ± 3

Values are means ± SE
a: P<0.05 Normal vs. NAG, AG unpaired t-test
b: P<0.05 with vs. without metoclopramide paired t-test

atrophic gastritis are given in Table 3. The differences in the gastric emptying rates were independent of the lag duration times which were statistically similar in all three groupings. In the chronic superficial gastritis group the lag duration was shortened by metoclopramide. The emptying rate in those with chronic gastritis (superficial & atrophic) was normalized by metoclopramide. Emptying rates in normal volunteers were unaffected by metoclopramide.

Significantly delayed gastric emptying was defined as exceeding the rate found in histologically normal volunteers (59 ± 10 min). Histological gastritis correlated the best with Significantly delayed gastric emptying, 93% (14/15)(Table 4). Metoclopramide improved the delayed gastric emptying in these patients (Fig.2).

Table 4. The correlation between gastric emptying and histological gastritis

Emptying rate \ Subject	Histological Normal	Histological Gastritis	Total No.
Normal	5	19	24
Faster	1	0	1
Delayed	1	14	15

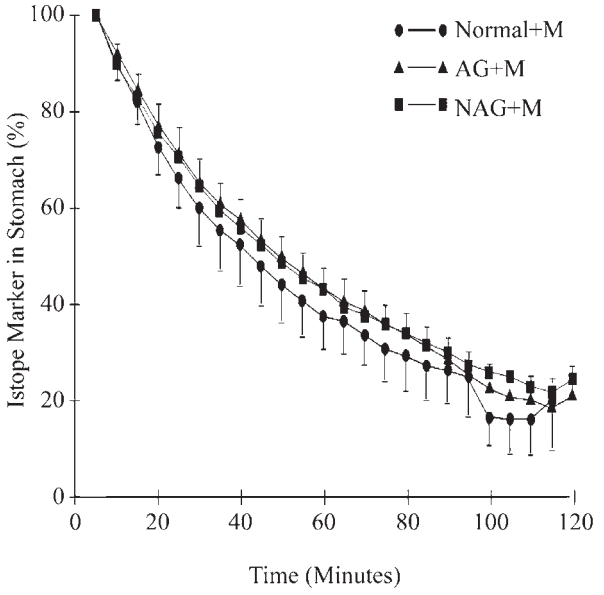

Fig. 2

DISCUSSION

The finding that 78% of otherwise healthy, young, asymptomatic military personnel in Taiwan had histological evidence of antral gastritis is both surprising and disturbing. While no previous information about the incidence of gastritis in the population of Taiwan has been reported, the experience of community-based gastroenterologists is that it is high among both the young and the old. Thus, the data here support that impression.

The reason(s) for the increased prevalence of gastritis in these healthy volunteers is unknown. The potential exogenous factors which may be related to the development of gastritis, putative factors such as cigarette smoking[11], alcohol ingestion[12], and excessive use of gastric irritating condiments do not account for the high incidence since overall they were used in only half of the subjects and were present in those both with and without histological gastritis (Table 1). Surprisingly, the incidence of smoking in normal (11/13) was higher than in those with gastritis (23/41)(Table 2). These results suggest that there is a natural protective mechanism in those normal individuals who smoke. Alternatively the prediction for smoking may be influenced by the presence or absence of gastritis.

Chang et al.[13] has been suggested that the incidence of H. pylori infection in the non-ulcer dyspepsia patients was 58.6% which is similar to the asymptomatic volunteers in Taiwan. However the incidence of H. pylori infection did not relate to delayed gastric emptying[13]. While not investigated in this study the most likely cause for the high incidence of asymptomatic gastritis among these volunteers is infection by H. pylori as the organism induces chronic gastritis in virtually all infected subjects[14]. In developing Countries such as Taiwan, gastritis attributable to H. pylori is acquired early in life (less than 20 years) in more than half of the individuals[15].

Studies among the Chinese population in Singapore have noted a 70% incidence of asymptomatic histologically prove gastritis among both the old and the young. There were also differences in the prevalence of gastritis in the other main racial groups in Singapore, being 22% in Malay patients and 57% in Indians[16,17]. The Australian Aborigine and white Australian also have different susceptibility to H. pylori infection (14.6% vs.0.7%)[18]. Thus a racial susceptibility to gastritis and to H. pylori infection may exist.

The correlation between the gastroscopic and microscopic diagnosis of gastritis is poor[19,20,21]. Although the dyspeptic symptoms did not related to the presence or absence of gastritis[22] our findings suggest that gastric emptying is strongly correlated with the presence of histological gastritis but not endoscopic gastritis.

Patients with normal antral biopsies had prompt and normal gastric emptying times. Metoclopramide did not affect their gastric emptying rates. In contrast all volunteers with histological evidence of gastritis had delayed gastric emptying (Fig. 1, Table 3). Their different severity of gastritis and degree of delay in gastric emptying may not been discriminated but metoclopramide was effective in improving gastric emptying, not only of the emptying phase (gastric motor component) but also by decreasing the lag duration time in chronic superficial gastritis patients. But chronic in atrophic gastritis patients is only improved the emptying phase. There results imply that chronic gastritis does not affect gastric intramural neural conduction but chronic superficial gastritis does.

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膀胱輸尿管尿液逆流治療之新方法：逆福仕內視鏡注射之評估

殷約翰

膀胱輸尿管尿液逆流是很常見的兒童泌尿系統異常，傳統的治療方法包括長期口服抗生素及手術治療。近年來內視鏡逆福仕（Deflux）® 治療，提供了另一項治療選擇。本院自 2004 年 1 月至 2006 年 12 月，使用內視鏡逆福仕注射治療了 16 位病人，注射一次後就痊癒的機率有百分之五十，注射二次後所有的病人都可痊癒。內視鏡逆福仕注射治療是治療膀胱輸尿管尿液逆流的有效方法之一。

（童綜合醫誌 2007; 1: 6-10）

關鍵詞：膀胱排尿攝影 Voiding Cystourethrography (VCUG)、內視鏡注射治療 Endoscopic injection、逆福仕 Deflux®、玻尿酸 Hyaluronan

膀胱輸尿管尿液逆流是孩童很常見的泌尿系統異常，它會造成腎臟功能的破壞、腎盂腎炎、高血壓、及尿路感染，年齡愈小的兒童若有尿路感染，他同時有膀胱輸尿管尿液逆流的機會越大，甚至可高到 30~50%。[1] 就全體兒童來，它發生的機率約爲 1%。因此對於所有有尿路感染的兒童，均有必要做膀胱排尿攝影（VCUG, voiding cystourethrography）檢查以確定是否有膀胱輸尿管尿液逆流。

VCUG 是診斷膀胱輸尿管逆流最常用的方法。當診斷出膀胱輸尿管尿液逆流以後必須給予適當的治療，以免產生腎臟的破壞。過去傳統的治療方法可以分成三大類：

㈠長期口服藥物預防尿路感染：等孩子逐漸長大後，膀胱輸尿管尿液逆流就會改善。對於輕度，第一、二、三型（Gr. Ⅰ、II、Ⅲ）的膀胱輸尿管尿液逆流，此方法確實有用，但是必須吃藥數年。

㈡手術治療（reimplantation）：對於重度的膀胱輸尿管尿液逆流第四、五型（Gr. Ⅳ、Ⅴ），口服藥物效果較差，手術治療，則可立刻解決逆流的問題。近年也有人用內視鏡微創手術取代傳統開刀手術。

㈢內視鏡注射填充物至輸尿管口（endoscopic injection of bulking agent）：雖然早在二十年前就有人用此一方法治療膀胱輸尿管尿液逆流，但由於所用的注射填充物，不是效果不長，就是填充物本身會移位或是無法吸收。因此一直沒有廣泛地被使用。直到 1995 年瑞典醫師提出一種新的填充物逆福仕（Deflux®）它是一種有效又穩定的物質。逐步被許多醫師採用，到了 2001 年 9 月也通過了美國食品及藥物管理局（FDA）的審核。得以在美國全面被採用。本院自 2004 年開始使用逆福仕（Deflux®）治療兒童的膀胱輸尿管尿液逆流。

病人與方法

從 2004 年 1 月至 2006 年 12 月本院共有 16 位病人實行逆福仕注射治療膀胱輸尿管尿液逆流，其中有 7 男 9 女，年齡由 1 歲 4 個月至 19 歲，平均年齡 4 歲。16 位病人中 10 位是雙側性膀胱輸尿管尿液逆流，所以共有 26 條輸尿管接受逆福仕注射。依逆流的程度來分 Grade Ⅰ 4 條、Grade II 4 條、Grade Ⅲ 12 條、Grade Ⅳ 6 條輸尿管尿液逆流。所有 Grade Ⅰ的病人皆爲雙側輸尿管尿液逆流，而對側爲 Grade II 以上的逆流。沒有因爲單側 Grade Ⅰ的膀胱輸尿管尿液逆流的病人接受逆福仕注射治療。16 位病人中有 7 位接受二次逆福仕注射（表一）。我們治療病人的適應症是膀胱輸尿管尿液逆流但沒有其他膀胱異常如憩室，神經性膀胱，或是輸尿管開口異常，（ectopic ureter），或是雙套輸尿管（duplux），所有病人

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之尿路感染在注射逆福仕前，均先加以藥物控制至小便完全無菌。

術前的準備和一般手術相同，包括血液，尿液常規、生化肝腎功能、電解質、胸部 X 光、心電圖等，術後隔天即可出院。

手術方法：一般我們將病人放在 lithotomy 的姿勢，較小的小孩用 Frog leg position。在全身麻醉下，用 9.5 Fr. 的 cystourethroscope，3.7 Fr 的針，將逆福仕（Deflux®），打入輸尿管膀胱開口之後壁的黏膜下層，注射的量太約是 0.4~1.0 ml，直至輸尿管口完全被注射入的逆福仕隆起關閉（圖一、二），呈現山丘的形狀。平均注射約 0.6 ml，注射時若看不見輸尿管後壁隆起，則需調整入斜的深淺，直到看見隆起。注射完畢後，針頭會留在黏膜下層約半分鐘後再拔出，並放置一根 6~8Fr Foley catheter 導尿管。第二天早上拔除導尿管後病人即可出院。出院後病人仍然會服用磺胺藥 1~2 週，等到小便檢查沒有白血球，即可停藥。到了 1-3 個月，12 個月我們會再做膀胱攝影 voiding cystourethrography，看看逆流是否痊癒，若是三個月的膀胱排尿攝影仍然有逆流我們會安排第二次的注射，在術後的追蹤期，並不需要用磺胺藥控制感染。但病人若有發燒或腸胃道異常，仍需做尿液檢查，

表 1 Reflux ureter 經 Deflux 注射後之效果

	Reflux ureter	1st injection	2nd injection
Grade Ⅰ	4	4	0
Grade Ⅱ	4	2	2
Grade Ⅲ	12	8	4
Grade Ⅳ	6*	4	1

*有一例Grade Ⅳ的病人在注射乙次後不願再次注射，而改成傳統手術治療。

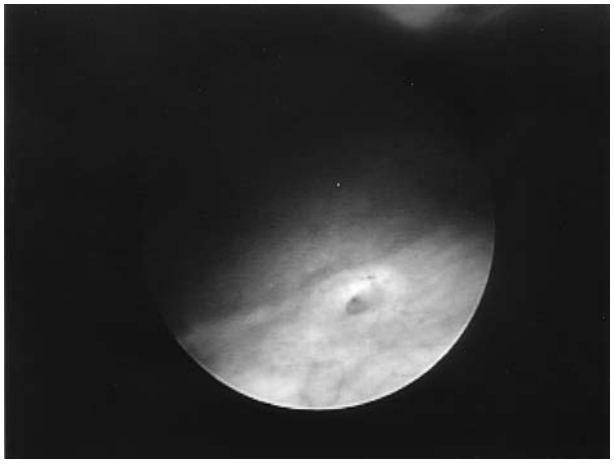

圖 1 逆福仕注射前之輸尿管

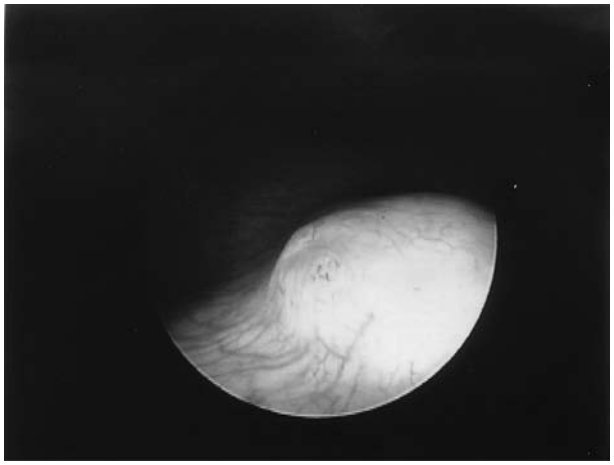

圖 2 逆福仕注射後之輸尿管口。

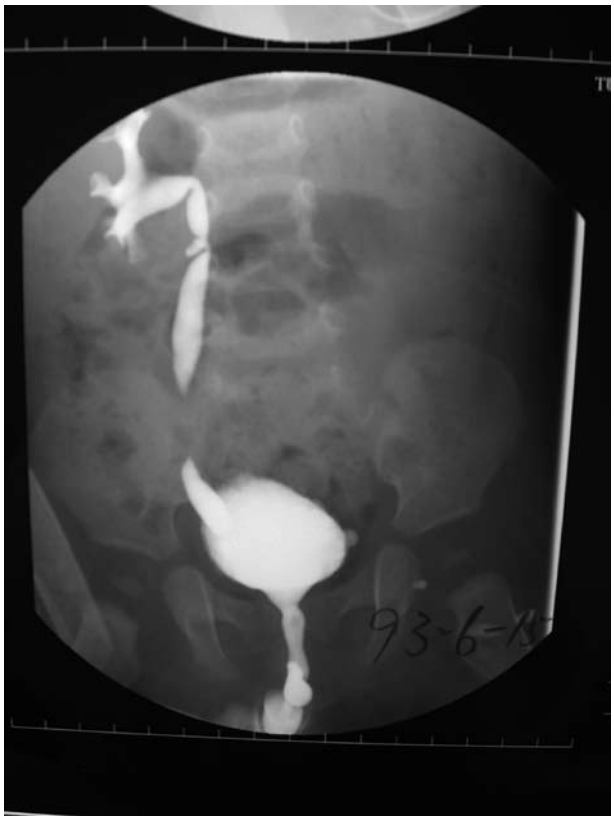

圖 3 右側膀胱輸尿管尿液逆流注射前。

看看是否有尿液感染。在 19 位病人中我們至少追蹤了 3 個月，最長 17 個月，平均 6 個月。

結　果

在 16 位病人中，第一次注射逆福仕後即痊癒的機會是百分之五十，其他的病人都需要第二次的逆福仕注射，在 16 位病人中有一位注射一次後，沒好，不願接受第二次的注射而改成傳統開刀治療，其他 7 位病人接受第二次注射後，所有病人在追蹤的 VCUG 檢查中，逆流均已消失。而逆流程度的輕重和痊癒的機會沒有絕對的關係，有 Grade II 的病人需要注射二次才會好，也有 Grade IV（圖三、四）的病人注射乙次逆流就完全消失。所有的病人也沒有因逆福仕的注射造成輸尿管阻塞，也沒有膀胱或尿道的併發症。

討　論

輸尿管穿過膀胱壁時分成二部份，一部份是穿過膀胱肌肉層的 intramural ureter, 穿過肌肉層後就在膀胱黏膜下層穿過 submucosal tunnel 最後才開口在膀胱三角的二端。黏膜下層輸尿管的長度及輸尿管背後的肌肉層堅實度決定是否產生逆流。治療膀胱輸尿管尿液逆流最重要的是使輸尿管穿過膀胱壁以後在黏膜下層的長度加長，也就是增加黏膜下層輸尿管的長度，或是增加輸尿管黏膜下層這一段背後的堅實度。傳統的開刀手術就是重建輸尿管進入膀胱後黏膜下層的長度及輸尿管背後的堅實度。它的成功率高達 95~99%，但因爲要開刀很多病人不願接受。早在二十年前就有醫師想到用內視鏡注射一種物質到輸尿管黏膜下層的背後增加它的堅實度，來治療逆流。[2,3] 但早期能用的注射性物質是 Teflon，它是一種石化產品，少數的醫師用它來治療逆流，確實有一定的效果，但因爲它的黏稠度很高，很難用手推，必須用特殊的注射槍，而且它的顆粒很小，在動物實驗發現它會移動到腦部 [4]，因此一直沒能通過衛生主管機關的核准，並未被廣泛使用。後來又有人用 collagen[5]，它是從牛身上提煉的膠原蛋白，初期有效，但幾個月後由於身體吸收，效果完全消失。直到 1995 年瑞典的 Dr. Stenberg 等人提出用逆福仕（Deflux）注射治療逆流 [6]，它是用 Dextran 及 Hyaluronan 的混合物，每 1ml 的逆福仕中含有 0.5ml 的 Dextran 顆粒，顆粒大小是 80~120 μm，遠大於 Teflon 的顆粒，所以它不會有遠端移動的機會，另外 0.5ml 是液態的玻尿酸 Hyaluronan，玻尿酸經過一段時間會被身體吸收，但 Dextran 的顆粒會留在輸尿管黏膜下層造成炎症反應纖維化，使輸尿管黏膜下層的背後更加堅實，而達到防止逆流的目的。在我們有限的病例中，雖然第一次注射的成功率只有百分之五十，較其他報告略低，大部份的報告一次注射的成功率在 67~70%[7,8]，但我們二次注射的成功率就很好，幾乎所有病人都可治癒，雖然我們的病例數目還不是很多，就初期的經驗我們覺得它是一個可靠治療膀胱輸尿管尿液逆流的方法。

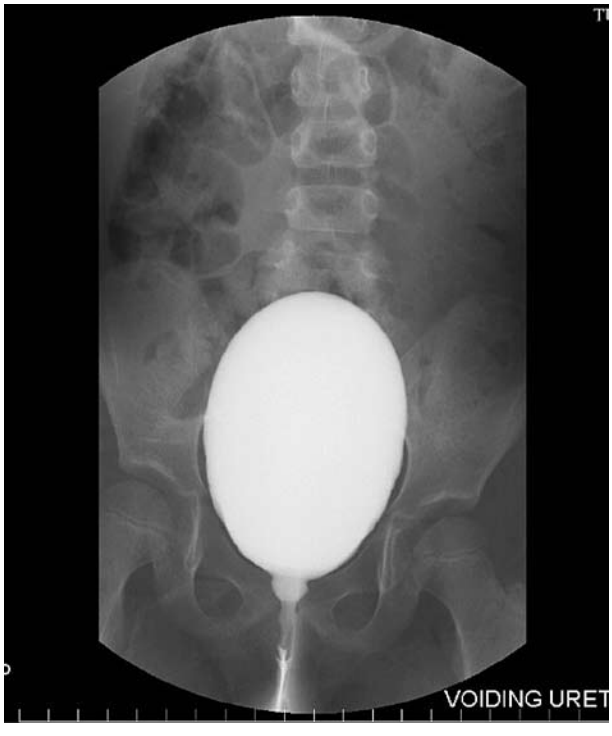

圖 4　注射後膀胱輸尿管尿逆流消失，一年後之追蹤。

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Evaluation the New Method for Vesicoureteral Reflux: Deflux Endoscopic Injection

Jue-Hawn Yin

Vesicoureteral reflux is a common disease in children. The classical treatments included long term oral antimicrobials or surgery. We collected 16 patients of vesicoureteral reflux from Jan. 2004 to Dec. 2006 treated with Deflux® endoscopic injection. After one jection, 50% cases cured. With twice injections all the remaining cases are healed also. We recommended it as a primary treatment for vesicoureteral reflux.
(Tungs' Med J 2007; 1: 6-10)

Key word: Voiding Cystourethrography(VCUG), Endoscopic injection, Deflux, Hyaluronan

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納入糖尿病共同照護網後影響病患遵醫囑行為之研究 - 以某區域教學醫院為例

蔡政麟[1]　葉玲玲[2*]　楊志良[3]　孫培然[4]

前言：除了適當的藥物治療，長期的糖尿病防治必需有完善的衛教計畫。為使各醫療院所加強對糖尿病患的衛教照護，健保局於 2001 年 11 月開始推出糖尿病醫療給付改善方案，期盼透過財務誘因最終能減緩病患病情並控制醫療耗用。

目的：探討納入糖尿病共同照護網後，那些因素會影響病患之遵醫囑行為，以及衛教是否能改善遵醫囑行為。

方法：本研究利用回溯性世代研究法進行資料之收集及分析。從 2002 年 5 月開始至 2005 年 3 月底，累計加入研究醫院糖尿病共同照護網已 25 個月之個案共 1243 人，再經過兩階段以篩檢高比率取藥日，即 25 個月之總取藥日大於或等於 684 日（90%），並至少接受 4 次衛教之研究對象共 487 人。以 SPSS 10.0 統計軟體來進行資料分析。

結果：相較於文獻，本研究個案的平均年齡略低、教育程度較低、罹病時間於 5 年以下者較多、注射胰島素者較少、有嚴重併發症者為少數，而研究資料收集期間則較久（25 個月）；但男女個案比例相近，且體重問題、高血壓、高膽固醇血症及吸煙習慣等心血管危險因子的比例也與文獻極為接近。遵醫囑行為的評量，以藥物治療配合的平均分數最高，其次依序是護照使用、飲食學習、飲食改變、運動，實際得分及其滿分百分比分別是 3.42（85.5%）、2.46（82.0%）、2.69（67.2%）、2.68（67.0%）、2.26（56.5%）。以病患個人資料及接受照護次數為自變項，藥物治療配合、運動配合及護照使用配合為依變項之複迴歸分析分別達到統計意義（p=0.022, 0.001, <0.001）。

結論：在納入糖尿病共同照護網經過 25 個月後，本研究發現年齡、初次糖化血色素、有嚴重合併症及照護總次數是影響病患多項遵醫囑行為最重要的因素，年齡高者較能配合，照護愈多次者配合的愈好；病患的初次糖化血色素偏高及有嚴重合併症則配合的不好。
（童綜合醫誌 2007; 1: 11-22）

