台灣胸腔暨重症加護醫學會
Taiwan Society of Pulmonary and Critical Care Medicine

台灣胸腔暨重症加護醫學會

2020夏季會 (線上會議)

2020 Summer Workshop of Taiwan Society of Pulmonary and Critical Care Medicine (Virtual Meeting)

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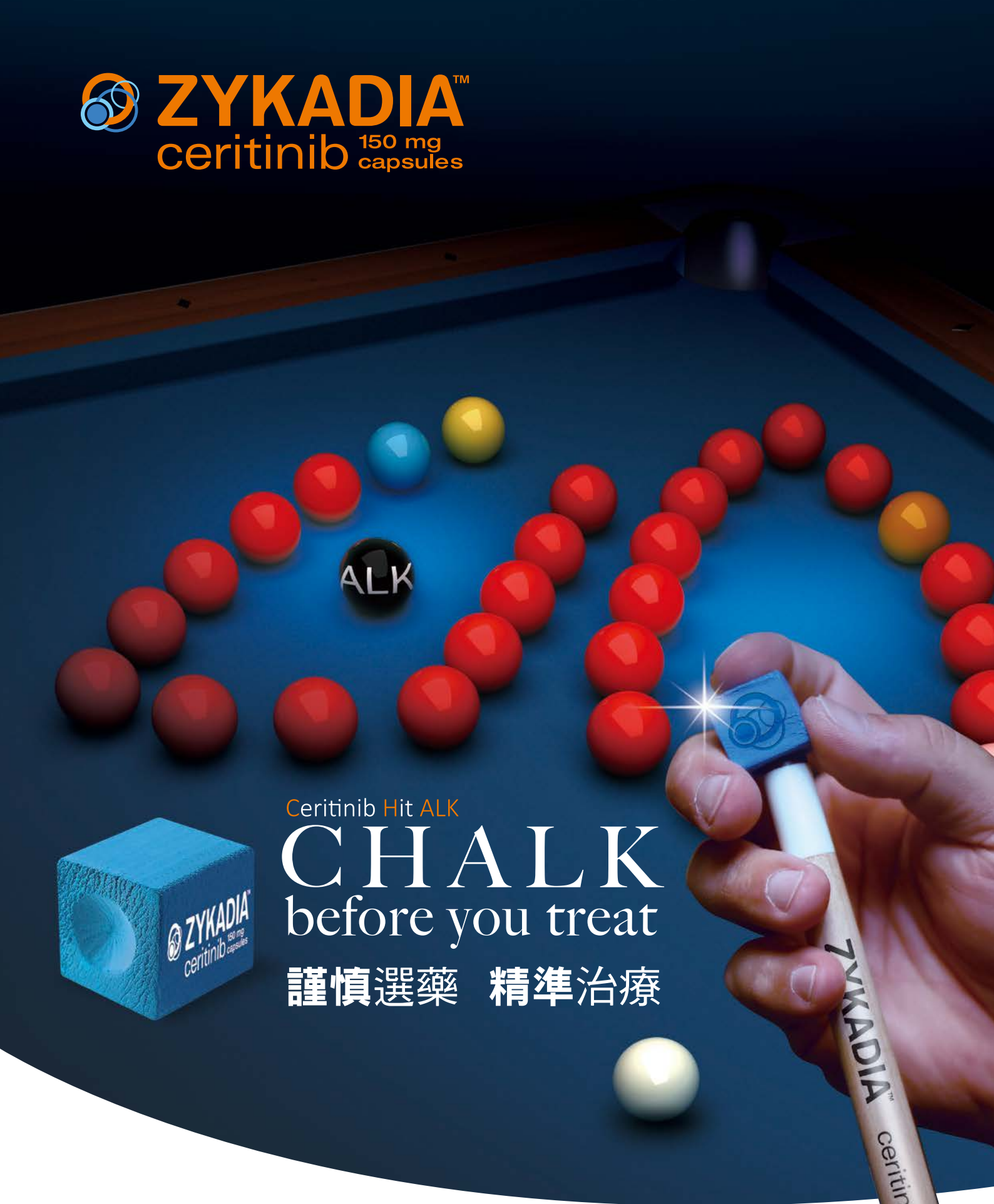

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【適應症及使用方法】治療 ALK 陽性的晚期非小細胞肺癌患者。治療前須經合適之檢驗方式測得 ALK 陽性。【用量】Zykadia 的建議劑量為每日 1 次，於同一時間隨餐口服使用 450 mg 的劑量。每日最大建議劑量為 450 mg 隨餐服用。只要病患仍從治療得到臨床效益，即應持續治療。如果錯過劑量，病患應補服該次劑量，除非該次劑量與下一次服藥時間相距不到 12 小時。對於每天服用劑量低至隨餐服用 150 mg Zykadia 仍耐受不良的病患，應中止使用 Zykadia。【禁忌】對活性成分或下列之任一賦形劑過敏者：膠囊內容物組成：Microcrystalline cellulose，Low substituted-hydroxypropylcellulose，Sodium starch glycolate (type A)，Magnesium stearate，Silica colloidal anhydrous。Capsule shell: Gelatin，Indigotine (E132)，Titanium dioxide (E171)。Printing ink: Shellac (bleached, de-waxed) glaze 45%，Iron oxide black (E172)，Propylene glycol，Ammonium hydroxide 28%【警語】肝臟毒性：應在開始治療前監測病患的肝臟實驗室檢測數值 (包括 ALT、AST 及總膽紅素)，之後第一個月每 2 週監測一次，以及接下來每個月監測一次。在發生轉胺酶升高的病患中，應視臨床需要進行更頻繁的肝臟轉胺酶及總膽紅素監測。間質性肺病/肺炎：應監測病患是否出現為間質性肺病/肺炎徵兆的肺部症狀。QT 間隔延長：此症狀可能增加心室頻脈 (例如 Torsade de pointes 型 心律不整) 或猝死的風險。應避免讓有先天性長 QT 症候群的病患使用Zykadia。已知有心搏過慢 (心跳速率低於每分鐘 60 下[bpm]) 的病患、有 QTc 延長史或具有 易發生 QTc 延長體質的病患、正在使用抗心律不整藥物或已知會延長 QT 間隔之藥物的病患，以及已知有重大心臟疾病及/或電解質異常的病患，在開始接受治療之前，應考量治療效益及可能的風險。心搏過慢：Zykadia 應儘量避免與已知會造成心搏過慢的其他藥物 (例如 β 阻斷劑、非 dihydropyridine 類鈣離子通道 阻斷劑，clonidine 及毛地黃) 併用，應定期監測心跳速率及血壓。腹瀉甚不良反應：應監測病患並以標準照護方式處置，包括視需要給予止瀉劑、止吐劑或輸液補充。必要時可中斷劑量或降低劑量。如果治療中發生嘔吐事件，病患不應補服額外劑量，應持續依照下一次既定排程與劑量服藥。高血糖症：糖尿病病患以及/或同時使用類固醇的病患，有較高的高血糖風險。開始 Zykadia 治療之前應監測空腹血糖值，之後則視臨床需要定期監測。視情況開始使用或調整降血糖藥品。脂肪酶及/或澱粉酶增加：接受 ceritinib 治療的病患曾有通報發生胰臟炎的案例，開始 Zykadia 治療之前應監測病患的脂肪酶及澱粉酶是否增加，之後視臨床需要定期監測。【極常見副作用】貧血，食慾減退，腹瀉，噁心，嘔吐，腹痛，便秘，食道異常，皮疹，疲勞，肝實驗室檢測結果異常，體重減輕，血中肌酸酐增加