關鍵詞：糖尿病醫療給付改善方案、糖尿病共同照護網、遵醫囑行為、糖化血色素。

全世界罹患糖尿病人口逐年不斷增加，國際糖尿病聯盟（IDF）於 2003 年預估全球至 2025 年會達到 3 億的病患。這些年來，糖尿病也成爲國人主要慢性疾病之一，在全人口中糖尿病的盛行率約爲 4.0%，而 20 歲以上成年人盛行率則在 5%~9% 之間，40 歲以上民眾的盛行率則提高到 11%~13%[1,2]。爲加強糖尿病患的病情防治，健保局於 2001 年 11 月開始推出糖尿病給付改善方案，希冀以醫療保險給付的調高來提升醫療照護品質。糖尿病共同照護模式之特色是以疾病管理爲基礎之共同照護理念。疾病管理視病患完整的疾病經驗爲一個連續性臨床流程，並非在不同的醫療照護體系中作分段的醫療處置[3]。疾病管理運用治療指引的建立、醫療資訊的分享、資源管理的技巧及轉診制度的建立，以達到用最低成本創造最高效能[4]。規律的藥物治療、飲食控制及

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適當的運動，這三者是糖尿病醫照護團隊經常要求或建議糖尿病患者應該遵從的行為。而依據研究 [5,6] 指出，糖尿病患者的遵醫囑行為並不理想。

WHO[7] 在 2003 年特別提出對慢性病患遵醫囑行為（adherence）的定義”醫療照顧提供者所建議的各種行為，包括使用藥物、配合飲食、改變生活方式，經病患同意後，病患能夠配合的程度”。遵醫囑行為是慢性病治療過程中影響治療結果最大的因素，尤其生活形態的改變最為困難 [8]。糖尿病患者遵醫囑的行為並不高，約 25%~53% 不等 [9]。綜合國內數個研究 [3,10-12] 可歸納出糖尿病患的遵醫囑行為包含藥物治療、運動、飲食控制三項，而多以服藥遵醫囑行為表現最好，飲食遵醫囑行為次之，以運動遵醫囑行為表現最不好；而病患之人口學資料、健康信念、家庭支持、健康人格控制及併發症情形都可能影響其遵醫囑行為。讓病患瞭解糖化血色素的變化、平常自我監測血糖及糖尿病護照的使用，都能增進動機和配合度 [13]。本研究在探討納入糖尿病共同照護網後，病患不同的遵醫囑行為與那些個人資料有關？增加衛教次數是否能加強遵醫囑行為之改變？

研究設計與方法

本研究利用回溯性世代研究法進行資料之收集及分析。從 2002 年 5 月開始至 2005 年 3 月底累計加入研究醫院糖尿病共同照護網之個案數共 3642 人，為同時收集醫療利用資料做後續研究，先從中進行資料之篩選，初選加入共同照護網後已經過 25 個月之個案共 1243 人，再經過兩階段以篩檢高比率（90% 以上）取藥日之研究對象共 487 人（圖 1）。「遵醫囑行為」分成 5 項 - 藥物治療配合、飲食學習意願、飲食改變配合、運動配合及護照使用，這些資料的取得，則依據衛教護理師和營養師的歷次衛教後評值予以量化計分之加總平均。以 SPSS10.0 統計軟體來分析收案時糖尿病患個人資料、實際接受衛教次數及遵醫囑行為資料。

研究結果

壹、描述性統計

一、收案時病患個人資料及共同照護網的介入頻率

本研究之研究個案全部是加入糖尿病共同照護達 25 個月的病例，總共 487 人符合研究條件，女性較多，有 265 人，佔 54.4%。年齡分佈從 19 歲到 87 歲，平均年齡是 58.78 歲，標準差為 11.11 歲。依年齡分 3 組，以 45 歲至 64 歲組最多，有 276 人，佔 56.7%。教育程度方面，國小以下較多，有 382 人，佔 78.4%。絕大部分已婚，只有 1 人未婚。絕大部分非獨居，只有 1 人獨居。在身體質量指數（BMI）方面，共分成 3 組，依衛生署標準，小於 18.5 kgs/m^2 為過輕組，大於或等於 18.5 kgs/m^2 而小於 24 kgs/m^2 為正常體重組，大於或等於 24 kgs/m^2 為過重或肥胖組。以過重或肥胖組最多，有 350 人，佔 71.9%。

初次糖化血色素（Hba1c）平均是 8.41%，標準差為 1.84%。初次糖化血色素分成 5 組，以 7.1% 至 8.0% 組最多，有 117 人，佔 24.0%。糖尿病罹病時間方面，共分成 4 組，以 5 年以下組最多，有 269 人，佔 55.2%。一二等親糖尿病家族史方面，236 人有糖尿病家族史者，佔 48.5%。323 人有高血壓，佔 66.3%。198 人有高膽固醇血症，佔 40.5%。83 人有吸煙習慣，佔 17.0%。47 人已經有每日注射胰島素，佔 9.7%。腦血管疾病、冠心症、尿毒症、下肢截肢手術等嚴重併發症方面，僅 13 人有嚴重併發症，佔 2.7%（表 1）。所有研究個案在 25 個月內至少接受 4 次的衛教，以 8 次最多，有 205 人，佔 42.1%（表 2）。

二、遵醫囑行為評量資料

藥物治療配合、飲食學習配合方面、飲食改變配合方面、運動配合意願方面這 4 項滿分都定為 4 分，護照使用方法方面，滿分則定為 3 分。以藥物治療配合的平均分數最高，其次依序是護照使用、飲食學習、飲食改變、運動，實際得分及其滿分百分比分別是 3.42（85.5%）、2.46（82.0%）、2.69（67.2%）、2.68（67.0%）、2.26（56.5%）（表 3）。

貳、推論性統計

總結遵醫囑行為評量中，以病患個人資料及接受衛教次數為自變項，藥物治療配合、運動配合及護照使用配合之複迴歸分析達統計意義。年齡在運動配合的迴歸係數為 0.010，在護照使用配合的迴歸係數為 0.004，其解釋力達統計意義。初次糖化血色素在藥物治療配合的迴歸係數為 -0.024，在護照使用配合的迴歸係數為 -0.032，其解釋力達統計意義。有嚴重合併症在運動配合的迴歸係數為 -0.443，其解釋力達統計意義。衛教總次數在藥物治療配合的迴歸係數為 0.034，在運動配合的迴歸係數為 0.088，在護照使用配合的迴歸係數為 0.124，其解釋力達統計意義（表 4）。

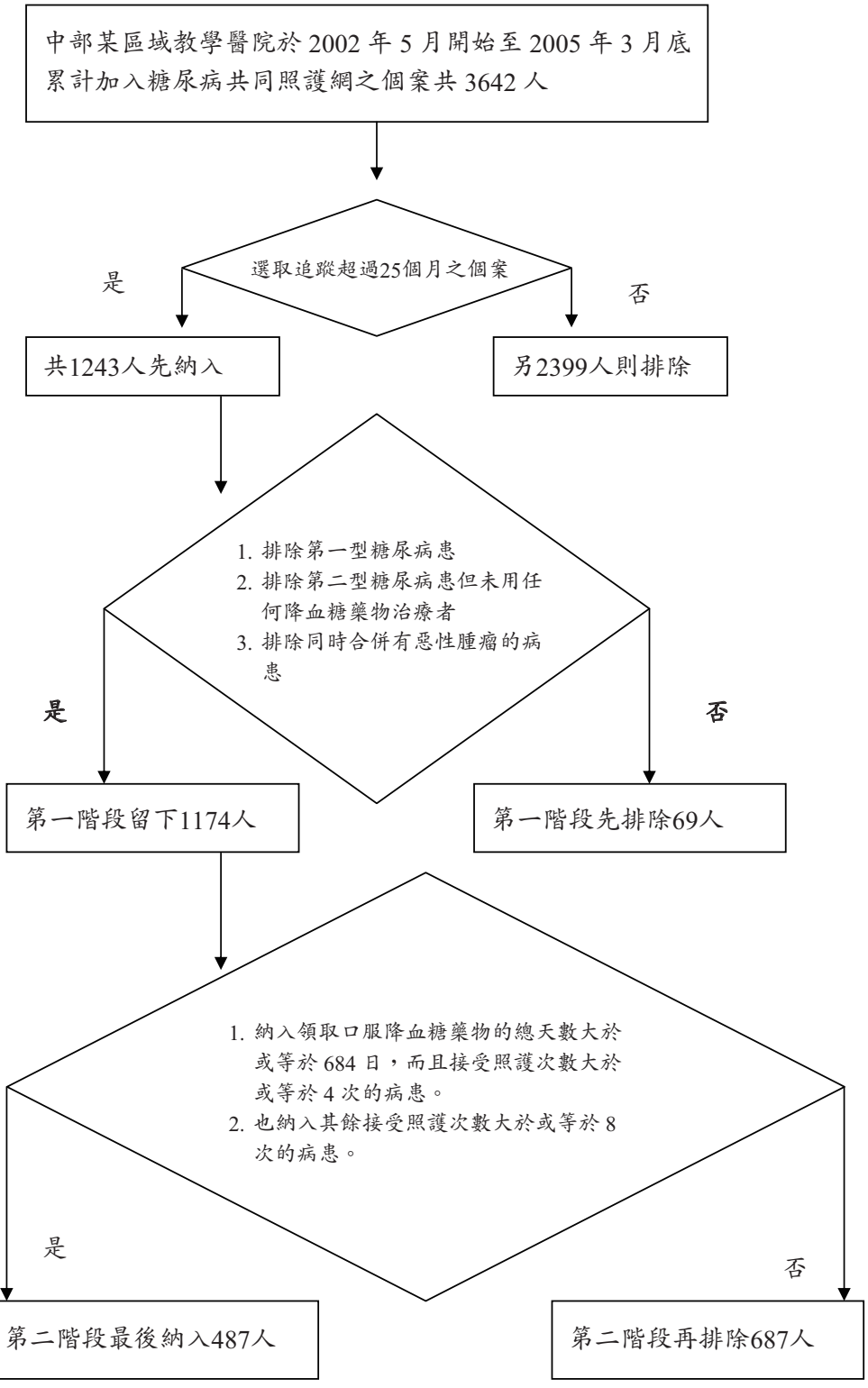
中部某區域教學醫院於 2002 年 5 月開始至 2005 年 3 月底累計加入糖尿病共同照護網之個案共 3642 人
選取追蹤超過25個月之個案
是
否
共1243人先納入
另2399人則排除
1. 排除第一型糖尿病患
2. 排除第二型糖尿病患但未用任何降血糖藥物治療者
3. 排除同時合併有惡性腫瘤的病患
是
否
第一階段留下1174人
第一階段先排除69人
1. 納入領取口服降血糖藥物的總天數大於或等於 684 日，而且接受照護次數大於或等於 4 次的病患。
2. 也納入其餘接受照護次數大於或等於 8 次的病患。
是
否
第二階段最後納入487人
第二階段再排除687人

圖 1 篩檢個案流程圖 -

表 1 收案時病患個人資料

		人數	百分比	平均值	標準差
性別	女	265	54.4		
	男	222	45.6		
年齡(年)				58.78	11.11
年齡分3組	<45	48	9.9		
	45-64	276	56.7		
	≧65	163	33.5		
教育程度	≧中學	105	21.6		
	≦國小	382	78.4		
BMI分3組	BMI<18.5	5	1.0		
	18.5≦BMI<24	132	27.1		
	BMI≧24	350	71.9		
初次Hba1c				8.41	1.84
初次Hba1c 分5組	≦7	115	23.6		
	7.1-8.0	117	24.0		
	8.1-9.0	96	19.7		
	9.1-10.0	69	14.2		
	≧10.1	90	18.5		
糖尿病罹病時間	≦5年	269	55.2		
	6-10年	134	27.5		
	11-20年	72	14.8		
	≧21年	12	2.5		
一二等親糖尿病家族史	無	251	51.5		
	有	236	48.5		
高血壓	無	164	33.7		
	有	323	66.3		
高膽固醇血症	無	289	59.5		
	有	198	40.5		
吸煙習慣	無	404	83.0		
	有	83	17.0		
注射胰島素	無	440	90.3		
	有	47	9.7		
嚴重合併症	無	474	97.3		
	有	13	2.7		

討　論

本研究是回溯性的次級資料分析，只能對各項遵醫囑行爲簡要的衛教紀錄做量化分析，並非問卷調查，也無法回溯病患接受衛教當時的家人支持狀態、健康信念、自我照顧能力、醫病關係及醫療可近性等資料，故難以進行較深入的探討。同時本研究因缺乏加入共同照護網之前的病患遵醫囑行爲資料，以致無法探討加入前後的差異，而著重在參與之後累加的成效探討。再者本研究也同時收集 25 個月內病患醫療利用及費用的資料

另外進行分析，爲盡可能減少這些變項的誤差，故只選取在研究醫院有高比率（≧ 90%）取藥日及至少接受 4 次衛教的病患納入，這些病患的配合度普遍較佳，故研究結果並無法完全代表所有參與糖尿病共同照護網之病患。

本研究的女性略多，佔 54.4%，非常接近蔡文惠 [14] 對北區分局糖尿病患的研究（54.4%）及盧菀淇 [15] 的研究（全國 52.34%、宜蘭縣 56.49%）。平均年齡爲 58.78±11.11 歲，略低於 2004 年糖尿病衛教學會 [16] 全台糖尿病品管調查的 61.9±12.3 歲。依年齡分 3 組，本研究以 45

表 2 共同照護網的介入頻率

		人數	百分比
衛教總次數	4次	9	1.8
	5次	13	2.7
	6次	24	4.9
	7次	54	11.1
	8次	205	42.1
	9次	172	35.3
	10次	9	1.8
	11次	1	0.2

表 3 遵醫囑行爲資料

	平均值	標準差	平均得分百分比
藥物治療配合分數	3.42	0.34	85.5
飲食學習配合分數	2.69	0.34	67.2
飲食改變配合分數	2.68	0.35	67
運動配合分數	2.26	0.76	56.5
護照使用配合分數	2.46	0.49	82

註：藥物治療配合、飲食學習配合、飲食改變配合及運動配合之滿分各爲4分，護照使用配合之滿分爲3分。

表 4 病患個人資料與照護總次數在遵醫囑行爲複迴歸分析之迴歸係數總表

變項	藥物治療配合		飲食學習配合		飲食改變配合		運動習慣配合		護照使用	
	B	標準差	B	標準差	B	標準差	B	標準差	B	標準差
男性（女性爲參考）	-0.010	-0.033	-0.063	0.033	-0.068	0.034	0.036	0.073	-0.073	0.045
年齡	0.002	0.002	-0.001	0.002	-0.001	0.002	0.010**	0.003	0.004*	0.002
中學以上教育程度（國小以下爲參考）	-0.007	0.043	0.039	0.044	0.038	0.045	0.009	0.097	0.079	0.060
過重或肥胖（BMI正常爲參考）	-0.007	0.035	-0.010	0.035	-0.010	0.036	-0.070	0.077	0.059	0.048
初次糖化血色素罹病年數（5年以下爲參考）	-0.024*	0.009	0.012	0.009	0.013	0.009	-0.019	0.020	-0.032*	0.012
6-10年	-0.018	0.037	-0.006	0.038	-0.006	0.038	-0.002	0.083	-0.028	0.052
11-20年	-0.060	0.048	-0.014	0.048	-0.021	0.049	-0.139	0.106	-0.081	0.066
21年以上	-0.018	0.102	-0.133	0.104	-0.125	0.106	-0.112	0.228	0.025	0.142
有家族史	0.051	0.032	0.010	0.032	0.013	0.033	0.007	0.071	-0.005	0.044
有高血壓	0.064	0.033	-0.015	0.033	-0.027	0.034	0.129	0.073	-0.022	0.046
有高膽固醇血症	-0.053	0.032	-0.017	0.032	-0.006	0.033	0.005	0.070	-0.030	0.044
有吸煙習慣	0.020	0.041	0.047	0.042	0.053	0.042	-0.046	0.091	-0.048	0.057
有注射胰島素	0.070	0.056	-0.036	0.057	-0.033	0.058	-0.004	0.124	0.024	0.078
有嚴重合併症	-0.033	0.097	-0.099	0.099	-0.076	0.101	-0.443*	0.217	0.031	0.135
照護總次數	0.034*	0.013	0.009	0.014	0.010	0.014	0.088**	0.030	0.124**	0.019
R^2	0.027*		-0.002		-0.001		0.046*		0.102**	

註：*p<0.05, **p<0.01

歲至 64 歲中壯年組最多（56.7%），超過一半，盧菀淇[15] 的研究發現以 70 歲以上者佔最多（全國 40.49%、宜蘭縣 46.01%），其差異很可能是取樣不同所造成。國小以下的教育程度較多人（78.4%），明顯高於許惠恒[17] 之研究（52.3%）及李玉宥[18] 之研究（56.4%），應與本研究的個案普遍在中壯年以上有關，而且研究醫院不在都會區，相對教育資源較少、教育程度較低。絕大部分個案已婚且非獨居，顯示應有基本的家庭支持。計算身體質量指數則以過重或肥胖組最多（71.9%），介於 2004 年糖尿病衛教學會[16] 全台糖尿病品管調查（66.9%）及世界糖尿病聯盟[19]（International Diabetes Federation IDF）2004 年的資料（80%）之間，可見糖尿病患大多數有體重問題，更加重疾病防治的困難。

初次糖化血色素分組，良好控制組（7.0% 以下）只佔 23.6%，不到四分之一，控制不良分組（9.0% 以上）則佔 32.7%，而 2004 年糖尿病衛教學會[16] 全台糖尿病品管調查，前者佔 17.0%，後者則佔 43.4%。相較之下，本研究個案群的血糖控制略好，主要原因很可能是選取了高比率取藥日之研究個案。糖尿病罹病時間以 5 年以下組最多（55.2%），高於許惠恒[17] 之研究（38.3%），以此推估，嚴重併發症應該是少數，也能部分解釋前述本研究個案群的控制較全台糖尿病品管調查略好的原因。一二等親有糖尿病家族史者佔 48.5%，接近李玉宥[18] 之研究（50.0%），可見後天環境因素的影響也很大。三分之二有高血壓，高於李玉宥[18] 之研究（52.8%）及林育慈[20] 之研究（51.6%），而低於美國糖尿病學會[21]2006 年的資料（73%），高血壓更加重了糖尿病患心血管疾病的危險。40.5% 有高膽固醇血症，接近林育慈[20] 之研究（46.5%），糖尿病患合併高膽固醇血症的比例很高，也加重病患心血管疾病的危險。17.0% 有吸煙習慣，很接近李玉宥[18] 之研究（17.6%），突顯戒煙是不容忽略的照護重點，若能夠結合現行戒煙門診，應該事半功倍。90.3% 只用口服藥而未注射胰島素，比例偏高，應與本研究排除第一型糖尿病患，以及五分之四個案因罹病時間小於 10 年多數還不需要注射胰島素，這兩因素有最大關係。97.3% 並無腦血管疾病、冠心症、尿毒症、下肢截肢手術等嚴重併發症，佔絕大多數，原因應與收案條件有關。

與上述文獻相較，本研究之個案平均年齡略低、教育程度較低、罹病時間於 5 年以下組較多、注射胰島素者較少、有嚴重併發症爲少數，而研究資料收集期間則較久（25 個月）；但男女個案比例相近，且體重問題、高血壓、高膽固醇血症及吸煙習慣等心血管危險因子的比例也與文獻極爲接近。共同照護網的介入頻率以接受衛教 8 次最多，9 次其次，4 次至 6 次僅佔 9.4%，應與本研究篩選高比率取藥日之研究個案有關係。雖然共同照護網有配套衛教照護和標準流程，實務上受到很多因素干擾，不太可能完全規律的進行，這些不等次的衛教正好提供研究的變項做分析探討。

護照使用配合以外，病患的遵醫囑行爲以藥物治療配合的平均得分最高，應與本研究篩選高比率取藥日之研究個案有相當關係，其次低序是飲食學習配合、飲食改變配合、運動配合，此結果與國內多篇研究相同[2,10-12]。若同時評比護照使用配合，其得分僅次於藥物治療配合。除了本研究設有選案條件外，病患的藥物治療遵醫囑行爲相對較好的原因至少還有二個，第一，用藥屬於治療行爲，病患基於有病治病的心態，非得接受這項行爲[22]，何況糖尿病是慢性病。第二，用藥處方較飲食、運動處方清楚，病患容易依從。而飲食配合遵醫囑行爲方面，由於多數民眾欠缺正確的營養知識，加上傳統「能吃就是福」的觀念及個人偏好，多已養成固定的飲食習慣，日常生活中的社交、應酬及各種限制更會干擾健康的飲食計劃，造成配合上的困難。

由表 4 可知在控制其它變項後，只有初次糖化血色素及衛教總次數會影響藥物治療之配合，初次糖化血色素愈高配合愈不好，衛教愈多次配合愈好，而初次糖化血色素的解釋力大於衛教總次數。年齡、有嚴重合併症及衛教總次數會影響運動習慣之配合，年齡增加愈能配合，有嚴重合併症較不能配合，衛教愈多次配合愈好，其解釋力大小依序爲年齡、衛教總次數、有嚴重合併症。年齡、初次糖化血色素及衛教總次數會影響護照使用之配合，年齡增加愈能配合，初次糖化血色素愈高配合愈不好，衛教愈多次配合愈好，其解釋力大小依序爲衛教總次數、初次糖化血色素、年齡。年齡及衛教總次數會影響整體配合，年齡增加愈能配合，衛教愈多次配合愈好，衛教總次數的解釋力大於年齡。飲食學習配合及飲食改變配合在複迴歸分析並未出現統計意義，有可能是衛教及評量方式不盡理相，或病患未據實回答，宜做修訂調整。未來經由評量工具的改良，如題庫抽考、回覆示教，有可能更清楚呈現出病患學習及行動上的差異。鄭英裕[10] 的研究發現健康信念最可預測糖尿病患的飲食控制，而大專以上者的藥物治療配合優於其它教育程度，可見病患的認知及學習能力在照護過程仍有相當影響；另發現無職業者比有職業者有較好的飲食控制行爲，可能原因是有職業者平日忙於工作，較容易誤餐，加上外食多，使飲食控制更加困難，其研究足供專業參考。