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序言

歡迎各位會員、先進及同道參加夏季會線上會議。

在兩個月前考量全世界及台灣的 COVID-19 疫情影響，我們決定將原定於 2020 年 6 月 20、21 日舉行的第三屆夏季會於改為線上會議方式辦理。雖然目前台灣疫情已受控制，並已解封。但考慮再次變動的時間緊迫，我們仍決定維持視訊方式。

本次學術研討會依往例主題涵括了呼吸道疾病、重症醫學、呼吸道感染、肺癌、間質性肺病、睡眠醫學、肺部環境醫學等最新進展。另特別邀請衛生福利部國民健康署 賈淑麗副署長擔任大會貴賓談論以實證為基礎之慢性呼吸道疾病防治政策推展。題目多元精彩，希望在 COVID-19 疫情影響下這些課程能讓會員們掌握胸腔領域最新知識學習，了解未來的發展，也能相互交流。

感謝學術委員會的精心規劃，受邀講者的用心準備，全體理監事的支持協助、更歡迎大家熱烈參與！

林孟志 理事長

Agenda

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6月20日（星期六）

13:20 | 13:30	理事長致詞		
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14:05 | 14:15	各公司產品簡介		
14:15 | 14:50	Moderator: **楊政達** 院長 Speaker: **呂紹煒** 醫師 Topic: *Update of ventilator induced lung injury*	Moderator: **蘇維鈞** 教授 Speaker: **莊校奇** 副教授 Topic: *Association between air pollution and tuberculosis infection*	Moderator: **余忠仁** 院長 Speaker: **柯信國** 醫師 Topic: *Rapidly progressive interstitial lung disease (RP-ILD)*
14:50 | 15:00	各公司產品簡介		
15:00 | 15:30	Moderator: **林孟志** 理事長 Speaker: 衛生福利部國民健康署 **賈淑麗** 副署長 Topic: *The Evidence-based of CRD Prevention and Promotion*		
15:30 | 15:40	各公司產品簡介		
15:40 | 16:15	Moderator: **蔡煥煌** 院長 Speaker: **張厚台** 醫師 Topic: *Evidences of sedation and paralysis for critically ill MV patients*	Moderator: **黃明賢** 教授 Speaker: **陳俊谷** 醫師 Topic: *Quantitative CT imaging and analysis of pulmonary fibrosis*	Moderator: **曹昌堯** 副校長 Speaker: **王才郁** 醫師 Topic: *OSA and COPD: the overlap syndrome*
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16:25 | 17:00	Moderator: **蔡煥煌** 院長 Speaker: **張志豪** 醫師 Topic: *Role of lung biopsy in acute respiratory distress syndrome with unknown etiology*	Moderator: **黃明賢** 教授 Speaker: **李枝新** 醫師 Topic: *Update on Medical Treatment for COVID-19*	Moderator: **曹昌堯** 副校長 Speaker: **蕭慈慧** 醫師 Topic: *Impulse Oscillometry to Predict the Severity of Obstructive Sleep Apnea*

A
B
C

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6月21日(星期日)

	直播室 A	直播室 B	直播室 C
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09:15 | 09:25	各公司產品簡介		
09:25 | 10:00	Moderator: **林恒毅** 院長 Speaker: **郭家佑** 醫師 Topic: *Tissue vs liquid biopsy: the advantage, correlation and clinical outcome*	Moderator: **徐武輝** 副院長 Speaker: **黃萬均** 醫師 Topic: *A Global Perspective of Implementing Evidence-Based Strategies for COPD*	Moderator: **鍾飲文** 教授 Speaker: **蘇一峰** 醫師 Topic: *E-cigarette and vaping: help or harm?*
10:00 | 10:10	各公司產品簡介		
10:10 | 10:45	Moderator: **夏德椿** 主任 Speaker: **黃建勝** 醫師 Topic: *Preoperative biopsy and tumor recurrence of stage I adenocarcinoma of the lung*	Moderator: **薛尊仁** 教授 Speaker: **廖信閔** 醫師 Topic: *Recent advancement in biologic therapy of severe asthma*	Moderator: **黃坤崙** 教授 Speaker: **孫文榮** 醫師 Topic: *Shared Decision Making in Patients with Terminal Lung Disease*
10:45 | 10:55	各公司產品簡介		
10:55 | 11:30	Moderator: **夏德椿** 主任 Speaker: **謝燿宇** 醫師 Topic: *Is there difference in first line TKIs? Perspectives from clinical trial to real world data*	Moderator: **薛尊仁** 教授 Speaker: **陳彥甫** 醫師 Topic: *The role of lung microbiome in chronic lung disease*	Moderator: **黃坤崙** 教授 Speaker: **藍胄進** 醫師 Topic: *The role of pulmonary rehabilitation in palliative care*