整體看來，病患的教育程度並不造成太明顯的影響，而年齡、初次糖化血色素、有嚴重合併症及衛教總

次數是影響遵醫囑行爲最重要的因素，較年輕的病患很可能因爲工作或健康危機意識不夠，無法做良好的配合；初次糖化血色素偏高代表以往就沒控制好；有嚴重合併症會造成看診及活動的障礙，以上族群特別要依標準給予定期衛教照護，可望提升其遵醫囑行爲。

結　論

綜合許多研究[15,17,18,20,23-28]，我國糖尿病共同照護網實施以來，在照護的結構面及過程面都有明顯的成效，包括治療團隊專業人力的增加和普及、衛教師的訓練及認証、各種糖尿病相關的檢驗檢查執行率增加、病患就醫可近性的改善等。本研究發現藥物治療配合之遵醫囑行爲的平均分數最高（85.5%），其次依序是護照使用配合（82%）飲食學習配合（67.2%）飲食改變配合（67%）、運動配合（56.5%）。整體看來，病患的年齡、初次糖化血色素、有嚴重合併症及接受衛教總次數是影響各項遵醫囑行爲最重要的因素，年齡高者較能配合，衛教愈多次配合會愈好，至於病患的教育程度則不造成的明顯的影響。此研究結果相當支持在糖尿病共同照護網的架構下，愈依照醫政單位現行標準的照護，病患的遵醫囑行爲會愈好。而愈好的遵醫囑行爲是否對治療成效及醫療耗用有愈多的正面影響，有待更進一步探討。

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附錄-

童綜合醫院　糖尿病個案護理衛教紀錄

共照碼					
次數					
日期					
是否按時按量服藥	□是□否	□是□否	□是□否	□是□否	□是□否
是否按時就診	□是□否	□是□否	□是□否	□是□否	□是□否
按醫囑注射胰島素	□常常□有時□很少	□常常□有時□很少	□常常□有時□很少	□常常□有時□很少	□常常□有時□很少
個案目前治療方式	□飲食□運動 □OHA_____種 藥名及服用方式： □Insulin____種 保存□對□錯 注射方法□對□錯 時間□正確□不正確 部位□有更換□未更換 藥名及注射方式：	□飲食□運動 □OHA_____種 藥名及服用方式： □Insulin____種 保存□對□錯 注射方法□對□錯 時間□正確□不正確 部位□有更換□未更換 藥名及注射方式：	□飲食□運動 □OHA_____種 藥名及服用方式： □Insulin____種 保存□對□錯 注射方法□對□錯 時間□正確□不正確 部位□有更換□未更換 藥名及注射方式：	□飲食□運動 □OHA_____種 藥名及服用方式： □Insulin____種 保存□對□錯 注射方法□對□錯 時間□正確□不正確 部位□有更換□未更換 藥名及注射方式：	□飲食□運動 □OHA_____種 藥名及服用方式： □Insulin____種 保存□對□錯 注射方法□對□錯 時間□正確□不正確 部位□有更換□未更換 藥名及注射方式：
合併使用其他藥物	□心臟病藥□降三酸甘油□降血壓□降膽固醇□甲狀腺製劑□氣管擴張□腸胃道藥物	□心臟病藥□降三酸甘油□降血壓□降膽固醇□甲狀腺製劑□氣管擴張□腸胃道藥物	□心臟病藥□降三酸甘油□降血壓□降膽固醇□甲狀腺製劑□氣管擴張□腸胃道藥物	□心臟病藥□降三酸甘油□降血壓□降膽固醇□甲狀腺製劑□氣管擴張□腸胃道藥物	□心臟病藥□降三酸甘油□降血壓□降膽固醇□甲狀腺製劑□氣管擴張□腸胃道藥物
是否運動	□是□偶爾□否	□是□偶爾□否	□是□偶爾□否	□是□偶爾□否	□是□偶爾□否
□阻礙運動之健康問題： 1.心臟 2.膝關節 3.肩關節 4.脊椎 5.中風 6.其他	無運動原因： □ 懶□沒時間 □認為每天勞動量已很大□阻礙運動之健康問題：______	無運動原因： □ 懶□沒時間 □認為每天勞動量已很大□阻礙運動之健康問題：_____	無運動原因： □ 懶□沒時間 □認為每天勞動量已很大□阻礙運動之健康問題：_____	無運動原因： □ 懶□沒時間 □認為每天勞動量已很大□阻礙運動之健康問題：_____	無運動原因： □ 懶□沒時間 □認為每天勞動量已很大□阻礙運動之健康問題：_____
運動種類：1.爬山 2.打球 3.散步 4.游泳 5.騎腳踏車 6.太極拳 7.跳舞 8.體操 9.其他	種類：_____ 時間：_____分/次 次數：_____次/週	種類：_____ 時間：_____分/次 次數：_____次/週	種類：_____ 時間：_____分/次 次數：_____次/週	種類：_____ 時間：_____分/次 次數：_____次/週	種類：_____ 時間：_____分/次 次數：_____次/週
每次運動時段及時間	□ 飯前□飯後	□ 飯前□飯後	□ 飯前□飯後	□ 飯前□飯後	□ 飯前□飯後
血糖自我監測頻率（多久一次）	□ 否□是 次/ 天/週	□ 否□是 次/ 天/週	□ 否□是 次/ 天/週	□ 否□是 次/ 天/週	□ 否□是 次/ 天/週
低血糖	□ 否□是 □ 1 個月內 □ 1 年內 □ 1 年以上 處理方式： □對□不對	□ 否□是 □ 1 個月內 □ 1 年內 □ 1 年以上 處理方式： □對□不對	□ 否□是 □ 1 個月內 □ 1 年內 □ 1 年以上 處理方式： □對□不對	□ 否□是 □ 1 個月內 □ 1 年內 □ 1 年以上 處理方式： □對□不對	□ 否□是 □ 1 個月內 □ 1 年內 □ 1 年以上 處理方式： □對□不對
抽煙	□否□是 支/天	□否□是 支/天	□否□是 支/天	□否□是 支/天	□否□是 支/天
穿鞋襪情形	□ 適當□尚可 □不適當	□ 適當□尚可 □不適當	□ 適當□尚可 □不適當	□ 適當□尚可 □不適當	□ 適當□尚可 □不適當
心理社會適應	□ 佳 □尚可 □ 不佳	□ 佳 □尚可 □ 不佳	□ 佳 □尚可 □ 不佳	□ 佳 □尚可 □不佳	□ 佳 □尚可 □ 不佳

門診需接受護理衛教的項目						
	衛教項目/日期					
第一階段	1.認識糖尿病					
	2.口服降血糖藥物					
	3.胰島素注射					
	4.糖尿病護照使用					
	5.低血糖處理					
第二階段	6.慢性合併症					
	7.高血糖處理					
	8.血糖自我監測					
	9.運動（建議運動項目） 時間(　　分鐘)					
	10.生病時的處理					
第三階段	11.足部照顧					
	12.戒菸、戒酒					
	13.社會心理支持					
	14.旅遊注意事項					
	15.其他					
護理師簽名						
病患或家屬簽名						

＊衛教評值：　○：佳　　△：尚可　　X：需加強　　C：其他照顧者

＊衛教成果評估

＊藥物配合情形：□良好　□尚可　□不佳　□拒吃

＊運動配合意願：□強烈　□普通　□勉強　□無意願

＊ 病患整體狀況總評：□良好　□尚可　□不佳

＊ 態度部分：□ 完全配合 □ 大部份配合 □普通 □ 大部份不配合 □ 完全不配合

童綜合醫院　糖尿病病患營養衛教記錄表(一)

病患學習狀況

病患對糖尿病飲食控制之學習意願: 強烈　普通　勉強　無

病患是否主動參與飲食習慣改變:　強烈　普通　勉強　無

營養衛教項目與計劃

飲食計劃及餐次分配

糖尿病的飲食原則

食物代換

外食技巧

生病時的飲食調整

低血糖的處理

旅遊飲食注意事項

節慶飲食原則

低熱量點心建議

低蛋白飲食原則

低油飲食原則

低鹽飲食原則

低普林飲食原則

高脂血症飲食原則

其他

_____ k/d___餐/天　Protein:_____g(　%)　Fat:_____ g(　%)　CHO:_____ g(　%)

	ex	P	F	C	B	Bs	L	Ls	D	Ds
cereal										
meat										
vegetable										
fruit										
milk										
oil										

◎衛教營養師		追蹤日期	年　　月　　日

Impact of Patients' Adherence Under the Share Care Disease Management Program for Diabetes – An Example from A Regional Teaching Hospital

Cheng-Lin Tsai[1], Ling-Ling Yeh[2*], Chih-Liang Yaung[3], Pei-Ran Sun[4]

Background: Besides adequate medications, long-term diabetes mellitus (DM) control depends on comprehensive education programs. To enhance delivery of education programs to diabetic subjects in various medical setting, the Bureau of National Health Insurance proposed the Share Care Disease Management Program for Diabetes in November 2001, and expected to slow down diabetes progression and control medical utilization via financial incentives.

Purpose: To investigate diabetic patients' adherence after they participated in the program and to study whether this program improved adherence.

Methods: A total of 487 subjects were selected via 2-steps from 1,243 patients who had participated in this program for at least 25 months between May 2002 and March 2005. These patients had received at least 684 days (90% of the observed duration) medication and at least 4 periods of education from nurses and dietitians. All the data were collected retrospectively and analyzed with SPSS 10.0 software.

Results: Compared to the literature, the baseline characteristics of the study cases included a lower average age, lower education background, majority with less than 5 years history of DM, fewer receiving insulin injections and fewer with severe complications. Further, the duration of data collection was longer (up to 25 months) but the gender ratio and cardiovascular risk factors, such as abnormal body weight, hypertension, hypercholesterolemia and smoking, were similar. Adherence to medication (85.5%) was scored the highest in the adherence evaluation results. Other scored items in sequence were DM passport usage (82%), diet behavior learning (67.2%), diet modification (67%) and exercise (56.5%). With baseline characteristics and education frequency of the patients as independent variables, the dependent variables of medication, exercise and DM passport usage were significant according to multiple regression analysis ($p = 0.022, 0.001, <0.001$).

Conclusion: We analyzed 25-months of data from patients after they had enrolled in the Shared Care Disease Management Program for Diabetes. This research found that age, baseline HbA1c, severe complications and education frequency were very important factors for adherence. The older patients and those who had received more diabetic education showed better adherence. Patients with higher baseline HbA1c and those with severe complications showed poorer adherence. (Tungs' Med J 2007; 1: 11-22)

Key words: Shared Care Disease Management Program for Diabetes, Shared Care Network for Diabetes, adherence, Hemoglobin A1c.

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Transesophageal Echocardiography-An Alternative Introduction Method

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Yiu-Sion Chen[1], Kuo-Sheng Huang[1], Liang-Chieh Wang[1]

Background: Intraoperative transesophageal echocardiography (TEE) is now being done in many operations for monitoring and diagnostic procedure. It is generally contemplated as a relative safe and minimally invasive technique. However, unfavorable events may occur during TEE introduction or manipulation. Despite a high successful rate of TEE introduction was reported, special care may be taken in endotracheal intubated patients. Endotracheal tube may interfere with TEE probe introduction and requiring additional manipulations. Failure to introduce or advance the TEE probe in 0.18%-1.9% of anesthetized patients. In our study, we assessed whether the Portex laryngeal mask airway introducer (Portex introducer) can be an aid for TEE introduction in patients under endotracheal general anesthesia.

Methods: 80 ASA II-III adult patients undergoing cardiac surgery were randomly allocated to TEE group, n=40 or TEE-I group, n=40 (TEE with the Portex introducer). Endotracheal intubation after the induction of fentanyl (3-5 ug/kg), etomidate (0.4mg/kg) and rocuronium (0.6mg/kg) was given to facilitate tracheal intubation. TEE introduction was done with regular blind method in TEE group, but the Portex introducer was placed in the mouth prior TEE introduction in TEE-I group. TEE probe was advanced along the Portex introducer as an "artificial hard palate" which intends to guide the TEE probe sliding through the pharyngeal turn and deflects the probe down into hypopharynx, and maintains a midline approach for the probe toward the esophageal inlet. The frequency of attempts of TEE introduction and introduction time were compared with each group. Complications caused by TEE were also recorded in two groups. Chi-square test with Yates' correction, Fisher's exact test and two tailed Student's t-test were used for statistics.

Results: Not only the successful first-time introduction rates, TEE group 34(85)% vs TEE-I group 40 (100%, $p<0.05$), were lower for the TEE group, but also the TEE introduction was assisted by laryngoscope in a patient of TEE group. There were 5 patients succeed in introduction after subsequent second attempt in TEE group. The introduction time was much significant faster in TEE-I group (9.2 ± 1.69sec., $p<0.05$) than in TEE group (12.2 ± 3.84 sec.). The frequency of postoperative sorethroat were 11 (28.2%) in TEE group and 6 (15%) in TEE-I group. A case of dysphagia and 3 cases had blood-tinged sputum after removal of probe in TEE group were noted. The patients

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experienced small tear over left upper lip were 2 in TEE group and 1 in TEE-I group.

Conclusion: We showed that TEE introduction can be done more smoothly and promptly by the aid of the Portex introducer. It helps the TEE probe to overcome the major impediment of the pharyngeal turn during introduction and maintains a midline approach for the probe toward the esophageal inlet even the patients were endotracheal tube intubated.
(Tungs' Med J 2007; 1: 23-28)

Key words: Transesophageal Echocardiography, Introduction, Portex Introducer, Cardiac Anaesthesia

Transesophageal echocardiography (TEE) is used rountinely in cardiac surgery and other high risk surgery for diagnostic or monitoring procedures during anesthesia. It is generally considered a safe and minimally invasive technique.[1] However, adverse events may occur during introduction and manipulation of the TEE probe. These included dental, oropharyngeal, esophageal injuries and failure of TEE introduction may be also happened.[2] The presence of pre-existing endotracheal tube may disturb and even impede the introduction of TEE. The range of failed intraoperative TEE introduction were reported from 0.18% to 1.9%.[3,4]

There are some techniques such as the finger-guided,control-guided and laryngoscope-guided maneuvers or other bare-handed manipulations:for instance, deflate the endotracheal cuff, extending the patient's neck and anterior displacement of the patient's mandible that may be some helpful in suchlike dilemma of failed TEE introduction.[5,6] The purpose of the study was to investigate the feasibility and safety of the Portex introducer to be an aid for TEE introduction in patient under endotracheal general anesthesia (Fig. 1) .

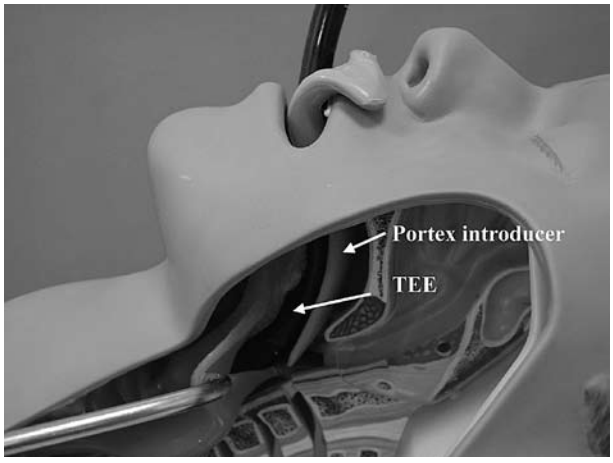

Fig. 1 The Portex introducer intends to guide the transesophageal echocardiography (TEE) probe to slide through the pharyngeal turn during introduction.

MATERIALS AND METHODS

After obtaining the approval of institutional ethics committee and informed consent from each patient.80 adult patients (ASA physical status II or III) scheduled for cardiac surgery were enrolled in the study. Patients were randomly allocated into two groups. One group of patients received TEE introduction under control-guided maneuver (TEE group); the other group of patients were performed with TEE introduction by the aid of the Portex introducer (Portex Ltd., Kent, UK.) (TEE-I group). The Portex introducer is developed and manufactured with silicone material by Portex Ltd. And it is designed both to navigate the laryngeal mask airway to the right placement of hypopharynx and to reduce trauma during insertion. The Portex introducer is available in one adult size. Pateints were excluded on the basis of history or symptoms and physical signs of gastrointestinal tract disease, oropharyhgeal disease, periodontitis, bleeding tendency and difficult airway.

All patients were continuously monitored on five-lead EKG, arterial line, pulse oximetry, capnography and pulmonary artery pressure. General anesthesia was induced intravenously with fentanyl (3-5 ug/kg), etomidate (0.4mg/kg) and rocuronium (0.6mg/kg)

was given to facilitate endotracheal intubation. The TEE (multiple plane probe) introduction and manipulation were carried by three anesthesiologists who had been performing TEE studies for at least 2 years. The protective bite guard was placed to the patients of TEE group prior to TEE introduction. On the other hand, the Portex introducer was put into the patient's mouth of TEE-I group as an "artificial hard palate" to guide the TEE probe negotiating the pharyngeal turn (the angular junction between the oral cavity and the posterior pharyngeal wall) (Fig. 1, Fig. 2).[7]

We recorded and compared the frequency of attempt of TEE introduction and introduction time with each groups. The TEE introduction time was counted from the beginning of insertion of TEE probe into the patient's mouth via the protective bite guard in TEE group or Portex introducer in TEE-I group, individually. The laryngoscope can be used to assist the difficult TEE introduction in the case of more than two attempts. The case of laryngoscope-assisted introduction will be excluded from the study. All the complications assessment were executed by an blinded observing anesthesiologist. Data regarding age, weight, height and time for TEE introduction were expressed as mean±standard deviation. An two tailed student's *t*–test was used for comparison of means between groups. Chi-square test with Yates' correction and Fisher's exact test were applied for statistical analysis of frequency of attempts and complications of TEE introduction when appropriate. Results were considered statistically significant at $p<0.05$.

RESULTS

There were no significant differences in sex, age, weight and height between two groups (Table 1). Not only the successful first-time introduction rates, TEE group 34(85)% vs TEE-I group 40(100%, $p<0.05$), were lower for the TEE group, but also the TEE introduction was assisted by laryngoscope in a patient of TEE group (Table 2). There were 5 cases (12.5%,) of more than 1 attempt occurred in TEE group but not happen to TEE-I group (Table 2). The time for TEE introduction were 9.2±1.69 sec. in TEE-I group and 12.2±3.84 sec. in TEE group, respectively (Table 3). The results showed TEE introduction is statistically significant faster in TEE-I group ($p<0.05$) than in TEE group. No major complication attributable to the use of TEE occurred in both groups. Even though it was not statistically significant in complications among both groups, 1 dysphagia and 3 blood-tinged sputum were noted in TEE group. The incidence of sorethroat were 11 (28.2%) in TEE group and 6 (15%)

Fig. 2 The pharyngeal turn is the major deterrent and the common bleeding point during transesophageal echocardiography(TEE) introduction.

Table 1. Demographic Data

	TEE (n=40)	TEE-I (n=40)
Sex(M/F)	28/12	23/17
Age(yr)	48.4±5.2	53.6±2.3
Weight(kg)	54.5±3.9	58.1±2.4
Height(cm)	163.6±4.6	159±6.2

Values are mean±SD. TEE=transesophageal echocardiography group.
TEE-I= transesophageal echocardiography with Portex introducer group.
There were no statistically significant differences between groups.

Table 2. Efficacy of Transesophageal Echocardiography Introduction

Introduction	TEE (n=40)	TEE-I (n=40)	P Value
Frequency of Attempt			
First Attempt	34 (85%)	40 (100%)	P< 0.05
Second Attempt	5 (12.5%)	0	NS
Laryngoscope Assisted Introduction	1 (2.5%)	0	NS

TEE=transesophageal echocardiography group.
TEE-I= transesophageal echocardiography with Portex introducer group.
Statistical difference analyzed by Fisher's exact test.
P<0.05 was statistically significant between groups.
NS= no statistically significant differences between groups.

in TEE-I group, there was no significant difference in both groups. The accident of upper lip tear happened 2 cases in TEE group and 1 cases in TEE-I group (Table 4).

DISCUSSION

TEE is an invaluable intraoperative diagnostic monitor that has acquired widespread use in cardiac surgical procedure. It is considered reasonably noninvasive and safe; however, introduction and manipulation of TEE probe may cause hypopharngeal, esophageal, or gastric trauma and even mortality.[2-4,8] The overall morbidity is 0.2%-0.4% with no difference between patients receiving sedation or general anesthesia.[3,9] Furthermore, the range of failed intraoperative TEE introduction were reported from 0.18% to 1.9%.[3,4] During general anesthesia, endotracheal tube in situ may interfere and cause as an obstacle to the TEE introduction and requiring additional manipulations. There are a few methods to assist or improve the TEE introduction into an anesthetized, tracheally intubated patient by some bare-handed manipulations such as displacing the mandible anteriorly or flexing the neck will get some help in cases.[6]

An alternative technique for TEE introduction was used and assessed in our study other than the traditional techniques such as the finger-guided, control-guided and laryngoscope-guided maneuvers described in the manufacturer's application guideline. 5 The Portex introducer acts as an "artificial hard palate" to conduct the TEE probe negotiating the pharyngeal turn which is the place to cause usually mucosal bleeding during TEE introduction (Fig. 2).[9] The probe will be advanced along the ramp of the Portex introducer and guided into the esophagus straightly neglecting the interference of endotracheal tube in situ (Fig. 1, Fig. 3). The achievement of the Portex introducer in TEE introduction was proved in the study. As the TEE introduction in TEE-I group were completed in all patients at the first attempt and was statistically significant faster than TEE group ($p<0.05$) (Table 2). TEE introduction were successful in 5 patients (12.5%) after two attempts in TEE group. It is compatible with the result of requiring second attempt to insert the TEE probe in 6 patients (13.3%) in previous study.[10] Nevertheless, a case was excluded in the study owing to the requirement of laryngoscope-assisted introduction after second attempt in TEE group.

No study has been addressed to the time for TEE introduction. We recorded the data and found out that

Table 3. Time for Transesophageal Echocardiography Introduction

	TEE (n=39)	TEE-I (n=40)	P Value
Introduction Time (sec)	12.2±3.84	9.2±1.69	P<0.05

Values are mean±SD, TEE=transesophageal echocardiography group.
TEE-I= transesophageal echocardiography with Portex introducer group.
Statistical difference analyzed by two-tailed Student's *t*-test.
P<0.05 was statistically significant between groups.

Table 4. Complication of Transesophegeal Echocardiography Introduction

	TEE (n=39)	TEE-I(n=40)	P Value
Sorethroat	11 (28.2%)	6 (15%)	NS
Dysphagia	1	0	NS
Blood-tinged Sputum	3	0	NS
Upper lip small tear	2	1	NS

TEE=transesophageal echocardiography group.
TEE-I= transesophageal echocardiography with Portex introducer group.
Statistical difference analyzed by Chi-square test with Yates' correction.
NS= no statistically significant differences between groups.

Fig. 3 The successful introduction of transesophageal echocardiography by using the Portex introducer in endotracheal intubated patient.

it was statistically significant faster in introduction time of TEE-I group (9.2±1.69 sec., $p<0.05$) than that in TEE group (12.2±3.84 sec.). It revealed obviously that the TEE probe passes through the oropharyngeal cavity smoothly and promptly when it is nestling along with the Portex introduction.

Complications of TEE introduction and manipulation can be conveniently divided into three groups. They are (1) those attributable to direct mechanical trauma, (2) those attributable to displacement or traction on contiguous structures (such as the recurrent laryngeal nerve), and (3) those attributable to stimulation of visceral reflexes. 6 There were 1 dysphagia case and 3 blood-tinged sputum cases in TEE group, but not in TEE-I group (Table 4). The Portex introducer seems to avoid the direct mechanical trauma to oropharyngeal mucosa during TEE introduction and manipulation. And the compressible force exerted by the shaft of TEE against the upper airway is reconciled with protection of the Portex introducer. Although 18% of sorethroat was reported by Dingley et al when the Portex introducer was used to assist the insertion of laryngeal mask airway,[11] the incidence of sorethroat in TEE-I group was less than that in TEE group in spite of there was not significant difference between both group as 11(28.2%) in TEE group and 6 (15%) in TEE-I group. Apparently, endotracheal intubation itself may induce postoperative sorethroat rather than that caused by TEE introduction with or without the aid of Portex introducer.[12-13] Furthermore, the accident of upper lip tear happened 2 cases in TEE group and 1 cases in TEE-I group,individually (Table 4). The reasons for lip tear were thought to be caused by laryngoscopy and endotracheal intubation.

In conclusion, intraoperative TEE introduction may be failed and caused complications especially with the interference of endotracheal tube in situ during general anesthesia.[3-4,8-9] We demonstrated that TEE introduction can be done smoothly,easily and promptly by the facilitation of the Portex introduction. It helps the TEE probe to overcome the major deterrent of pharyngeal turn during TEE introduction and maintain a midline approach toward the esophageal inlet even the patients are endotracheal tube intubated.

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經食道心臟超音波－另一種置放技術

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背景：經食道心臟超音波（TEE）經常在手術中被用作心臟功能的監視及診斷。一般而言，這是一種既安全又侵入性少，而且食道置放成功率相當高的技術。然而，在超音波探頭置入操作時，可能會發生一些狀況。尤其當病人已有氣管內管插管時，氣管內管可能會妨礙超音波探頭的置入，甚至需要一些特別的方法。我們研究 Portex 導入器是否能幫助超音波探頭置入食道時操作得更形容易。

方法：選擇 80 位 ASA II ～ III 需手術的病人，分為兩組：一組為經食道心臟超音波組（TEE 組，n ＝ 40），另一組為經食道心臟超音波和導入器組（TEE － I 組，n ＝ 40），以 fentanyl (3-5 ug/kg), etomidate (0.4mg/kg) 和 rocuronium (0.6mg/kg) 施行氣管內管插管全身麻醉後，所有病人在手術中均接受經食道心臟超音波的監視或診斷。在 TEE 組是以一般方式把超音波探頭置入食道中，但在 TEE － I 組中，先在病人嘴中放入導入器，作為“人工硬顎”。然後，超音波探頭置入時，便順著導入器滑入口腔中，繼續導引其通過口腔與咽部的轉折處，到達下咽部再送入食道的正確位置。在研究中，比較兩組操作時的置入成功率、成功置入食道所需的時間及手術後發生的併發症。

結果：兩組的第一次便成功置入的比率，以 TEE － I 組 40（100%，p<0.05）優於 TEE 組 34（85%）。在繼續操作後，TEE group 也全數成功。超音波探頭置入食道所需的時間亦以 TEE － I 組（9.2±1.69 sec, p<0.05）快於 TEE 組（12.2±3.84 sec）。術後發生喉痛的比率上，兩組分別為：TEE － I 組，11（28.2%）及 TEE 組，6（15%），沒有統計上的差異。另在 TEE 組中有一位手術後吞嚥困難和三位在拔除的超音波探頭上發現沾有血絲的口水及二位的左嘴唇上有輕微撕裂傷，在 TEE － I 組中則只有一位是左嘴唇上有輕微撕裂傷外，並無其他嚴重的問題發生。

結論：我們証明在氣管內管插管全身麻醉的病人中，Portex 導入器使超音波探頭克服口腔與咽部轉折處的障礙及氣管內管的干擾，讓超音波探頭置入食道時操作得更形容易。

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男性脊髓損傷者的性健康狀態初探

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背景：性健康照護隨著社會的進步成為臨床重要的照護需求，本研究主要目的在探討男性脊髓損傷患者損傷後的性健康狀況。

方法：本研究採訪談記錄回顧性描述性研究，期間從 2001 年 9 月 1 日至 2005 年 9 月 30 日止，樣本為 18 份男性脊髓損傷患者性諮商護理記錄。

結果：研究結果顯示：1. 受評者性的健康問題不分年齡層及損傷程度；2. 受評者 72% 有勃起功能，其中近六成硬度不足，有一半無精液排出；3. 損傷後的性慾、性滿意度及性關係皆受影響；4. 受評者一半以上損傷前的性教育與性資訊來自學校公民課、影片及報章雜誌，損傷後有六成提出無性資訊來源；5. 受評者 50% 的人損傷後三個月開始思考到性問題，而近一半的人在半年後才開始接受性諮詢。

討論：臨床健康照護者應更加主動關注損傷者性健康問題，建議臨床人員宜在損傷後滿 2 個月即可提供男性脊髓損傷患者相關的性護理措施。

（童綜合醫誌 2007; 1: 29-32）

關鍵詞：脊髓損傷、性健康、性評估

前　言

當醫療發達疾病的治療與控制有相當的成就後，病人的健康問題也從危急生存的生理問題發展到社會功能性的問題，因此性健康照護隨著社會的進步成爲臨床重要的照護需求，近年臨床護理人員也漸漸重視相關的議題。馬與駱 [1] 的研究指出病人在住院中大部份沒有性需要，但一半以上的病人希望醫療人員能主動與他們討論有關性方面的問題，蔡等人 [2] 研究指出護理人員執行性衛教時最需要協助及最大的問題是缺乏足夠或適當的性衛教教材。「性」是不容易敞開討論的問題，它需要建立在信任的機制 [3, 4]，病人的信任來自對醫療人員專業肯定及其接納的態度，因此護理人員應先做好性知識、性照護技巧準備。

脊髓損傷直接影響男性病患性慾、勃起、射精、高潮及生育功能 [5, 6]，而性功能又間接造成損傷者低自尊、低情緒、兩性關係互動困難、不當的性感覺 [7, 8]。不同文化對性的需求不同，Ide 與 Fugl-Meyer[9] 對瑞士及日本脊髓損傷者生活滿意度調查，發現瑞士損傷者的生活滿意度高於日本損傷者，瑞士損傷者認爲性的重要性高於日本損傷者，並提出從事性活動及有伴侶的生活滿意度較高。要提升生活品質，專家提出鼓勵損傷者從事性活動 [10]，復健醫療是要提升失能者的生活品質，因此護理人員不能忽略脊髓損傷者的性復健護理。

Herson 等人 [11] 提到復健護理人員的性照護障礙來自知識不足，對提供失能者相關的性資訊沒有自信，且害怕提供錯誤的訊息。執行護理過程的第一步要收集健康資料，資料的收集前應對病人的想法及其健康要有整體的了解，才能建立信任的關係及有效能的收集工作。本研究目的在探討男性脊髓損傷後的性健康狀況，包括個人生理、性經驗、兩性關係及性資訊來源等，以提供護理人員執行性健康照護時的基礎概念。

材料與方法

本研究採訪談記錄回顧性描述性研究。回顧期間從

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2001 年 9 月 1 日至 2005 年 9 月 30 日止，樣本爲 18 份男性脊髓損傷患者性護理評估記錄，最初個案選取是依臨床醫療人員的觀察，取得患者的同意後填寫結構式問卷調查，然後再依問卷討論完成性護理評估記錄。研究者先依文獻自擬結構式內容，經復健醫師及護理同仁審閱建議，再由脊髓損傷者試填並接受訪談；結構式內容共做 3 度修改，取消避孕題，增加損傷前狀況及兩性關係調查，並將內容做明確分類，包括人口學、個人生理、性經驗、兩性關係及性資訊來源五項。每位受評者皆被告知可以不願意回答，或有權免填討論的題項，或直接表示“不想回答”。訪談時間約一小時，確認受評者意見的正確性及深度的完整性，最後依護理過程寫成記錄，本研究依記錄內容做分析討論。

結　果

受評者平均年齡：39.3 歲，最年輕爲 24 歲，最年長爲 70 歲，以 30-39 歲居多有 10 人（56%），已婚 11 人（61%），離婚 2 人（11%），未婚 5 人（28%）。受評者皆有性經驗，性功能及性反應皆正常，無伴侶 4 人（22%），1 人已婚無夫妻之實，以性交易滿足性需求。失能狀況見表 1。由護理人員轉介評估者 17 人（94%），由醫師轉介 1 人（6%），其中由護理長轉介者 12 人（67%），所有的轉介者皆已婚。受評者中 5 人（28%）主動提出性評估需求者，13 人（72%）由護理人員詢問表示願意接受性評估。大部份的受評者其性功能皆有影響，無勃起功能共 5 位，1 位頸髓，3 位胸髓，1 位腰髓，其中 4 位爲不完全損傷，其狀況見表 2，有精液者 4 人中有 3 人爲遺精及滴精，並主訴精液品質變差。受評者損傷後開始思考性問題及接受性諮商的時間如表 3。

兩性關係評估中受評者損傷前對性生活表示愉悅者有 12 人（67%），損傷後其中 6 人（50%）無性關係，3 人（25%）轉平淡，3 人（25%）保持愉悅，其中 1 位第 5、6 頸髓不完全損傷，勃起正常日常生活依賴度爲 25%，另 2 位皆爲第 12 胸髓損傷，1 位爲不完全損傷勃起硬度不足，1 位是完全損傷，有勃起障礙，日常生活依賴度皆爲 50%。性慾與性滿意評估採用李克特等量表（5-point Liker Scale）計分，接受性慾評估者有 14 人，

表 1　受評者損傷後失能狀況（N=18）

項　　目	人數	%
日常生活依賴度		
完全依賴	4	22
75%	1	5
50%	8	44
25%	3	17
完全獨立	2	11
損傷部位		
頸髓	8	44
胸髓	7	39
腰髓	1	6
馬尾束	2	11
損傷程度		
完全損傷	7	39
不完全損傷	9	50
馬尾束	2	11

表 2　受評者損傷後性功能狀況（N=18）

項　　目	人數	%
勃起功能		
有	13	72
無	5	28
有勃起者硬度		
還好	5	38
不足	7	54
不清楚	1	8
精液		
有	4	22
無	9	50
不清楚	5	28

表 3　受評者損傷後開始思考性問題及接受諮詢的時間（N=18）

項　　目	人數	%
開始思考性問題時間		
損傷後1個月	3	17
損傷後3個月	6	33
損傷後6個月	4	22
損傷後1年	1	6
從未	1	6
沒做答	3	17
受傷後接受諮商時間		
3個月以內	4	22
3-6個月	6	33
6個月至1年	1	6
1年以上	7	39

損傷前平均分數爲 4.3，回答不清楚者 1 人，損傷後平均分數爲 2.4，回答不清楚有 4 人（29%）；接受性滿意評估者有 12 人，損傷前平均分數爲 4.3，回答不清楚有 2 人，損傷後平均分數爲 1.5，回答不清楚有 6 人（50%）。

性資訊來源與接受性教育受評者皆爲 15 人，損傷前 8 人（58%）的性教育與性資訊來自學校公民課、影片及報張章雜誌。損傷後有 9 人（60%）提出無性資訊來源，4 人（27%）來自醫療人員，來自其他脊髓患患者 1 人（7%），有關性問題求教的對象，11 人（73%）不曾向任何人求教，向護理人員及復健科醫師各 2 人佔（13%），向其他科醫師有 1 人（7%）。

討論與結論

性健康是成年男性脊髓損傷者不分年齡所關注的問題，而壯年期更明顯，對於青壯年男性的損傷者，臨床健康照護者應更加關注其性健康問題。有性經驗的男性損傷者對性問題更加注意，脊髓損傷不論受傷程度對男性的性功能影響至巨，但有三分之一的男性損傷者對其功能狀況仍感到不清楚；性功能障礙進而影響到性慾、性滿意度及性關係，損傷後的性慾與性滿意度皆呈下降現象；脊髓損傷後有一半的人中斷其兩性生活，有性生活保持愉悅關係者與損傷程度及勃起功能無絕對的關係。由轉介者皆爲已婚得知已婚的醫療人員較能重視患者的性健康問題；受評者三分之一的人是主動提問，其他由臨床健康照護者主動關懷後才表示願意接受性諮詢；受評者表示有性問題但有 73% 未向任何人求教，可見性問題仍是一保守難以啓齒的問題，性教育貧乏不足仍需努力；受評者 50% 的人損傷後三個月即開始思考性問題，而近一半的人在半年後才開始接受性諮詢，因此臨床健康照護者對男性損傷者的性健康照護應採積極的態度，由研究得知建議臨床人員宜在損傷後滿 2 個月即可提供男性脊髓損傷相關性照護活動。本研究限制於個案來自轉介者主動觀察及個案自己提出需求，並無先預設收案標準，及訪談記錄由研究者以電腦文書建制，偶在複制格式中不堪覆蓋消失，因而個案數多有遺漏，另外訪談記錄由研究者以詮釋後書寫，故本研究只對結構性結果做分析，未採質性內容分析，訪談中個案表示很少或選擇性與伴侶討論損傷後的性問題，因此並未進一步評估性伴侶態度是否影響損傷者的性健康狀態。未來研究可對男性脊髓損傷者的性健康狀態做更多的評估，以了解他們的性健康問題做爲臨床性健康照護介入的依據，且脊髓損傷者的性健康評估表在臨床可再推廣評值其適用性。

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A Preliminary Study of Sexuality Statue for Male Patients with Spinal Cord Injury

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Background and purpose: Sexual-related health care is going to be a critical issue in clinical nursing services. This study aims to describe the male patients' sexual-related health condition after spinal cord injury.

Methods: A retrospective design was adopted in this study, which started from Sep 1st, 2001 to Sep 30th, 2005. The data was collected from 18 reports of sexual consultation of male patients with spinal cord injury.

Results: The results indicated that (1) participants' sexual-related health condition was not related to the ages and the severity of the illness; (2) 72% of the participants had the impotence problems; moreover, 60% of them encountered with hardness difficulty and half of them were absence of ejaculation; (3) the levels of sexual desire, sexual satisfaction and sexual relationships were all affected; (4) half of participants obtained sexual-related information from school, movie, and magazines before they were injured; nearly 60% of the victims voiced that resources of sexual-related information. (5) 50% participants concerned about sexual problems after three months of the injury; but 45% participants accessed to sexual consultation after 6 months of the injury.

Discussion: The clinical health care professionals need to be more proactive about the sexual-related health condition of these patients with spinal cord injury, and are suggested to provide the relevant nursing care to the male patients with spinal cord injury after two months of injury.

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Key words: spinal cord injury, sexual-related health condition, sexuality assessment.

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腹腔鏡卵巢濾泡燒灼打孔術治療多囊性卵巢症候群患者六年半的臨床經驗分析

汪文生

目的： 評價腹腔鏡卵巢濾泡燒灼打孔術對排卵藥物產生耐藥性（clomiphene resistant）的多囊性卵巢症候群（polycystic ovary syndrome, PCOS）患者所產生的療效。

對象、方法： 以回溯性研究收集 2001 年 3 月至 2007 年 8 月在本院婦產科不孕症門診就醫的多囊性卵巢症候群患者，確定診斷為 PCOS 是其不孕的主要原因，並且對排卵刺激藥物反應不良之不孕症患者共 20 例，接受腹腔鏡卵巢濾泡燒灼打孔術（laparoscopic ovarian drilling with diathermy, LOD）的治療並評估治療後的懷孕率、流產率、以及再次懷孕的機率。

結果： 懷孕率達 60%（12/20）其中自然受孕的有 50%（6/12），生下健康寶寶的機率 91.7%（11/12），流產率 8.3%（1/12），LOD 術後第一次懷孕後的 12 位婦女當中有 8 位想再次懷孕，其中有 5 位自然受孕成功並順利生產，想再次自然懷孕的機率有 62.5%（5/8）。

結論： 對藥物治療無效以及想要再次或多次懷孕的多囊性卵巢症候群患者，腹腔鏡卵巢濾泡燒灼打孔術是優先考慮的選擇。

（童綜合醫誌 2007; 1: 33-38）

關鍵詞： 燒灼；腹腔鏡；卵巢打孔術；多囊性卵巢症候群

前　言

在不孕症的門診當中有一群（5-10%）被診斷爲多囊性卵巢症候群（PCOS）的婦女，這群婦女可能出現肥胖、月經不調、月經量少、不孕症、或者男性化特徵 [1]。傳統上我們要治療這類病人，首先應該設法鼓勵其減低體重，有時候就可以恢復排卵；如果不行，接下去第一線藥物就是排卵藥 clomiphene，但是使用 clomiphene 只有 75~80% 可以成功的排卵，且其中只有 40% 患者得以受孕；雖然胰島素活化物如 metformin 對於胰島素的敏感度有加強的作用，單獨或合併排卵藥使用，亦可治療 PCOS 患者，很不幸的有些患者對於 metformin 並不具有反應，而且無法適用在不同的種族。因此再下一線藥物多半就是性腺刺激素（gonadotropins），包括「濾泡刺激素」Follicle stimulating hormone（FSH）與「人類停經後性腺刺激素」Human menopausal gonadotropin（HMG）等，但仍有部分患者使用以上藥物治療皆無效；如何對排卵藥物產生耐藥性的多囊性卵巢症候群患者進行治療，一直是困擾婦產科醫生的一項棘手難題。腹腔鏡下雙側卵巢濾泡燒灼打孔術因其療效好，併發症少，似乎可以取代併發症較多的卵巢楔形切除（Ovarian wedge resection）手術，爲難治性多囊性卵巢症候群患者在接受試管嬰兒治療之前提供了一條安全有效的途徑。

來就診的不孕患者其目的是想懷孕並獲得健康的寶寶，然而被診斷爲 PCOS 的婦女，自然排卵非常的困難，即使使用藥物也不見很好的成效；不但不易受孕，而且一旦受孕也容易流產。況且藥物治療僅單一療程，無法持續到下一個月經週期，必須不斷重覆給與，一旦停藥，

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情況又復原；即使這胎幸運成功生子，想生下一胎仍需再次給藥治療。因此本研究不僅評估當 PCOS 的婦女對排卵藥物產生耐受性，無有效排卵時，轉接受腹腔鏡卵巢濾泡燒灼打孔術治療後的懷孕率，亦評估能生下健康寶寶的機率，以及再次懷孕的機會。

材料與方法

收案的病例從 2001 年 3 月至 2007 年 8 月在本院婦產科不孕症門診就醫的多囊性卵巢症候群患者，這些患者臨床上的特徵是肥胖、月經不順、排卵異常、超音波顯示多囊性卵巢，其不孕因素已經過內分泌檢查，排除輸卵管因素，免疫因素，男性因素等，確定診斷爲 PCOS 是其不孕的主要原因，並且經過對排卵刺激藥物治療反應不良及暫且不考慮做試管嬰兒技術之不孕症患者共 20 例（表 1），經討論後願意接受腹腔鏡卵巢濾泡燒灼打孔術的治療。患者年齡 21~35 歲，平均 28.75±3.95 歲，不孕年限 1~12 年，平均約爲 3±2.53 年。70%（14/20）爲原發性不孕症。30%（6/20）曾懷孕過，其中 3 位生過一位孩子另外 3 位有流產病史。

表 1 PCOS 不孕患者的基本資料

編號.	年齡	孕次	不孕時間(年)
1	26	G0P0	2
2	30	G0P0	1
3	35	G0P0	5
4	35	G1P1	5
5	30	G0P0	2
6	30	G0P0	1
7	21	G0P0	1
8	25	G0P0	1
9	25	G2P0A2	2
10	24	G0P0	3
11	33	G0P0	4
12	23	G0P0	1
13	33	G0P0	12
14	29	G0P0	2
15	28	G1P1	2
16	32	G1P1	4
17	26	G0P0	2
18	30	G1P0A1	2
19	29	G2P0A2	5
20	31	G0P0	3
平均	28.75±3.95		3±2.53

腹腔鏡卵巢濾泡燒灼打孔術方法：在月經乾淨 7 天內進行，行氣管插管全身麻醉，于肚臍正中直切口約 5mm，氣腹針穿刺後 CO_2 形成氣腹，壓力維持在 12mmHg，腹壁套管針穿刺成功後置入內視鏡；另在下腹兩側各穿刺 5mm 套管針後，患者右側助手執無齒夾幫忙固定卵巢，患者左側主刀醫師操作使用單級電燒針（圖 1）；根據 1997 年加拿大多倫多大學的 Tulandi *et al* [2] 的建議，先將單級電燒輸出功率設定在 40 瓦特，對兩側卵巢表面透亮的濾泡進行電燒持續 2~4 秒各鑽 10-12 個孔，每孔深約 4-6 mm，直徑 3-4 mm，10-12 個洞之總能量爲 800-1200 焦耳，處理完後使用生理食鹽水沖洗卵巢表面（圖 2）。患者可以門診手術或是住院觀察一天，出院後門診持續追蹤治療。