(25~30 分鐘演講，5~10 分鐘 Q&A)

直播室 A

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June 20

June 21

阮聖元 醫師 / Sheng-Yuan Ruan, M.D., Ph.D.

現職：臺大醫院內科部主治醫師、臺大醫學院臨床助理教授
專長：呼吸衰竭之臨床流行病學及生理學、重症醫學、胸腔醫學

Phenotypes of ARDS
急性呼吸窘迫症候群的表現型

Acute respiratory distress syndrome (ARDS) is a life-threatening condition of critically ill patients characterized by rapid onset of widespread inflammation in the lungs. The prevalence of ARDS is estimated to be 25% in ventilated ICU patients and the mortality ranges from 30% to 45%, depending on the severity. The diagnosis of ARDS relies on the clinical criteria of acute onset of hypoxemia with a PaO2/FiO2 ratio less than 300 mmHg, bilateral pulmonary opacities on chest radiograph, and exclusion of cardiac origin of lung edema. Despite with a well-defined operational definition in diagnosis, there has been increasing recognition of the heterogeneity within ARDS. The heterogeneity in ARDS has been reported in clinical trajectory, radiological presentation, respiratory mechanics, biomarker profiles, outcomes and responses to therapy. It’ s considered that the heterogeneity in ARDS has contributed to the statistically negative results of many clinical trials. In the last decade, significant progress has been made in finding homogeneous subsets within ARDS. Several strategies have been proposed for identifying the phenotypes of ARDS, including etiology based, physiology based, biomarker based and omics-derived methods. I aim to review the current strategies in identifying phenotypes of ARDS and the potential implications of these approaches in prognostication and management of ARDS.

呂紹煒 醫師 / Shaw-Wei Leu, M.D.

現職：林口長庚醫院胸腔內科系主治醫師
專長：胸腔醫學、重症醫學

Update of Ventilator Induced Lung Injury
呼吸器相關肺損傷之新進展

Acute respiratory distress syndrome (ARDS) consists of heterogeneous pathophysiology with mortality rate as high as 40 to 50% in severe cases. Currently there are no specific therapies for ARDS. The major supportive treatment is mechanical ventilation. However, inadequate ventilator setting could result in ventilator induced lung injury (VILI), which caused further damage of injured lung and contributed to the mortality of ARDS. The ventilator related causes of VILI included volume, pressure, respiratory rate and flow. A lung protective strategy with low tidal volume of 4-8 ml/kg predicted body weight, low plateau pressure < 30 cm H2O, higher PEEP in moderate to severe ARDS patients were suggested. Furthermore, lower driving pressure (plateau pressure minus PEEP) has been shown to correlate with better survival in ARDS patients. Recently a novel concept "mechanical power" (MP) have been proposed as an integrative variable of VILI. With calculation of MP, each component of ventilator settings could be evaluated for their relative contribution to VILI. Mechanical power threshold may be a survival predictor for ARDS patients. Retrospective studies showed lower MP in the early days of ventilation is correlated with survival benefit not only for ARDS patients but also for the critically ill patients admitted to ICU. Considering varying severity of lung injury in individual patients, such as the "baby lung" ratio, absolute level of MP may need normalization to the functional lung volume for better reflection of the mechanical energy transmitted to the lung unit. Further studies are needed to confirm the role of MP in predicting survival and to guide the ventilator strategy in patients with ARDS.