結　果

PCOS 不孕患者接受腹腔鏡下雙側卵巢濾泡燒灼打孔術後追蹤 6 年半之結果（表 2），術後懷孕的有 12 位，佔 60%（12/20），其中不使用任何藥物能自然受孕的有 6 位，佔 50%（6/12），12 位懷孕當中編號 19 懷孕早期流產，流產率爲 8.3%（1/12），生下健康寶寶的機率 91.7%（11/12）。除編號 9 因再婚因素，從手術至懷孕的時間較長，花了 2 年 5 個月外，其餘從手術後 1 個月到 1 年 7 個月不等的時間順利懷孕。11 位生產當中有 3 位早產，早產原因編號 1 與 2 是因爲嚴重度子癇前症，編號 10 是因爲雙胞胎，早產率爲 27.3%（3/11）；非自然受孕的 6 位患者當中有 1 位僅服用排卵藥及注射排卵針（FSH），1 位除服用排卵藥及注射排卵針（hMG）外再加上胰島素活化物 metformin，有 3 位接受人工受孕，而編號 10 患者手術後就私自轉向其他醫院接受試管嬰兒治療。目前術後第一次懷孕已生的 12 位嬰兒（2 位雙胞胎）都健康 無發現任何先天的疾病或缺陷。11 位生產後的婦女，有 2 位已不想再生育，2 位產後沒多久，這 4 位除外，其餘 8 位有 5 位再次自然受孕成功，想再次懷孕的機率有 62.5%（5/8），5 位已出生的嬰兒亦相當的健康。編號 8 的患者，已順利生完 LOD 術後的第 3 胎並接受輸卵管結紮。

討　論

PCOS 的治療最早採用 1964 年 Stein Leventhal[3] 的腹式卵巢楔形切除術。切除了一部份的卵巢並且保留卵

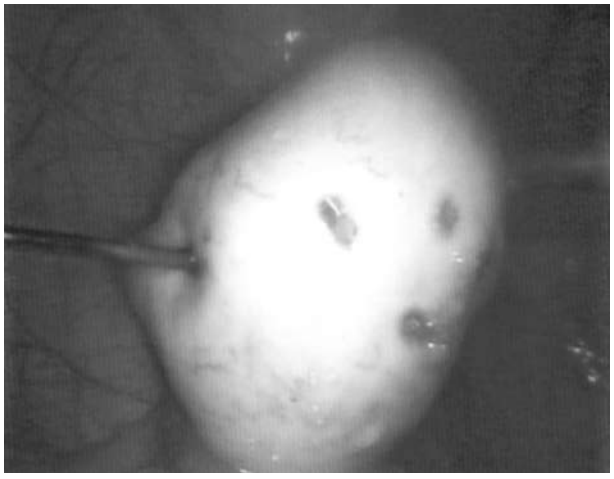

圖 1　單級電燒正在多囊性卵巢表面透亮的濾泡處進行電燒

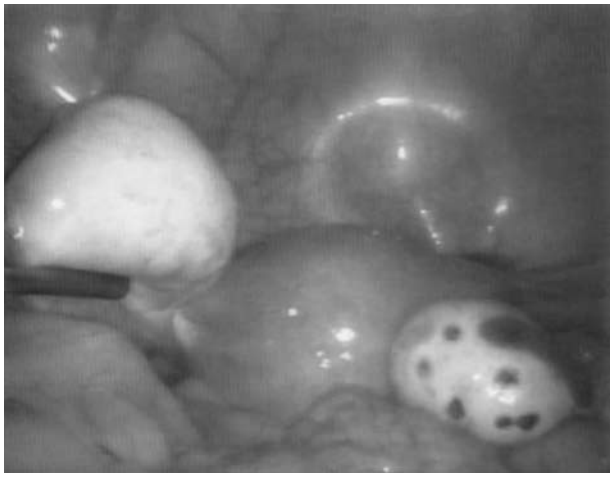

圖 2　兩側卵巢明顯增大，卵巢表面有多發性小濾泡，燒灼後的右側卵巢

表 2　PCOS 不孕患者接受腹腔鏡下雙側卵巢濾泡燒灼打孔術後追蹤 6 年半之結果

編號	術後妊娠	第一胎受孕方式	術後至第一次受孕時間（天）	再次妊娠	第二胎受孕方式	胎兒週數		胎兒體重(公克)	
						第一胎	第二胎	第一胎	第二胎
1	是	自然	190	是	自然	33	37	1900	3340
2	是	CC+IUI	580	否		32		1850	
3	否								
4	否								
5	是	自然	93	是	自然	38	38	3534	3618
6	是	自然	111	是	自然	40	39	3538	3352
7	是	CC+FSH+IUI	440	是	自然	38	38	2946	2756
8	是	Metformin+CC+hMG	87	是	自然	39	38	3489	4250
9	是	自然*	897	否		41	39	3200	
10	是	IVF(twins)	426	節育		35		2054&2046	
11	否								
12	否								
13	否								
14	否								
15	是	自然	300	節育		38		2880	
16	否								
17	否								
18	是	自然	54	產後		40		3678	
19	是	CC+FSH	282	否		流產			
20	是	CC+FSH+IUI	34	產後		38		2300	
百分比	60% (12/20)	自然50%(6/12)		自然 62.5% (5/8)					

CC=Clomiphene citrate; IVF=in vetro fertilization; IUI=intrauterine insemination; *=再婚；# =計算至受孕時的最後一次月經日期；

表 3 術後懷孕成功與懷孕失敗的年齡與不孕時間分析

	懷孕成功（12/20）	懷孕失敗（8/20）	P值
1平均年齡	27.42 ± 3.15	30.75 ± 4.37	0.062
2.不孕時間（年）	2.08 ± 1.16	4.38 ± 3.42	0.044
*P*值<0.05有統計學上的意義			

巢的其餘部分，接著將它們一起縫合回去，這將導致 LH 分泌和雄性素荷爾蒙的分泌減少。然而術後會發生嚴重的沾黏，目前已很少採用。1984 年，Gjonnaess[4] 首先報導腹腔鏡單極電凝電灼卵巢治療 PCOS，主要是將卵巢表面上的小濾泡給灼燒掉， 透過灼燒這些小濾泡，使雄性素的生產減少。因發生沾粘的機率較小、創傷小、恢復快，逐漸替代了腹式卵巢楔形切除術。上述兩種手術方式，其作用機轉主要爲術後卵巢產生雄激素的組織減少，卵巢卵泡液得到引流，使血中睪固酮（T）濃度下降 [5]。而雄激素濃度的下降使血中雌酮（E_2）濃度下降，因此減少對腦下垂體分泌 LH 的正回饋作用，使腦下垂體對 GnRH 刺激的反應性降低，減少 LH 的分泌。E_2 濃度的下降，消除了 FSH 分泌的負反饋抑制。同時，E_2 濃度下降刺激 FSH 的分泌。T 值的下降，減少了對卵子發育的抑制作用。幾種相互作用的結果，阻斷了高 LH、高 T 血症的惡性循環，使 PCOS 患者的內分泌紊亂得到糾正，誘發排卵。

在影響手術結果的因素中，不孕發生的時間短較時間長的患者在接受 LOD 手術成效好 [6, 7]，卻與患者的年齡大小無統計上的意義 [7]。本研究以 *t* 檢定比較以上兩因素是否影響手術的成敗，結果顯示（表 3）術後懷孕成功患者平均不孕的時間 2.08±1.16 年比懷孕失敗患者平均不孕的時間 4.38±3.42 年有統計上的意義（*P* 值 <0.05），術後懷孕成功患者平均年齡 27.42±3.15 歲比懷孕失敗患者平均年齡 30.75±4.37 歲無統計上的意義，與文獻上相同。至於術前孕次是否有影響，查無文獻闡述。

腹腔鏡卵巢濾泡燒灼打孔術的療效如何？ Naether 等人在 1994 年 [8] 文獻中統計 145 位 PCOS 患者在 LOD 治療後，於往後六年的追蹤中懷孕率可達 70%，同時文章內容也指出 LOD 手術所顯現之效果有長期的效應。根據 1997 年加拿大多倫多大學的 Tulandi *et al* [2] 的排卵率可達到 88.2%，一年的懷孕率 70%，其中 76% 是自然受孕。本研究懷孕率達 60%，其中自然受孕爲 50%，雖有些差距，本人認爲或許是統計的個案數量不夠大的關係。1998 年 Gjonnaess[9] 的文獻認爲 LOD 術後能維持功效長達 20 年之久，本研究結果顯示想再次懷孕的機率有 62.5%，而且全都是自然懷孕，不需再使用任何藥物的幫助，其中一位還懷了術後第 3 胎。另外 LOD 可減少流產率，可從術前的 54% 降至術後的 17%（Amer et al, 2002）[10]，本研究的流產率僅 8.3%，效果相當顯著。至於 LOD 手術後是否需積極治療，則因人而定。

Doneski 與 Adashi 在 1995 年 [11] 提出當 PCOS 的患者對排卵藥物產生耐藥性時，LOD 手術治療應擺在第二位，而不是採用性腺刺激素的排卵針劑。有六項理由：⑴ LOD 手術不會增加多胞胎的機率，⑵降低卵巢過度刺激的風險，⑶不需特別加強照護，⑷有較低的流產率，⑸單一次的治療能產生多次的排卵週期，⑹所花的費用相對較低。因此對排卵藥物產生耐藥性以及想要再次或多次懷孕的 PCOS 患者，腹腔鏡卵巢濾泡燒灼打孔術在嚐試試管嬰兒之前是可優先考慮的選擇。

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Laparoscopic Ovarian Drilling with Diathermy in the Treatment of Patients with Polycystic ovary syndrome: six and half years clinical experienced analysis

Wen-Sheng Wang

Objective: To evaluate the effect of laparoscopic ovarian drilling with diathermy on the pregnancy out come of polycystic ovary syndrome in clomiphene-resistant infertile women.

Methods: Twenty anovulatory infertility women with polycystic ovary syndrome who visited the Tungs' Taichung MetroHarbor Hospital between March 2001 and August 2007. All patients had been unresponsive to treatment with clomiphene citrate. Laparoscopic ovarian drilling with diathermy was performed. The rates of pregnancy, abortion and once again pregnancy were calculated.

Results: Pregnancy rate 60%(12/20) and 6 of the 12 pregnancy women (50%) were spontaneous pregnancy, take baby home rate 91.7%(11/12), abortion rate 8.3% (1/12), sequential pregnancy rate 62.5%(5/8).

Conclusion: Laparoscopic ovarian drilling with diathermy can be the good choice for anti-oestrogen resistant anovulatory infertility in women with the polycystic ovary syndrome who want to conception twice or more.

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Key words: diathermy; laparoscopy; ovarian drilling; polycystic ovary syndrome

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The Most Common Hematuria in Children: Clinical Evaluation and Differential Diagnosis

Kai-Li Wang[1], Chiung-Hui Chen[2], Yuan-Hao Chen[1*]

Hematuria occurs in approximately 1.5% of children. It is important in evaluating the patient who has hematuria to make sure that a positive dipstick test accompanied by RBCs on the microscopic examination. Hematuria is defined by several parameters, the most common is 5 cells per high-power field in a urinary sediment. Although the differential diagnosis for hematuria is extensive, the most important differentiating feature is the presence or absence of proteinuria. Those who have significant proteinuria deserve a rapid evaluation and early referral to a nephrologist. Those who do not have proteinuria should be followed and a step-wise evaluation performed. Most patients who have asymptomatic microscopic hematuria do not have clinically significant glomerular pathology, but in those patients who had poor prognostic indicators including significant proteinuria, hypertension or changes on biopsy, long-term evaluation by a nephrologist is needed.
(Tungs' Med J 2007; 1: 39-49)

Key words: hematuria, dysmorphic RBC, eumorphic RBC

INTRODUCTION

Blood found in the urine is concerned to patients, parents and pediatricians. Patients with hematuria presenting with a number of disorders of urinary system which can be the causes of progressive renal disease. The pediatrician must plan an evaluation that will identify the treatable or progressive condition quickly enough to allow for treatment. The purpose of this review is to outline a plan for the evaluation of the patient who has hematuria, list a differential diagnosis, and describe the major causes of isolated microscopic hematuria, including clinical course and treatment.

Definition

It is important to remember that a number of substances can cause the red urine (Table l), and the test for identifying hematuria requires confirmation. Blood in the urine is identified most readily by a dipstick, using a peroxidase that reacts with hemoglobin; greater than l+ is considered positive. The dipstick tests for hemoglobin, not red blood cells. Any chemical that reacts with the peroxidase will produce a positive test. Therefore, all positive dipstick screens must be confirmed by the microscopic examination of the urine to confirm the present of RBCs. Two significant causes of a positive dipstick and a negative microscopic examination are free hemoglobin from hemolysis and myoglobinuria from rhabdomyolysis. Certain drugs and toxins also can cause red urine, although these are heme-negative. The presence of more than 5 RBCs per high-power field of urine sediment from a centrifuged urine sample should be considered abnormal.[1]

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Table 1. Red Urine without RBC

Heme-positive
Hemoglobinuria
Myoglobinuria
Heme-negative
Metabolites
Porphyrin
Melanin
Urate
Drugs
Salicylates
Phenophthalein
Pyridium
Iron sorbitol
Nitrofurantoin
Methyldopa
Metronidazole
Chloroquine
Others
Beets
Blackberries
Food coloring

Table 2. Hematuria: History

Associated Complaints
Concurrent illnesses
Edema
Rash
Arthralgia
Dysuria
Back or flank pain
Trauma
Diet
Past Medical History
Sickle cell
Cystic kidney disease
Systemic lupus erythematosus
Congenital heart disease
Malignancy
Medications
Family History
Hematuria
Deafness
Renal failure
Cystic kidney disease
Sickle cell
Nephrolithiasis
Systemic lupus erythematosus
Social History
Abuse

Prevalence

The prevalence of isolated microscopic hematuria in children and adolescents is approximately 1.5%. populations from a number of different regions have been studied.[2] In the US, the largest study was in Galveston schoolchildren.[3] There also have been large series of schoolchildren studied in Japan and Finland. All of these have had similar results. The majority of these patients were asymptomatic and never developed significant renal disease. Japanese children whose hematuria was isolated were more likely to undergo a diagnostic renal biopsy, with IgA nephrolpathy being a relatively common finding. All of these studies excluded children who were ill, specifically those who had acute glomerulonephritis or significant proteinuria.

History Taking

After the child has hematuria is identified, a careful history should be taken. The history often will provide the physician an enough information to make a presumptive diagnosis and will narrow the differential diagnosis to allow for a targeted evaluation.

Some of the important points are listed in Table 2. Timing of the episodes is important. For example, persistent microscopic hematuria with recurrent episodes of gross hematuria, particularly if the episodes are associated with viral illness, suggests IgA nephropathy. The same pattern associated with dysuria, back pain, or flank pain suggests urinary tract infection, hypercalciuria, or nephrolithiasis. Children or their parents frequently will not recognize that discolored urine is caused by the presence of blood. Hematuria from a glomerular lesion may cause the urine to appear to be brown, or tea-colored. Thus the color of the urine needs to be described accurately.

Associated illness need to be sought. The association with concurrent upper respiratory tract infections often is seen with IgA nephropathy, mentioned previously. Poststreptococcal glomerulonephritis generally is preceded by a sore throat or impetiginous lesion by 7 to 21 days. However, it is important to remember that patients and parents often cannot remember or did not recognize the infection that was associated the disease process. Abdominal pain can suggest urinary tract infection or Henoch-Schoenlein purpura (HSP). Rashes, such as the petechial/purpuric rash of the lower extremity seen in HSP or the malar rash of systemic lupus erythematosus (SLE), may lead to the diagnosis. The presence of edema or hypertension suggests clinically significant glomerulonephritis and requires a more aggressive evaluation. Voiding difficulties or dysuria are associated with urologic causes of hematuria. Causes of transient hematuria include significant trauma to the back, bladder, or genitalia and vigorous exercise, particularly running. Gross he-

maturia associated with minimal trauma can include the presence of an abnormal kidney, especially one having a ureteropelvic junction stenosis. Finally, if the patient has heme-positive urine without RBCs, causes for hemolysis or rhabdomyolysis need to be sought.

The medical history also can offer a number of clues. Certain medical conditions commonly are associated with hematuria; these need to be ruled out, including sickle cell disease or trait, cystic kidney disease, and SLE. Congenital heart disease may be associated with hematuria for various reasons. Patients who have septal defects or valvular lesion or have cardiac surgery can be predisposed to endocarditis, which can lead to an immune complex glomerulonephritis. The aggressive use of furosemide can cause hypercalciuria and subsequent hematuria. The treatment of malignancy with chemotherapy, radiation, or surgery often is associated with a variety of nephrologic or urologic complications, including hematuria.

Hematuria occurring during the neonatal period can be caused by thrombosis of the renal vein or artery, particularly if an umbilical catheter was used. Infants requiring prolonged furosemide therapy for bronchopulmonary dysplasia can develop hematuria associated with nephrocalcinosis and/or nephrolithiasis. Interstitial nephritis may be associated with microscopic hematuria, as well as pyuria and proteinuria. Causes of interstitial nephritis include a number of medications such as nonsteroid pain relievers.

Several conditions[4] that cause hematuria are genetic in origin and include Alport syndrome, thin basement membrane, polycystic kidney disease, and sickle cell disease or trait. Nephrolithiasis and IgA nephropathy also have familial association. A history of hearing loss and renal failure in males in a family that has relatively unaffected or late affected females suggests Alport syndrome. Thin basement membrane disease is a common cause of familial hematuria in children. Sometimes affected family members are not aware that they have microscopic hematuria. Therefore, screening the urine of all available first-degree relatives is important in the evaluation of isolated microscopic hematuria. Child abuse needs to be considered in the differential diagnosis if the physical examination is suspicious or the cause of hematuria is thought to be trauma.

Physical Examination

The physical examination of the patient who has hematuria is not specific, but certain points deserve comment (Table 3). The presence of an increased blood pressure should alert the pediatrician to the possibility of a more severe underlying condition that warrants a more aggressive evaluation. A fundoscopic examination of the retina, looking for signs of hypertension, should be performed. Edema frequently is observed first as a swelling around the eyes. Patients who have congenital renal disease also may have malformed ears. The presence of erythema or exudates of the throat may suggest streptococcal disease, although poststreptococcal nephritis occurs 7 to 21 days after the infection. The chest should be examined for signs of fluid overload, such as rales, murmur, gallops, or an increased or displaced cardiac impulse. Evidence of serositis such as rubs can help identify SLE or uremia. A careful examination of the abdomen for masses is critical for the identification of malignancy and polycystic kidney disease. Ascites suggests nephrosis. Renal bruits often are audible in renal vascular disease. Costovertebral angle or flank tenderness often is seen in infection and distention from ureteral obstruction. The genitalia should be ex-

Table 3. Hematuria: Physical Examination

HEENT
Periorbital edema
Malformation of ears
Erythema/exudates of pharynx
Chest
Rales
Rubs
Gallops
Murmurs
Strength and placement of precordial impulse
Abdomen
Masses
Ascites
Bruits
Trauma
Back
Flank tenderness
Genitourinary system
Meatal stenosis
Discharge
Trauma
Extremities
Edema
Arthritis
Skin
Rashes
Petechiae
Purpura

amined for trauma, discharge, and meatal stenosis, all of which can cause hematuria. Extremities should be examined for arthritis and edema. Rashes need to be evaluated, particularly for signs of old skin infections, malar rash, purpura, and petechiae.

Laboratory Evaluation

The laboratory evaluation (Fig. 1) of the patient who has hematuria should be based on the differential diagnosis that is suggested by the history and urinaysis. Patients who present symptomatically with hypertension, edema, proteinuria, or oliguria are likely to have significant glomerular pathology and require rapid and complete evaluation. This evaluation includes a complete blood count (CBC), electrolytes, blood urea nitrogen (BUN) and creatinine concentration, measurement of C3, C4, and fluorescent antinuclear antibody (FANA), hepatitis B serology, a sickle cell preparation or hemoglobin electrophoresis, quantitative protein excretion, and creatinine clearance. Renal ultrasonography with a Doppler flow study of the renal vessels also may provide valuable diagnostic information. The approach should identify all severe forms of glomerulonephritis rapidly. However, the patient who otherwise is healthy and is found on a routine screening to have isolated hematuria needs to be evaluated in a step-wise manner directed by the history, physical examination, urinaysis, and clinical

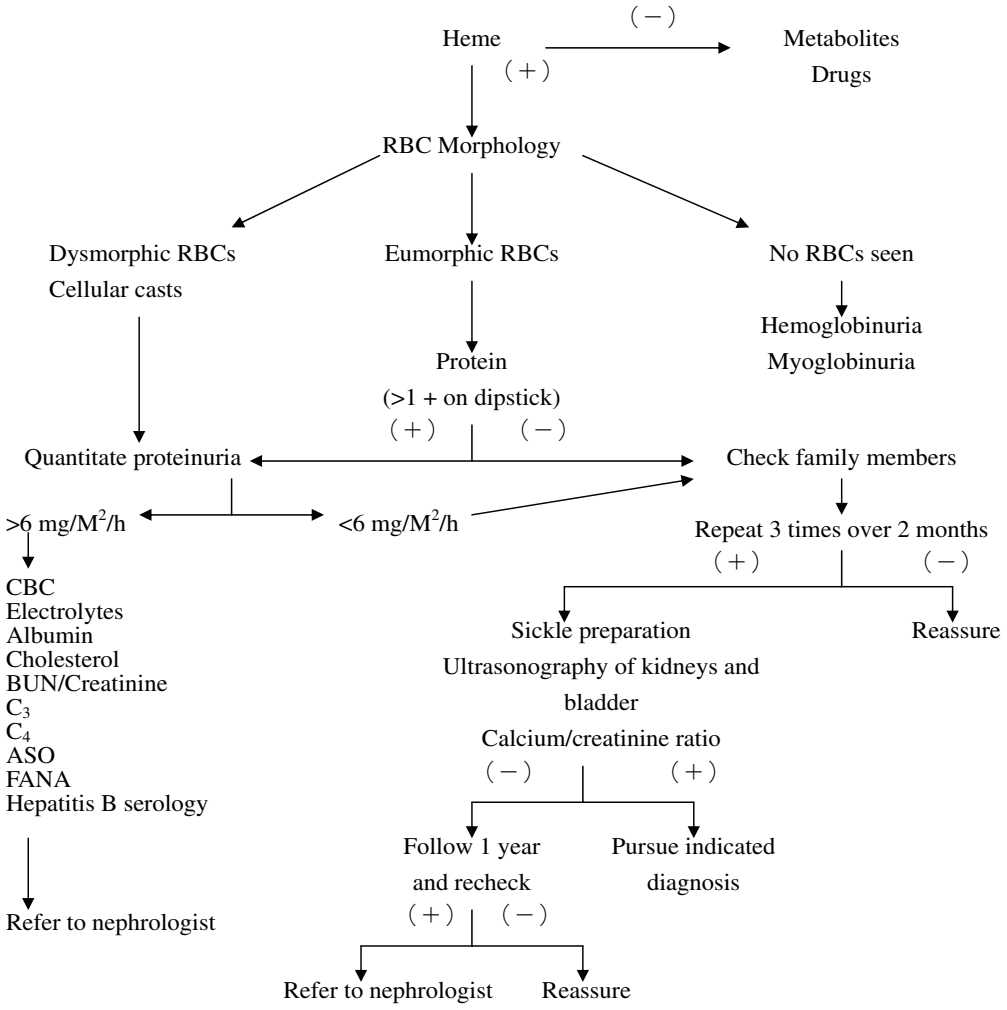

Fig. 1 Evaluation of hematuria in children.

course.

The only laboratory examination required for every patient is a urinalysis. This should include not only a screening dipstick for blood but also for protein. In addition, the urine should be examined microscopically to ensure the presence of RBCs and determine their morphology. The site of the blood loss can occur anywhere along the urinary tract, from the urethral meatus to the glomerulus. By inspecting the size and contour of the RBCs carefully (Fig 2), it often is possible to make this distinction with simple microscopic examination of the urine. The presence of cells that appear small and misshapen with outlines that show burrs and blebs (dysmorphic cells) indicates glomerular bleeding.[5] Cells that originated somewhere other than the glomerulus either can be normal in shape and size (eumorphic) or slightly small with a serrated edge (crenated). These differences are best seen with a phase-contrast microscope but can be appreciated with routine microscopy.[6] However, it is important to realized that although the presence of dysmorphic RBCs in the urine suggests a glomerular lesion, their absence does not rule one out. The presence of protein of 1 + or greater in the absence of gross hematuria suggests a renal cause of the hematuria. The presence of RBC cast indicates a glomerular lesion and the need for a more thorough evaluation looking for causes of glomerulonephritis.

White blood cells suggest inflammation either from nephritis when found alone or infection when associated with bacteria. Routine urine culture is not necessary in the evaluation of hematuria unless an infection is suggested by the presence of white blood cells and bacteria. If the culture is positive, the infection should be treated. The urinalysis should be repeated after the infection is treated adequately to determine whether the microscopic hematuria is persistent.

The following test always should be performed in patients whose hematuria is isolated, unless the etiology of hematuria is clear. A urinalysis should be performed in as many family members as possible. It is easy to do, inexpensive, and can be extremely helpful in identifying familial causes of hematuria. A spot urine calcium/creatinine ratio can rule out hypercalciuria as an etiology rapidly (<0.21 being normal).[7] Ultrasonography of kidneys and bladder should be used to rule out polycystic kidney disease, tumor, stones, and obstruction as causes of the hematuria.

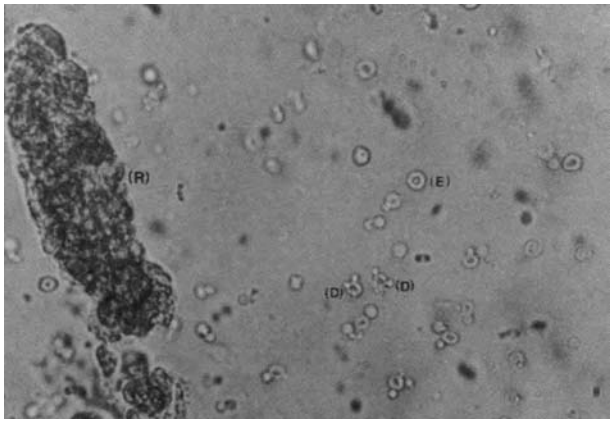

Fig. 2 Urine from a patient of poststreptococcal glomerulonephritis.
(D)=dysmorphic RBC, (E)=eumorphic RBC, (R)= red blood cell cast.

The remainder of the evaluation of the patient should be based on the history and results of the previous tests. The patient who has proteinuria demonstrated by dipstick should have the protein excretion quantitated.[8] The first test to perform is a simple timed (12- or 24-hour) urine collection for quantitation of urinary protein. Less than 4 mg/M^2 per hour is normal, and greater than 40 mg/M^2 per hour is proteinuria in the nephrotic range. [9] Values between 4 and 40 mg/M^2 per hour also are abnormal and require investigation.If the patient does have significant proteinuria, the presence of glomerular disease is likely.

The laboratory evaluation of glomerular disease that is consists of a CBC, electrolytes, BUN, and creatinine with a creatinine clearance performed. This will establish to what degree, if any, renal function is impaired. African-American children need to be screened early for sickle cell disease or trait. Additional laboratory work to be considered in most patients includes a serum C3 concentration and ASO titer or streptozyme. A depressed C3 level and positive streptococcal serology are consistent with the diagnosis of poststreptococcal glomerulonephritis. The C3 concentration often will be depressed in SLE membranoproliferactive glomerulonephritis and the nephritis of chronic bacteremia. If the diagnosis of SLE is a possibility then a FANA and additional SLE serology may be per formed. Hepatit B serology will identify those patients who have glomulonephritis secondary to hepatitis B infection. If the patient has significant edema and/or nephrotic range protein-

uria, serum albumin and cholesterol levels should be performed to document the presence of the nephrotic syndrome. If the patient who has hematuria and significant proteinuria does not have laboratory or clinical findings consistent with poststreptococcal glomeru-lonephritis,[10] a referral should be made to a nephrologist and for possible renal biopsy.

If the patient does not have dysmorphic RBCs and / or proteinuria, causes other than glomerulonephritis should be evaluated progressively. Two or more urinalyses should be done over next 2 months to confirm that the hematuria is persistent.

Cytoscopy is not indicated for the initial investigation of asymptomatic isolated microscopic hematuria. This invasive procedure virtually never provides a definitive diagnosis and adds considerably to the cost of the evaluation. In children, isolated microscopic hematuria is a rare presentation for tumor of the bladder and kidney. Rhabdomyosarcoma of the bladder usually is associated with voiding difficulties and gross hematuria. Wilm tumor only rarely presents with microscopic hematuria; this tumor should be detected by the renal ultrasonographic examination in the initial evaluation. Cystoscopy should be used only when clearly indicated.

A final point to be made is that children whose microscopic hematuria is isolated, without proteinuria, gross hematuria, or a positive family history, very seldom have significant pathology. Studies done in both the US and Finland have shown that approximately 20% of children whose findings are minimal have clinically significant changes on renal biopsy.[11] Furthermore, most of those having renal parenchymal abnormalities do not require specific therapy. The diagnosis is made for prognosis, planning for long-term follow-up, and/or genetic counseling.

Differential Diagnosis

There are many causes of hematuria in children. Figure 3 offers an approach to the differential diagnosis based on the urinary RBC morphology, presence or absence of proteinuria, history of the hematuria, and family history. There remainder of this discussion will focus on the most common causes of hematuria in children.

HYPERCALCIURIA

Hypercalciuria is present in approximately 5% of healthy Caucasian children in the US and is the most frequent cause of isolated hematuria in this group. Approximately 30% of children whose hematuria is isolated will be hypercalciuric. Hypercalcuria, as well as microscopic hematuria, less common in African-American children. Furthermore, there are regional differences, with well-documented areas of endemic urolithiasis within North America, so-called "stone-belts." The reason for the increased incidence is unclear. Urinary calcium excretion in these area is higher than in other areas of the country. It should be remembered that hypercalciuria also associated with episodic gross hematuria in the absence of demonstrable renal stones.[12]

Hypercalciuria is found in certain conditions that increase calcium excretion, such as hyperparathyroidism, immobilization, vitamin D intoxication, and the use of furosemide. However, most children whose hematuria is isolated will have idiopathic hypercalciuria. The mechanism of the hematuria is unclear，but may involve irritation to the renal tubules by calcium-containing crystals. The significance of hypercalciuria with hematuria in the well child is the apparent risk for later development of renal stones. The percentage of children who have hypercalciuria who later develop urolithiasis is unclear. However, more than two thirds of children who have urolithiasis will have associated hypercalciuria.

Hypercalciuria is screened for with a spot urinary calcium/creatinine ratio. A ratio of 0.21 or greater is indicative of hypercalciuria.[13] The diagnosis can be confirmed by collecting a timed (either 12- or 24-hour) urine for calcium excretion. An excretory rate of greater than 4 mg/kg per day is abnormal. Patients who have hypercalciuria and hematuria should have a renal ultras-onographic examination to rule out nephrolithiasis or nephrocalcinosis. Treatment consists of decreasing the concentration of calcium in the urine. Dietary measures include increasing fluid intake and salt restriction.

Patients should increase water intake, especially during warm weather, because dehydration increases the concentration of calcium in the urine. After these measures are taken, the patient should be reevaluated. Hydrochlorothiazide therapy will decrease urinary calcium excretion and is useful therapy in patients who are forming stones. A short course of hydrochlorothiazide often will result in the disappearance of microscopic hematuria and, thus, can be used as

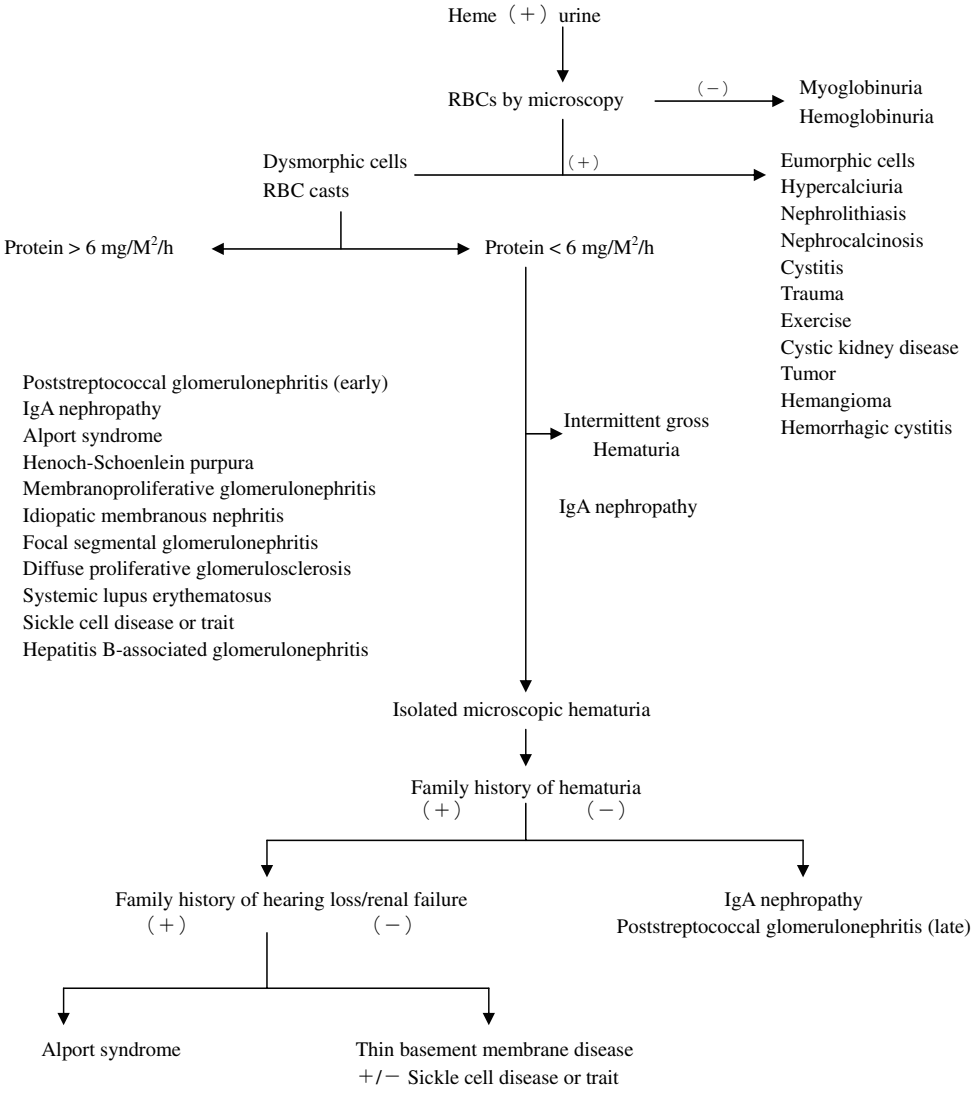

Fig. 3 Differential diagnosis of hematuria.

a diagnostic test. The long-term use of hydrochlorothiazide in a child whose hematuria is isolated and in whom there is no previous history of nephrolithiasis cannot be defended.[14] All patients who have nephrolithiasis should have a complete metabolic evaluation.

POSTSTREPTOCOCCAL ACUTE GLOMERULONEPHRITIS (PSAGN)

PSAGN typically begins 7 to 21 days after a group A beta-hemolytic streptococcal infection of either the throat or skin. Appropriate antibiotic treatment for the infection will not prevent the development of nephritis. The children typically present with tea-colored urine, edema, and hypertension. Often, the initial infection forgotten and the initial phase of the illness is missed. If the symptoms of nephritis are subclinical or missed, the patient may present later having isolated hematuria.

The serum C3 concentration is significantly depressed at onset in 90% of patients who have PSAGN and returns to normal within 6 weeks of onset.[15] The ASO titer usually is increased initially. Occasionally, a patient will have a normal ASO titer but a positive

streptozym, especially if the initiating infection was impetigo.

Microscopic hematuria usually resolves 6 to 12 months after onset, but may be present for as long as 2 years. Most children who have PSAGN will have normal renal function after recovery, with no long-term effects. If the patient fits the typical pattern, a more extensive evaluation, including a renal biopsy, is warranted. For instance, failure of the C3 concentration to normalize by 6 weeks indicates a more chronic condition such as membranoproliferative glomerulonephritis. The treatment in the acute phase of PSAGN consists of salt restriction, diuretics, and antihypertensives for some patients. Rarely, severely affected patients will require dialysis; most of these patients, however, also can be expected to have a good long-term outcome.[16]

IgA NEPHROPATHY (BERGER DISEASE)

IgA nephropathy is the most common type of chronic glomerulonephritis in children and adults of European or Asian descent. It occurs less frequently in African-Americans and is rare in Africans. IgA nephropathy also is a very common cause of hematuria in children. Approximately 15% of children having isolated hematuria that persists for more than a year will have IgA nephropathy. The typical presentation for IgA nephropathy is for gross hematuria to occur during a viral respiratory or gastrointestinal illness. Widespread routine screening of schoolchildren's urine for blood and protein commonly is done in Japan.[17] In that setting, isolated microscopic hematuria or microscopic hematuria with proteinuria constitutes the most typical presentation. A child diagnosed after presentation with microscopic hematuria often will have had a subsequent episode of gross hematuria during an intercurrent illness. Children who have IgA nephropathy usually are normotensive and rarely are edematous at presentation. Serum IgA concentration may be elevated, or circulating IgA-containing immune complexes may be present. However, there is no laboratory test diagnostic for the condition. Diagnosis depends on the typical demonstration on renal biopsy of IgA in the mesangium of the glomerulus.

IgA glomerulonephritis originally was thought to be a benign condition, particularly for children. This false assumption was based on small studies having brief follow-up. With longer follow-up, it has become apparent that up to 50% of patients identified during adulthood and approximately 25% of those identified during childhood eventually will progress to chronic renal insufficiency. Poor prognostic indicators definitely include significant glomerular injury on initial biopsy (lesions of focal segmental sclerosis), persistence of moderate-to-heavy degrees of proteinuria, and the development of hypertension. Some investigators have suggested that males and African-Americans are at risk for progressive disease. Up to 20% of children who have biopsy-proven IgA nephropathy eventually will have an apparent remission in which the urinalysis becomes completely normal. In patients who have minimal disease (ie, absence of hypertension, mildly affected or normal glomeruli, and low grades of proteinuria) no therapy is indicated. However, in those patients who have significant proteinuria, hypertension, or significant changes on biopsy, some nephrologists would attempt treatment.[18] An alternate-day prednisone regimen has been the most widely used treatment.

A large multicenter controlled study has been planned to determine the efficacy of such treatment. Due to the potential for progressive disease, patients who have IgA nephropathy should be followed by a nephrologist.

ALPORT HEREDITARY NEPHRITIS

The prevalence of Alport syndrome in patients who have isolated hematuria is approximately 15%. A presumptive diagnosis often made with a careful family history. A patient may present having isolated hematuria found during a screening examination. Episodes of gross hematuria may occur in association with intercurrent viral illness, and some patients even may have semicontinuous gross hematuria. A family history often will reveal one or more male individuals having nerve deafness and progression to end stage renal disease.[19]

Alport syndrome usually is inherited as an X-linked dominant trait. Recent studies indicate that the primary defect in Alport syndrome is an alteration in type IV collagen.[20] Further studies have implicated a mutation in the COL4A5 collagen gene. This mutation causes an abnormality in alpha 3 type IV collagen chain, a component of type IV collagen. This abnormality prevents the correct assembly of

alpha 3 and alpha 5 chains. Therefore, the type IV collagen is abnormal and the basement membrane is disrupted. Males have a strong tendency to be affected more severely than females. Affcted males have a large number of RBCs in their urine, even in the earliest stages of the disease. Affected females usually have less hematuria and may have clinical and renal biopsy findings suggestive of thin basement membrane disease. If Alport syndrome is suspected，hearing should be examined and urinalyses performed on other family members. Genetic markers for the disease are being developed and should become available in the future.[21] The diagnosis is confirmed by renal biopsy, which also may help in assessing prognosis. Again, because of the potential for progression to end stage renal disease, patients who have Alport syndrome should be followed by a nephrologist.

THIN BASEMENT MEMBRANE DISEASE

Thin basement membrane disease is a hereditary condition in which the lamina densa decreased in width to less than l000 to 2000 angstroms. The normal lamina dense width varies with age from 1100 angstroms in a child to 2700 angstroms in an adult. This condition probably is what was previously called benign familial hematuria.[22] Affecced individuals present having microscopic hematuria with dysmorphic RBCs without having significant proteinuria. There often are other family members who have microscopic hematuria.[23] The only way to confirm the diagnosis is with renal biopsy. Hoever, renal biopsy may not be necessary if the child does not have significant proteinuria, and other close relatives have been biopsied. There is no treatment or intervention other than monitoring urinalysis and renal function at 1- to 2- year intervals.

Summary

Hematuria may be gross or microscopic. Gross hematuria may originate from the kidney, in which case it is generally brown or cola and may contain red blood cell (RBC) casts or from the lower urinary tract; in which case the urine is red to pink and may contain clots. Microscopic hematuria is most commonly discovered at periodic health examinations, by dipstick or by microscopic examination of the urine sediment. Children with hematuria should be evaluated carefully including history taking, physical examination and laboratory evaluations because of the increased likelihood of finding hypertension and renal failure. If no etiology can be found in the presence of persistent hematuria, a nephrologist should be consulted; often, a renal biopsy is done.

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兒童常見血尿：臨床評估及其鑑別診斷

王凱立[1]　陳炯暉[2]　陳遠浩[1*]

兒童血尿之發生率約 1.5%。而以每視野鏡檢下，可見到有 5 個以上之紅血球時，則為定義血尿之最常取用的方法。當一位兒童之尿液試紙呈陽性血尿反應時，而其尿液顯微鏡檢中也同時存在者一些紅血球時，這對一個被稱之為“血尿病童”之確定診斷是很重要的。對一位血尿病童而言，需作鑑別診斷之疾病雖不少，但仍以有或無蛋白尿之存在代表腎疾病預後之重要指標。當一位血尿病童同時存在有明顯之蛋白尿時，則需一快捷之評估並及早轉介給小兒腎臟醫師診治。反之，當血尿病童並無蛋白尿之同時存在時，則可依血尿檢測之相關步驟來進行評估，並做追蹤檢查。雖然大部分無臨床症狀之顯微血尿病童，在其腎絲球病理檢查中，往往並沒有明顯之異常發現，但這些病童一旦出現有明顯之蛋白尿，高血壓、甚至於切片檢查之異常，則須腎臟專科醫師之長期追蹤評估。
（童綜合醫誌 2007; 1: 39-49）

關鍵詞：血尿、變形紅血球、原形紅血球

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Hepatoid Carcinoma of the Ovary: Immunohistochemical Finding of One Case and Literature Review

Tang-Yi Tsao[1]*, Kim-Seng Law[2]

Primary ovarian hepatoid carcinomas (POHC) are extremely rare. Especially rare are those with phenotypic properties of hepatocellular carcinoma (HCC) and an absence of clinical evidence of hepatic tumor. We report a case of POHC with a characteristically microscopic, immunophenotypic and in the absence of a liver mass. It is extremely difficult to differentiate POHC from metastatic HCC using any kinds of ancillary studies, with the exception of clinical identification of a hepatic tumor. A 49-year-old woman who had abnormal vaginal bleeding and lower abdominal pain was found to have a right ovarian mass on pelvic examination and subsquent computed tomography. She had high serum levels of alpha fetoprotein (AFP) and CA125. Histologically, the tumor resembled hepatocellular carcinoma by architectural and cytologic features. Immunohistochemically tumor cells were immunoreactive for AFP, anti-hepatocyte antibody, thyroid transcription facotr –1(TTF-1), CD34, alpha-1 antichemotrypsin (A-1 ACT), glypican-3, pancytokeratin (AE1/AE3), CK8, CK18, CK19 and negative for , CK5, CK7, Ck10, Ck20, and CEA.
(Tungs' Med J 2007; 1: 50-57)

Key words: Primary ovarian hepatoid carcinomas (POHC), anti-hepatocyte antibody, thyroid transcription facotr –1(TTF-1), alpha-1 antichemotrypsin, (A-1 ACT), glypican-3.