賈淑麗 副署長

Shu-Li Chia Deputy Director General

現職：衛生福利部國民健康署 副署長

The Evidence-based of CRD Prevention and Promotion
以實證為基礎之慢性呼吸道疾病防治政策推展

慢性呼吸道疾病為全球四大非傳染病之一，今年嚴重特殊傳染性肺炎 (COVID-19) 疫情蔓延全球，有呼吸道症狀的民眾及已罹患慢性呼吸道疾病的患者，皆為感染的高風險群，為提供國人良好的呼吸道照護品質，臺灣持續透過慢性病防治三段五級的架構，用完整生命歷程等全人健康照護概念，促進民眾健康、辦理疾病篩檢、早期偵測及早診斷並治療。

實證資料顯示，導致慢性阻塞性肺病 (COPD) 的原因有九成為吸菸所引起，其他可能為有毒之氣體如汽機車排放物二氧化硫、空汙等，因此在 COPD 防治政策的推動上，本署從前端菸害防治政策著手，強化對民眾宣導戒菸與不吸菸的衛教資訊，同時亦攜手實證醫學會與胸腔重症加護醫學會等專業學協會，從政策端、醫療專業端及民眾端等多面向的疾病防治策略，全面性的推動呼吸道相關計畫，提升疾病照護品質。另透過國際臨床照護實證及本土國人肺阻塞 (COPD) 之疾病情形，研發台灣本土性「肺阻塞臨床照護指引」，並於 2017 年結合健保署「慢性阻塞性肺病醫療給付改善方案」透過醫療院所之整合方案及分級管理，來提供慢性阻塞性肺部疾病患者較佳照護品質，強化肺阻塞病人照護品質。

由於台灣肺阻塞患者年齡分布以 65 歲以上長者居多，控制不良易導致多重共病症，因此在疾病的照護上更應提供以病人為中心整合照護，以 Value-base Payment 之疾病管理概念，將預防與治療並重，以符合病人照護需要。爰本署目前試行推動多重慢性病整合計畫，透過跨專科醫事人員提供多重慢性病整合照護，增強醫護的專業照護能力，建立一致性照護標準，並強化醫院與基層院所個案之分級照護，透過院際與診所之資訊系統串接強化個案資料轉接與相關數據之分享與應用，以順利個案照護。

因應 COVID-19 後疫情下的呼吸道照護，除持續透過媒體行銷民眾及肺阻塞患者的戒菸防治重要性，與既有臨床照護標準遵照醫師的治療計畫用藥外，如何結合雲端照護，例如以彰基應用「ICOPD 呼吸管家」APP，紀錄個案的健康狀況，並連結醫院資訊系統的協助，藉院方專業客服務提供線上即時協助，或是針對弱勢獨居或有需協助之個案應用遠距照護，都是提升疾病管理作法上需精進的作法。

張厚台 醫師 / Hou-tai Chang, M.D., Ph.D.