INTRODUCTION

Tumors that resembling hepatocellular carcinoma, but arising outside the liver are known as hepatoid carcinomas. Production of alpha fetoprotein (AFP) is an important characteristic of the tumor[1]. Despite immunohistologic findings favoring a surface epithelial origin, primary ovarian hepatoid carcinoma(POHC) is now categorized as a "miscellaneous primary tumor" in a recent World Health Organization classification[2,3]. Thus far, only 16 cases have been reported in the literature,[5]. We present here a case of POHC with clinical and pathologic perspectives and compare it with primay HCC from the standpoint of immunohistochemical findings.

CASE REPORT

A 49-year-old woman presented with abnormal vaginal bleeding and lower abdominal pain was admitted to Gynecology Clinic of Tungs' Taichung MetroHarbor Hospital. A right ovarian mass was detected on pelvic examination and subsequent computed tomography. The computed tomography disclosed a huge 9.0 × 8.6 × 8.0 cm right ovary tumor, with eccentric peripheral contrast enhancement and right iliac lymphadenopathies. The ultrasound and computed tomography showed one small hypoattenuated area in S8 of the liver and measuring about 0.59 cm, which was distinctly different from the liver cyst. Laboratory tests showed a high serum AFP level

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(9,968 ng/mL, normal range, 0–8.1) and high serum CA125 level (106 U/mL, normal range, 0–35). Under the impression of hepatoid yolk sac tumor during frozen section examination of the right ovary, subsequent, stging laparostomy with bilateral iliac lymphadnectomy, omentectomy, and washing cytology was performed.Grossly, the right ovarian tumor showed mixed solid and cystic appearance, measuring 10.5 × 10 × 5.5 cm in size, and convered by complete fibrous capsule. On cut surface, the solid component was reddish yellow and soft in consistence. The cystic component was hemorrhage (Fig. 1).

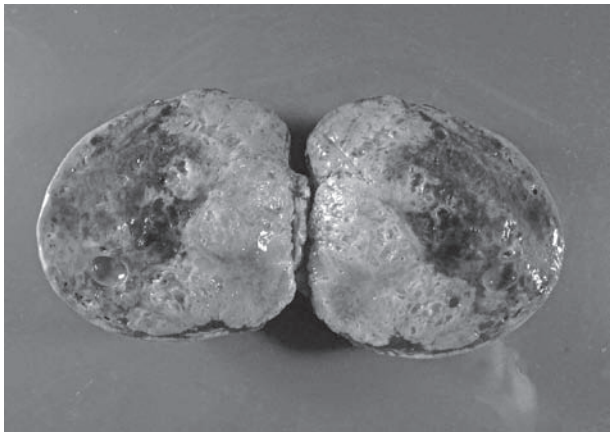

Fig. 1 Grossly, the right ovarian tumor showed mixed solid and cystic appearance, measuring 10.5 × 10 × 5.5 cm, and convered by complete fibrous capsule. On cut surface, the solid component was reddish yellow and soft in consistence. The cystic component was hemorrhage.

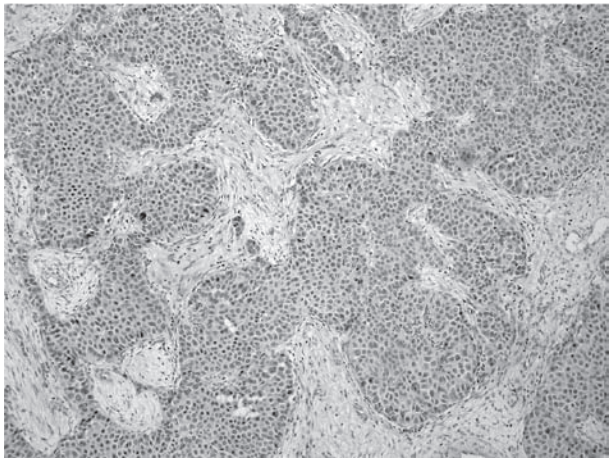

Fig. 2 Microscopically, tumor was composed of multiple nodules(trabeculated sinusoidal patterns) of neoplastic cells that contained abundant amounts of eosinophilic cytoplasm with prominent central nucleoli and had distinct cell borders resembling hepatocellular carcinoma. The cells were generally uniform in size and shape with minimal pleomorphism. Mitotic activity was generally inconspicuous(Hematoxylin-eosin stain, X 100)

Microscopically, tumor was composed of multiple nodules(trabeculated sinusoidal patterns) of neoplastic cells that contained abundant amounts of eosinophilic cytoplasm with prominent central nucleoli and had distinct cell borders resembling hepatocellular carcinoma. The cells were generally uniform in size and shape with minimal pleomorphism. Mitotic activity was generally inconspicuous(Fig. 2). There were no other associated germ cell components within ovarian tumors on examination of all slides. The dissected bilateral iliac lymph node tissue and

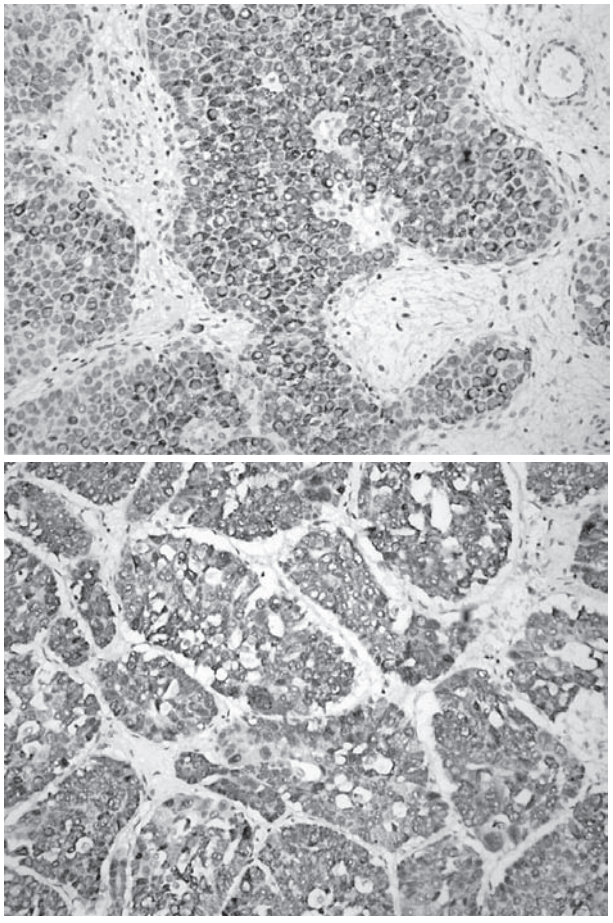

Fig. 3 Both POHC(upper) and hepatocellular carcinoma(lower) tumor cells were diffusely cytoplasmic pattern immunoreactive for antihepatoyte antibody stain (PAP stain, X 200)

washing cytology were all negative finding. Selective sections were stained with periodic acid–schiff (PAS) with and without diastase digestion histochemically. Rare PAS-positive and diastase-resistant PAS-positive hyaline globules were observed. PAS-positive and diastase-sensitive glycogen was also demonstrated.

Immunohistochemical staining was performed by avidin–biotin complex peroxidase complex method according the manufacturer's instructions. Our comparison of immunohistochemical findings between primary hepatoid carcinoma of ovary and hepatocellular carcinoma used the antibodies against antihepatocyte antibody, AFP, thyroid transcription facotr –1(TTF-1), alpha-1 antichymotrypsin(A-1 ACT), glypican-3, CD34, carcinoembryonic antigens(CEA), pancytokeratin (AE1/AE3), and cytokeratin 5, 7, 8, 10, 18, 19, and 20. Both the POHC and hepatocellular carcinoma tumor cells were diffusely cytoplasmic pattern immunoreactive for antihepatocyte antibody, AFP, thyroid transcription facotr –1(TTF-1)(Fig. 3, 4 and 5; Table 1). Both POHC and hepatocellular carcinoma tumor cells were strongly sinusoid pattern immunoreactive for CD34 stain. The HCC revealed continously sinusoid pattern, but the POHC was in-

Table 1. Comparison of immunohistochemical findings between primary hepatoid carcinoma of ovary and hepatocellular carcinoma.

Markers \ Tumors	(Hepatoid Ca of ovary)	(HCC)
1.hepeatocyte	+++ (cytoplasmic pattern)	+++ (cytoplasmic pattern)
2.AFP	+++ (cytoplasmic pattern)	+++ (cytoplasmic pattern)
3.TTF-1	+++ (cytoplasmic pattern)	+++ (cytoplasmic pattern)
4.CD34	+++ (intermittently sinusoid pattern)	+++ (continously sinusoid pattern)
5alpha-1 antichemotrypsin	++, focal (cytoplasmic pattern)	++, focal (cytoplasmic pattern)
6.CEA	-	+++, focal (cananicular pattern)
7.CK(AE1/AE3)	+++, focal (cytoplasmic and membranous pattern)-	-
8.CK5	-	-
9.CK7	-	-
10.CK10	-	-
11.CK20	-	-
12.CK19	++, focal (cytoplasmic pattern)	-
13.CK8	++ (cytoplasmic pattern)	-
14.CK18	+++ (cytoplasmic pattern)	+, focal (cananicular pattern)
15.Glypican-3	+++ (cytoplasmic pattern)	+++ (cananicular & focal cytoplasmic pattern)

Note: classifications of positive degree, +: slight, ++:moderate, +++:Marked

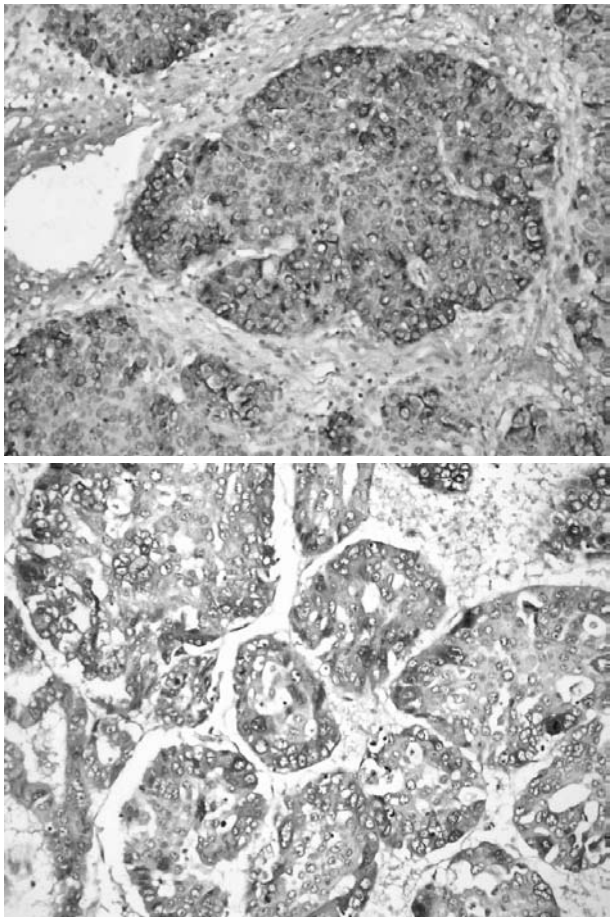

Fig. 4 Both POHC(upper) and hepatocellular carcinoma(lower) tumor cells were also diffusely cytoplasmic pattern immunoreactive for AFP stain (PAP stain, X 200)

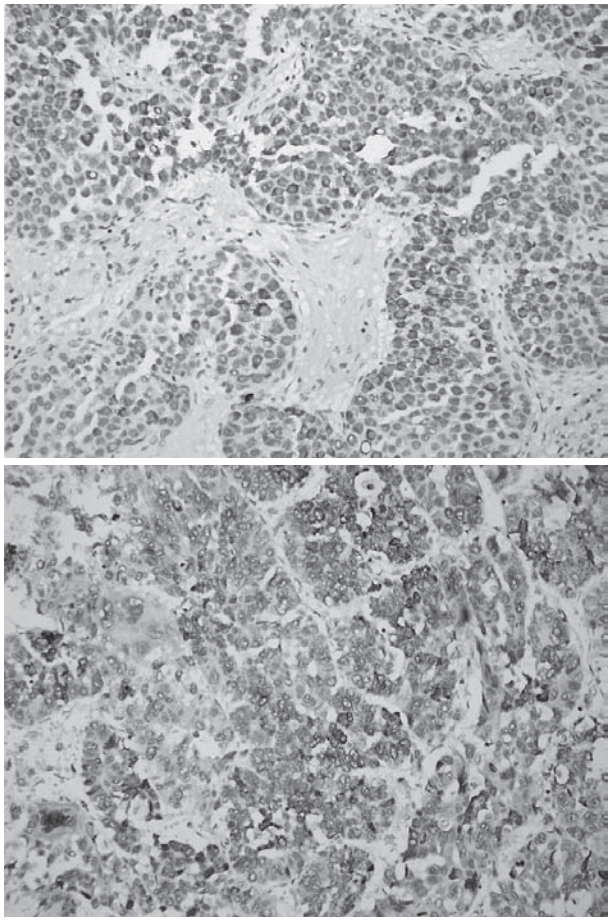

Fig. 5 Both POHC(upper) and hepatocellular carcinoma(lower) tumor cells were also diffusely cytoplasmic pattern immunoreactive for TTF-1 stain (PAP stain, X 200)

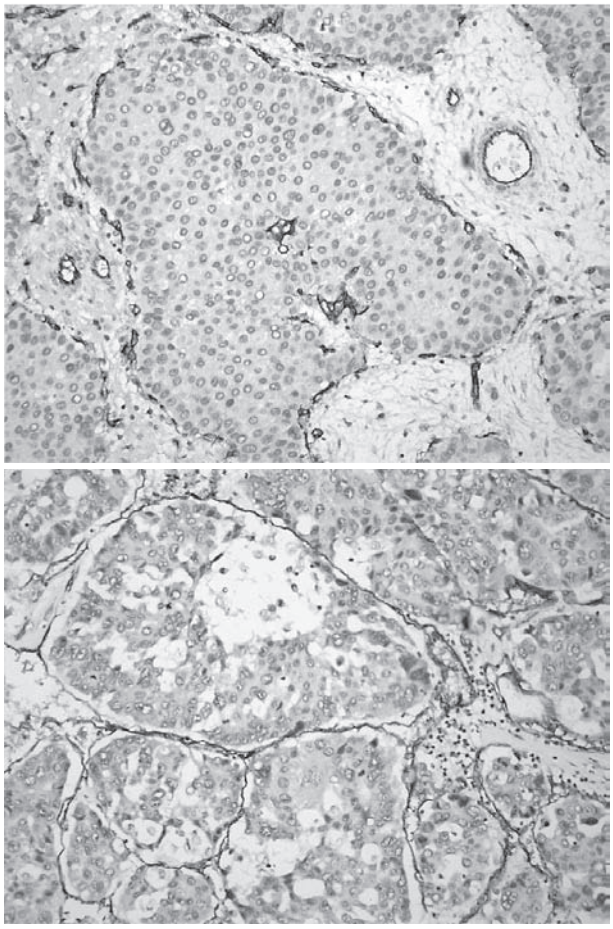

Fig. 6. Both POHC(upper) and hepatocellular carcinoma(lower) tumor cells were strongly sinusoid pattern immunoreactive for CD34 stain. The HCC revealed continously sinusoid pattern(lower) but the POHC was intermittently sinusoid pattern(upper) (PAP stain, X 200)

termittently sinusoid pattern(Fig. 6). Both the POHC and hepatocellular carcinoma tumor cells were focal cytoplasmic pattern immunoreactive for A-1 ACT(Table 1). The POHC tumor cells were diffusely cytoplasmic pattern immunoreactive for CK18 stain, but HCC tumor cells were focal cananicular pattern immunoreactive for CK18 stain (Fig 7). The POHC tumor cells were diffusely cytoplasmic and focal spotting pattern immunoreactive for glypican-3 stain but hepatocellular carcinoma tumor cells were focal cananicular(denoted by an arrow) and focal cytoplasmic pattern immunoreactive for Glypican-3 stain(Fig 8). The POHC was also focal cytoplasmic pattern positive for pancytokeratin (AE1/AE3), CK8 and CK19 (Fig 9), but the HCC was negative immunoreactive for pncytokeratin (AE1/AE3), CK8 and CK19 stains. The POHC was negative immunoreactive for CEA, but the HCC was focal cananicular pattern positive for CEA. The CK5, CK7, CK10 and CK20 were all negative in both POHC and HCC(Table 1).

The age and the histopathology of the present case were compatible with the current literature[1, 8-9]. High blood levels of CA125 and AFP had also supported the diagnosis. In our case, no tumor was found in the liver, and therefore, metastatic hepatoid carcinoma to the ovary was excluded. The tumor was diagnosed as “Primary ovarian hepatoid carcinoma(POHC),” stage IA. After surgery, the

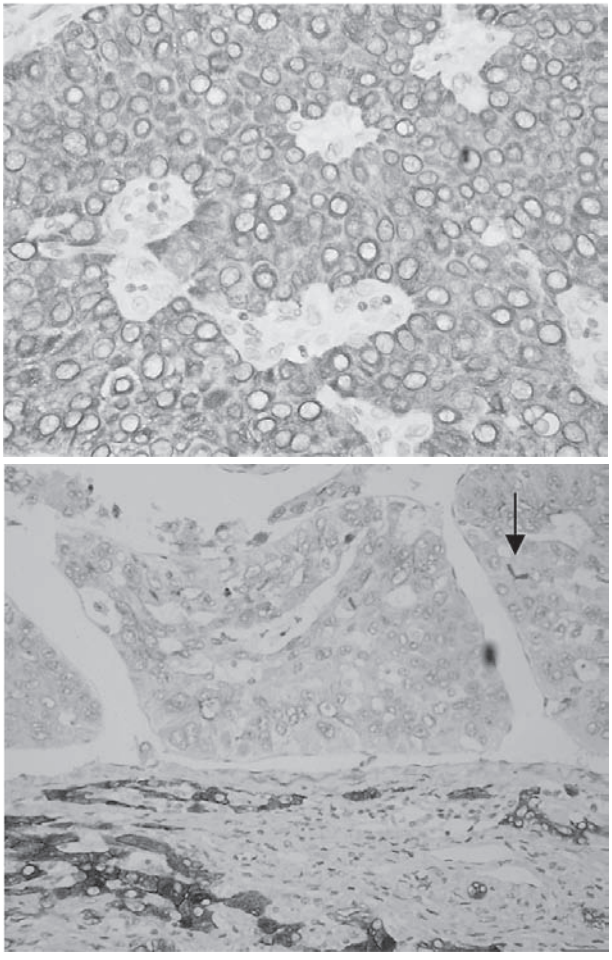

Fig. 7. POHC(upper) tumor cells were diffusely cytoplasmic pattern immunoreactive for CK18 stain but hepatocellular carcinoma(lower) tumor cells were focal cananicular pattern(arrow) immunoreactive for CK18 stain (PAP stain, X 200)

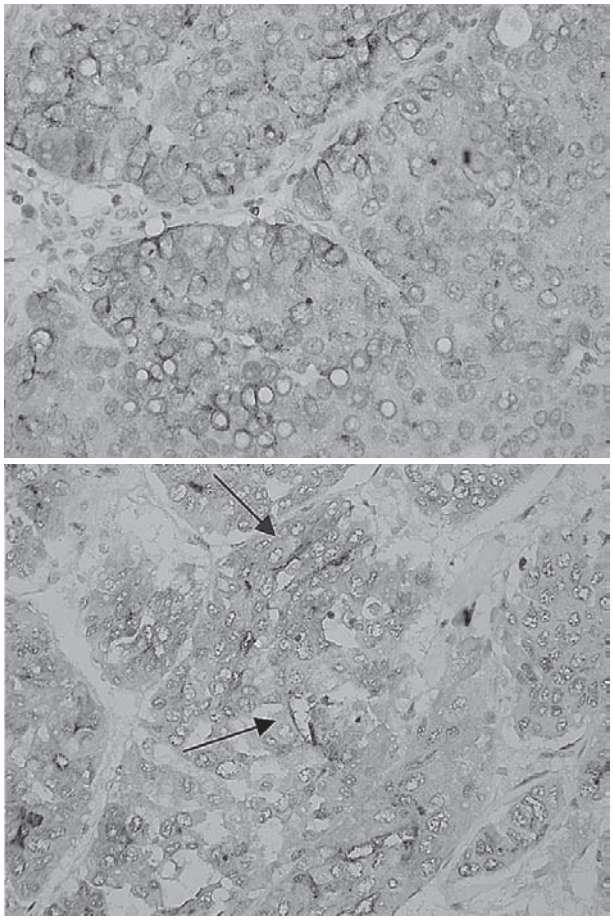

Fig. 8. POHC(upper) tumor cells were diffusely cytoplasmic pattern immunoreactive for glypican-3 stain but hepatocellular carcinoma(lower) tumor cells were focal cnanicular (arrow) and cytoplasmic pattern immunoreactive for Glypican-3 stain (PAP stain, X 400)

patient received combined chemotherapy with etoposide, paclipasel,and cisplatin. Serum CA125 and AFP level decreased to normal following the treatment procedure. The patient is alive at 5 years and 6 months following surgery and adjuvant chemotherapy.

DISCUSSION

Hepatoid carcinoma of the ovary has been introduced as a unique neoplasm in 1987 by Ishikura and Scully[1]. This tumor is an uncommon lesion; only 16 such cases were described elsewhere in the literatures[5]. The clinical features of these cases are shown in Table 2. POHC has been documented with phenotypic properties in common with HCC[1]. Except for the anatomic site, histopathologic and immunohistologic findings of POHC are similar to metastatic HCC. Despite studies of the immunoprofiles[1,6], the cell of origin in POHC remains uncertain. Tochigi et al.[6] proposed that POHC might arise from surface epithelial carcinomas on the basis of CK profiles (CK 8/18/19), presumably by a process of neometaplasia or transdifferentiation. In addition to immunoprofiles, a common association with other types of surface epithelial tumors suggests that the origin of this tumor is a common epithelial cell[4, 6-7].

The most clinical features of these cases are

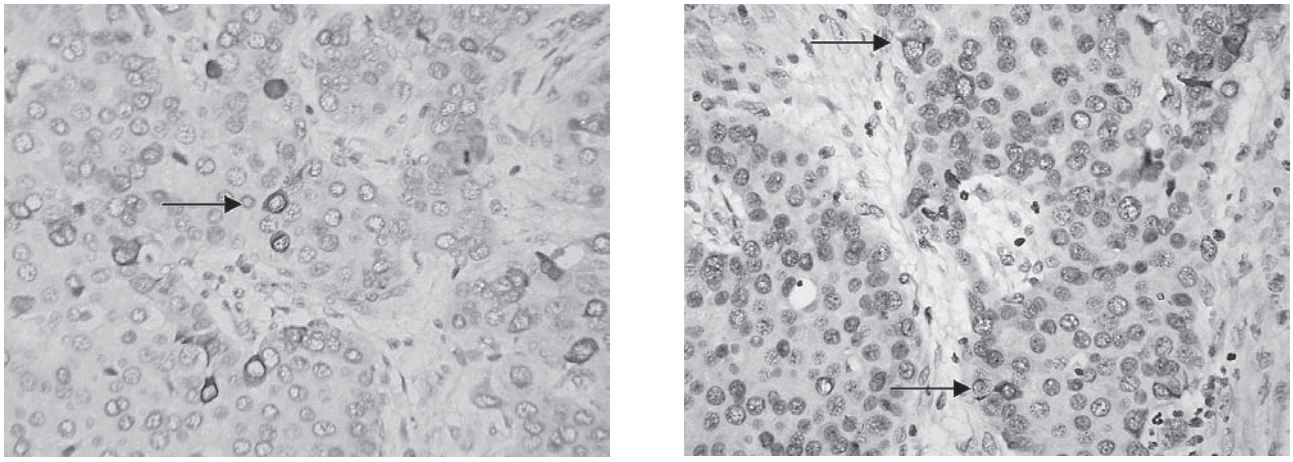

Fig. 9. POHCtumor cells were focal cytoplasmic pattern immunoreactive for CK(AE1/AE3)(left) and CK19(right) but hepatocellular carcinoma tumor cells were negative in both stains.(PAP stain, X 400)

Table 2. Clinical features of hepatoid carcinoma of the ovary

Case	Age (years)	Signs and symptoms	Site	Size(cm)	AFP (ng / mL)	CA125 (U / mL)	FIGO stage	Follow-up
1	42	Pelvic peritonitis	Bilateral	L: 6 × 5 R: 5 × 4	ND	ND	IIB	Died, 5 years
2	71	Abdominal distension	Left	20	ND	ND	III	Aliver,2 years
3	57	Abdominal distension, weight loss , bloating	Right	10.5 × 7.5 × 5.5	ND	ND	III	Died, 4 months
4	78	Abdominal distension, cramping	Left	ND	2420	ND	III	Died, 8 months
5	68	Abdominal pain, pelvic mass	Right	10 × 6 × 5	ND		III	Died, 10 months
6	64	Lower abdominal mass	Right	18 × 17 × 16	23,170	57.5	IA	Aliver,2 years
7	62	Lower abdominal pain	Right	8.2 × 7.8 × 6.4	2,450	ND	IA	Died, 13 months
8	72	Abdominal distension, dyspnea , lethargy	Bilateral	9.5 × 5.5	ND	802	III	Recurred, 6 months
9	52	ND	Bilateral	ND	2,500	Elevated	III	Recurred, 7months
10	61	Abdominal distension	Left	12 × 9	73,080	79.7	III	Died, 20 months
11	69	Vaginal bleeding, lower quadrant mass	Left	12	589.5	111	IA	ND
12	53	Ovarian mass	Left	10	257.522	Normal	IIB	Aliver, 13 months
13	76	Ovarian mass	Left	16	24,000	ND	IIB	Aliver, 4 years
14	64	Lower abdominal mass	Right	23	900	52.7	IIIC	Died, 5 years
15	36	Abdominal pain	Left	10 × 8 × 8	ND	888	IIIC	ND
16	63	Vaginal bleeding, lower abdominal pain	Right	16	457	84.59	IA	Aliver, 10 months
17	49	Vaginal bleeding, lower abdominal pain	Right	10.5 × 10 × 5.5	9968	106	IA	Aliver 5 years

ND = not determined
Case 17:present case.
Casse 1~16:Int J Gynecol Cancer 2006;16(4):1439–41.

lower abdominal pain and postmneopausal bleeding . A review of the literature reveals that hepatoid carcinoma of the ovary occurs almost exclusively in postmenopausal women is in contrast to hepatoid yolk sac tumor occurs in younger patients[4,5]. The present case is a 49-year-old premenopausal woman. Elevation of serum AFP and serum CA125 was seen in most cases in the literature (Table 2). Most of the hepatoid carcinoma cases were clinically advanced tumors. Four cases were presented as stage IA tumors like the present case (Table 2). Histopathologically, ovarian hepatoid carcinoma shows sheets, trabeculae, and cord of the cells with a moderate to large amount of eosinophilic cytoplasm and round to oval central nuclei resembling hepatocytes. Intracellular or extracellular PAS-positive, diastase-resistant hyaline globulin is always present. In this case, PAS-positive and diastase-resistant hyaline globulin was rarely seen. PAS-positive and diastase-sensitive glycogen could also be demonstrated. This is not consistent with the results reported elsewhere[4]. The most favorable finding for metastatic HCC under the microscopy alone has been regarded to be the presence of bile[6,8], except for a few studies to document the presence of bile pigments in POHC[1,4, 8]. However, we could not find bile pigments under the light microscopy in our case. Among immunoprofiles, we found that antihepatocyte antibody, and AFP were not useful to differentiate between ovarian tumors with hepatoid phenotypes, such as POHC and metastatic HCC[4,9]. In this study the antihepatocyte antibody, AFP, thyroid transcription facotr –1(TTF-1), alpha-1 antichymotrypsin (A-1 ACT) (both POHC and HCC all strongly cytoplasmic pattern positive), and CK5, CK7, CK10, CK20(both POHC and HCC all negative) were demonstrated to be not useful to differentiate between ovarian tumors with POHC and metastatic HCC(Table 1). In conslusion, with the exception of identification of a hepatic mass, we found that even ancillary tests were shown to be extremely difficult to differentiate POHC from HCC. In those immunohistochemical analyses, we noted that the positivity for pancytokeratin (AE1/AE3), CK8, CK18,CK19, glypican-3 and intermittently sinusoid pattern for CD34, along with negative for CEA may help to support a diagnosis of POHC.

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卵巢類肝細胞癌：免疫化學染色特徵—病例報告及文獻回顧

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原發性卵巢類肝細胞癌相當罕見，尤其是它與肝細胞癌有很多相同特徵。本文報告一例原發性卵巢類肝細胞癌具有典型的組織學與免疫染色特徵而且病人並無肝臟腫瘤，利用一般輔助的方法，很難鑑別原發性卵巢類肝細胞癌與卵巢轉移的肝細胞癌，除非在臨床上發現肝臟有腫瘤。本文報告一位 49 歲婦女主訴下腹疼痛及陰道出血，在理學檢查及電腦斷層掃瞄時發現右側卵巢有一腫瘤，同時血中 α- 胎兒蛋白及 CA125 上升。在組織學上，此腫瘤與肝細胞癌相似，在免疫染色上 α- 胎兒蛋白、抗肝細胞抗體（Anti- hepatocyte antibody）、TTF-1、CD34、α-1 ACT, AE1 ／ AE3, CK8, CK18, Ck19 以及 glypican-3 均呈陽性反應。

（童綜合醫誌 2007; 1: 50-57）

關鍵詞：原發性卵巢類肝細胞癌（POHC）、α- 胎兒蛋白（AFP）、甲狀腺轉譯因子 -1（TTF-1）、阿發 -1 抗胰凝乳蛋白酶（α-1 ACT），Glypican-3。

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* 通訊作者：曹唐義　童綜合醫院　病理部

新生兒尿液中高尿酸合併血尿一病例報告

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新生兒血尿之原因不少，但高尿酸之排泄所導致的新生兒血尿並不多見。本病例雖以紅色之尿布呈現，但在相關之檢測中，不僅有血尿之存在，且其尿液中尿酸排泄百分率（FEUA）可高達38%，約為成人（10%）之 3~4 倍之多，而證實為一高尿酸排泄合併血尿之新生兒病例。這種結果乃因於新生兒之腎小管功能之不足所導致。不過，這種新生兒高尿酸排泄情況只是短暫性的。隨著新生兒腎小管之不斷成熟發育，及其身體中細胞內、外液量之改變，其尿酸之排泄量也就隨之而減少了。

（童綜合醫誌 2007; 1: 58-61）

關鍵詞：新生兒、高尿液中尿酸、血尿、尿酸排泄率

簡　介

新生兒期之血尿原因甚多，如新生兒窒息，腎臟動、靜脈栓塞，多發性腎臟囊腫，尿路阻塞性病變，血液相關性疾病，及尿路感染等，[1-2] 需及早評估與治療，以減少日後導致高血壓，腎臟衰竭甚或換腎之困擾。病例爲一有紅色尿布之新生兒，被家長疑有腎臟或相關性疾病而求診，經一連串之檢測不僅確定此紅色尿布嬰兒爲一血尿之嬰 兒，且爲尿液中高尿酸排泄所導致之新生兒血尿。本文僅就此提出報告，以作爲臨床診斷之參考。

病例報告

本病例爲一出生四天大之男嬰，出生後均以配方奶哺育。其父母在爲該嬰兒更換尿布時發現嬰兒尿布爲紅色（見圖），且在數次之尿布更換中均有相同之情況發現，而被帶往醫院做進一步之檢查。其理學檢查爲：體重 3250 公克，體溫、心跳、呼吸分別爲 37.2℃（肛溫）、132 次 / 分及 42 次 / 分，血壓 68/42mmHg。活動力正常，奶量 60-80ml/q3-4h，呼吸音正常，無心臟雜音，腹部無腫塊，四肢無水腫，也無病理學之反射。該嬰兒爲母親之第一胎，經自然生產之足月新生兒，出生時體重爲 3300 公克，無羊水減少現象。家族史中，並無尿路系統或其他相關疾病之報告。實驗室檢查爲：WBC 14200/cumm, N/L/M 68/25/7, Hgb 14.9 gm%, Hct 44%, platelet 316×103/cumm。血清中 BUN 8mg/dl, creatinine 0.5 mg/dl, Uric acid 6.8 mg/dl。尿液檢查爲：pH 7.0, SpGr 1.010, protein(–), OB(++), 0-5 WBC /HPF, 5-10 RBC/HPF，尿液之尿酸排泄率（FEUA ＝）爲 38%，腹部超音波檢查，並無不正常之發現。在確定爲高尿酸排泄合併血尿之診斷後，因無腎臟及其相關之臨床病症發生，故並未給予任何之藥物治療，該嬰兒於住院第三天已無紅色尿布情況下，順利出院。

討　論

本病例在身體檢查及腹部超音波之檢測中並無任何異常之發現，而可排除腎臟囊腫性疾病或阻塞性尿路疾病，如水腎等。因本病例自出生到住院期間，從未有發燒現象，尿液中也無發炎細胞（pyuria）之存在，應與尿路感染無關。該嬰兒出生情況良好，從未放置過臍動脈或臍靜脈導管，無高血壓或腎臟之腫大等，而可排除其爲動、靜脈栓塞之存在。在本病例之血液檢測中，並無血小板等之

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Table 1. Red Urine without RBC

Heme-positive
Hemoglobinuria
Myoglobinuria
Heme-negative
Metabolites
Porphyrin
Melanin
Urate
Drugs
Salicylates
Phenophthalein
Pyridium
Iron sorbitol
Nitrofurantoin
Methyldopa
Metronidazole
Chloroquine
Others
Beets
Blackberries
Food coloring

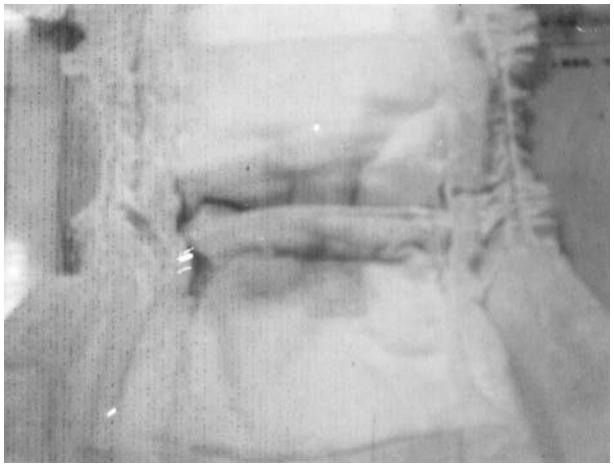

Fig. 1 Red diaper of this newborn.

異常，也無肝、脾之腫大，故應與血液或凝血等之相關疾病無關。

尿酸是 purine 之代謝產物，爲一弱酸，且溶解度低，量大時易在尿液中成晶體出現，而這種以紅色結晶體之尿酸，在新生兒之尿布上被發現，雖與紅色尿布之鑑別原因不少（如表一），但本病例並無相關之病史，而可以排除。[3] 新生兒無論其爲足月或早產，在其出生之頭 1-2 天，均可有相當大之尿酸排泄量（38-60%），正常成人之 FEUA 約爲 10%。[1,3] 而本病例爲一 4-5 天大之嬰兒，不僅也有較成人高出 3-4 倍之多 [4-5] 的高尿酸排泄量（38%），且合併有顯微血尿之產生，但並無任何與腎臟相關之病症出現，這種結果也與 Stapleton 等人 [6] 提出之高尿酸排泄可併有血尿之結果相似。

尿酸在血漿中幾乎不與血漿蛋白結合而呈游離形式，[7] 可近乎完全地從腎絲球濾出。[8-9] 而尿酸排泄之多寡，取決於腎小管之再吸收與分泌。[10] 濾出之尿酸大部分均由近端腎小管吸收，[11-12] 或許有人懷疑是否因近端腎小管之再吸收不足而導致了大量之尿酸排泄，若此，但在新生兒之尿液中卻不見因近端腎小管之再吸收不足，而使尿液中有大量之 Glucose，Na+，HCO_3 一流失。[13-15] 因此，除了腎小管之再吸因素外，腎小管之尿酸分泌在新生兒之高尿酸排泄量也扮演了重要角色。[16] 高量之尿酸在腎小管不僅可造成腎功能之不足，嚴重時甚至可導致腎臟之壞死及腎臟衰竭。[17-19] 至於新生兒有如此高量之尿酸排泄但在臨床上卻甚少見其造成腎臟病變之病例，這可能與新生兒腎臟的重碳酸鹽濾過之閥值較低而形成鹼性尿液及腎小管無法濃縮其尿液而形成了稀釋的尿液有關。因此在這雙重之效應下，反而使得尿酸鹽之結晶不容易在新生兒腎小管沉積造成腎臟損傷。隨著年齡之增長，新生兒腎小管功能漸漸成熟，及其細胞外液量減少之改變，[20-23] 而使腎小管對尿酸之再吸收增加，尿酸在尿液中之排泄量也就因此而減少了。

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Hyperuricosuria with Hematuria in Newborn -- Report of one case

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There were many causes of hematuria in newborn, but it was rarely induced by hyperexcretion of uric acid. Although red diaper was apparent in this case, hematuria was detedted in the correlated tests. Moreover, the value of the fractional excretion of uric acid (FEUA) was up to 38%, as much as 3 to 4 folds in adults (10%). Therefore, hyperuricosuria with hematuria was proved in this case. It may be caused by the impaired function of renal tubules in newborn. However, the hyperexcretion of uric acid in newborn was transient. As the newborn grew-up, the renal tubules matured, and the intracellular and extracellular fluids changed in proportion, the excretion of uric acid was diminished consequently.
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Key words: newborn, hyperuricosuria, hematuria, fractional excretion of uric acid (FEUA)

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童綜合醫學雜誌投稿相關規則

本雜誌刊載與醫學有關之論述，包括原著論文、臨床病理討論、病例報告等論述及特別約稿之綜論（review article）、special article、Editorial（編著的話）等。惠稿請送 43503 台中縣梧棲鎮中棲路一段 699 號童綜合醫學雜誌編審委員會。

壹、投稿前注意事項

1. 投稿時，需附原稿三份（一份原稿和兩份複印稿，但圖片應用三份原圖）並以電腦打字（請以 MS WORD 文書處理格式，中文字型以標楷體，英文字型以 Time New Roman 12 號字大小，稿紙之左右緣爲 2.54 公分，上下緣爲 3.17 公分），請勿裝訂，同時須提供最後版本之電子檔一份，若圖片或照片有電子檔提供者，請以附檔 jpg 的形式提供。
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3. 投稿請參照稿件核對表準備所需項目，同時附上著作權讓與同意書。所有作者必須實際參與並同意該論述。本院於接受稿件且印刷完成後，將贈送 20 份抽印本給通訊作者，如需額外抽印本請於校稿時言明，並酌收成本費用。第一作者若需抽印本可提出申請，依份數酌收成本費用。
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5. 凡刊載於本雜誌之著作，若涉及「研究用人體檢體採集」及「人體試驗」等情事，應遵守該注意事項，以落實保障受檢人權益。詳文請參考須附上相關審議認可之文件。
6. 論文中如涉及使用脊椎動物進行科學應用計畫者，應檢附該計畫業經所屬機構動物實驗管理小組審議認可之文件，以落實實驗動物之人道管理。

貳、寫作原則

1. 原著論文按下列順序撰寫：摘要、前言、材料與方法、結果、討論與結論、誌謝、參考文獻、附表、圖片說明、圖片（含照片）。
2. 病例報告按下列順序撰寫：摘要、前言、病例、討論、參考文獻、附表、圖片說明、附圖、照片。
3. 病例報告，每篇以五頁以內爲限（即約 9,000 字），依題目、所屬機構、作者姓名（作者以 5 人爲限）、病例之病史經過及重要之診療資料、主要之臨床問題、討論或分析、結論、推薦讀物等順序繕寫。凡病患顏面部位之相片必須遮去眼睛部位，表示尊重隱私。診療資料或臨床經過之圖表，原則上均限六個月以內。
4. 綜說不必按原著論文格式撰寫，但必須列出參考文獻。
5. 其他類文章連圖表，以不超過四頁（每頁約 2,000 字）爲原則，但特約稿例外。學術文章，題目、姓名均須以中文書寫。
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參、投稿須知

一、稿件須符合「生物醫學雜誌投稿之統一規定」[1]，請以電腦隔行 double space 書寫並編頁碼。

二、第一頁爲標題頁，須列出中文及英文之論文題目、簡題（running title）、中英文作者姓名、所屬機構及單位之中英文稱號（分屬不同單位，請以阿拉伯數字標出作者與單位）、聯絡人姓名、電話及中英文通訊錄。

三、第二、三頁爲中文及英文之摘要及關鍵詞 (請提供 3 至 5 個關鍵詞或簡短片語)，中英文摘要須完全相同，英文摘要不超過 250 字，中文摘要不超過 500 字，摘要分段撰寫，依序爲背景及目的（Background and purpose）、方法（Methods）、結果（Results）及討論（Discussion）。

四、請附三份原稿（一份原稿和兩份複印稿，但圖片應使用原圖），包括附表、附圖及照片。圖表應專業製作，一張紙僅一個附圖或附表，依引用順序以阿拉伯數字標出排列。附表須有標題及說明。照片須 5 × 7 吋光面黑白，背面以鉛筆編號，附圖須有簡單說明（Legend），並另頁撰寫。光學或電子顯微鏡照片，請註明擴大倍率或比例。

註：[1] 根據「生物醫學雜誌投稿之統一規定」第五版，刊載於 Annals of Internal Medicine 1997; 126(1): 36-47.

肆、參考文獻

未經發表之論文或摘要不得列爲參考文獻，但可於本文中說明並註明「未發表」（unpublished observations）。博碩士論文可引用。已被任何雜誌接受刊發但仍未發表之著作，請列出雜誌名稱及年份，並註明「in press」。

原著論文、臨床病理討論、病例報告等論述及特別約稿之綜論（review article）按下列格式撰寫：

一、雜誌名稱之簡稱須按照 Index Medicus 型式，作者人數小於 6 位時，詳列所有作者姓名，超過 6 位時，只須列出前 6 位，其它以「等」（et al）代替。

例：Bhasin S, Storer TW, Berman N, Callegari C, Clecenger B, Phillips J, et all. The effects of supraphysiologic doses of testosterone on muscle size and strength in normal men. N Engl J Med 1996; 335: 1-7.

二、本文內引用時，若兩名以下作者請列出姓氏。兩名以上則列出第一名之姓氏，其他以「等」（et al）代替，並以阿拉伯數字方括弧表示於引用之後。

例：One of the first well documented reports of ECH poisoning with fatality in young children was reported by Miller et al. in 1970[2].

例：Boulet 等人 [3] 報告氣喘患者接受衛教後的知識改變量不受個人因素影響。

三、參考範例

A. 期刊：[作者姓名：題目。雜誌簡稱　年代；卷數（期數）：起迄頁數]

1. 許吟姿、楊光道、張恆鴻：結締組織疾病併發間質性肺病變患者 99mTc-DTPA 肺廓清率之臨床研究。內科學誌 1992;3:79-83.
2. Yang KTA, Chen HD: A semi-automated method for edge detection in the evaluation of left ventricular function using ECG-gated single-photon emission tomography. Eur J Nucl Med 1994; 21: 1206-11.

B. 單行本：[作者姓名：書名，版數（卷數）。發行地；出版公司，年代：引用部份頁數]。

1. 楊志良：生物統計學新論，一版。台北；巨流圖書公司，1984：33-8.
2. Plum F, Posner JB: Diagnosis of Stupor and Coma. 3rd ed. Philadelphia: Davis, 1980:132-3.

C. 多重作者之單行本：[有關文章作者姓名：書名，版數（卷數）。發行地；出版公司，年代：引用部份頁數]。

1. 蔣欣欣：護理與健康，編輯：顧乃平：護理專業導論，一版。台北；匯華出版公司，1991：83-121。
2. Levinsky NG: Fluid and electrolytes. In: Thorn GW, Adams RD, Braunwald E, lsselbacher K, Petersdprf RG eds. Harrison's Principles of Internal Medicine, 8th ed. New York: Mcgraw-Hill, 1977:364-75.

伍、著作權

若著作人投稿於本刊經收錄後，版權即歸本院所有，除本院同意外不得轉載。若爲摘譯、譯稿或改寫稿，需附原作者之正本同意書，並附原文影本一份；來稿如涉及版權，概由作者自負文責。

童綜合醫學雜誌

原　著

病例報告