現職：亞東紀念醫院重症醫學部部長、內科加護病房主任、
胸腔內科主治醫師
專長：重症加護醫學、呼吸照護、超音波學、醫療品質與管理

Evidences of sedation and paralysis for critically ill MV patients
呼吸器使用病患之鎮靜原則

Critically ill patients required mechanical ventilators as part of their care. Use of sedations and analgesics makes critically ill patients comfort and calm down. The Society of Critical Care Medicine recently published their updated clinical practice guidelines for analgesia, agitation, sedation, delirium, immobility, and sleep in adult patients in the ICU[1]. Why the guideline suggested about sedation interruption with light sedation and how it reached the conclusion are the focus we should know. In a trial published in NEJM in 2000[2], daily sedation interruption decreased the duration of mechanical ventilation and length of ICU stay. The strategy also decreased mechanical ventilation related complications in other trial[3]. Extended trial with paired sedation interruption and spontaneous breathing trial showed shorter time to extubate the patients and ICU length of stay[4]. However, one study published in 2012[5] suggested higher midazolam dosage and higher nursing workload in protocolized sedation and daily sedation interruption. The trial also suggested benefit of daily sedation interruption in surgical and trauma patients. One study compared daily sedatives interruption with nursing implemented sedation algorithm failed to identify the benefit of daily sedation interruption[6]. to clarify the situation, a trial performed at a single ICU showed that the number of days of invasive ventilation was less in a group assigned to a strategy of no or minimal sedation than in a group assigned to a strategy of sedation with daily interruption[7].

1. Devlin, J.W., et al., *Clinical Practice Guidelines for the Prevention and Management of Pain, Agitation/Sedation, Delirium, Immobility, and Sleep Disruption in Adult Patients in the ICU*. Crit Care Med, 2018. 46(9): p. e825-e873.
2. Kress, J.P., et al., *Daily interruption of sedative infusions in critically ill patients undergoing mechanical ventilation*. N Engl J Med, 2000. 342(20): p. 1471-7.
3. Schweickert, W.D., et al., *Daily interruption of sedative infusions and complications of critical illness in mechanically ventilated patients*. Crit Care Med, 2004. 32(6): p. 1272-6.
4. Girard, T.D., et al., *Efficacy and safety of a paired sedation and ventilator weaning protocol for mechanically ventilated patients in intensive care (Awakening and Breathing Controlled trial): a randomised controlled trial*. Lancet, 2008. 371(9607): p. 126-34.
5. Mehta, S., et al., *Daily sedation interruption in mechanically ventilated critically ill patients cared for with a sedation protocol: a randomized controlled trial.* JAMA, 2012. 308(19): p. 1985-92.
6. de Wit, M., et al., *Randomized trial comparing daily interruption of sedation and nursing-implemented sedation algorithm in medical intensive care unit patients*. Crit Care, 2008. 12(3): p. R70.
7. Strom, T., T. Martinussen, and P. Toft, *A protocol of no sedation for critically ill patients receiving mechanical ventilation: a randomised trial*. Lancet, 2010. 375(9713): p. 475-80.

張志豪 醫師 / Chih-Hao Chang, M.D.

現職：林口長庚醫院主治醫師
專長：支氣管鏡檢查、重症照護

Role of lung biopsy in acute respiratory distress syndrome with unknown etiology
肺部切片在病因不明的急性呼吸窘迫徵候群的角色

Acute respiratory distress syndrome (ARDS) is not a rare syndrome in intensive care units. Despite lung protective strategy has been widely applied to avoid further lung injury, the morality rate of ARDS remains high in most studies. Many researchers tried to find medication to improve the outcome of ARDS, but no medication could be a game-changer for ARDS patients in recent decades.

The diagnosis of ARDS is defined by Berlin definition by clinical parameters from 2012. Therefore, the clinical definition includes a wide heterogeneous population and these patients have varied etiologies, different prognoses, and diverse response to specific therapies. Intensivists could give their patients specific therapies if they find out specific etiologies of ARDS.

Pathology is difficult to obtain in patients with ARDS. The typical pathological findings of ARDS is considered to be diffuse alveolar damage, presenting with alveolar hemorrhage, alveolar edema, or hyaline-membrane formation. However, the diffuse alveolar damage is not specific for ARDS. Besides, the autopsy or biopsy studies showed that diffuse alveolar damage is present half of patients with ARDS.

In the session, the speaker will share the experience of surgical lung biopsies and bronchoscopic cryobiopsies in patients with ARDS at Linkou Chang Gung Memorial Hospital.

楊景堯 醫師 / Ching-Yao Yang, M.D., Ph.D.

現職：國立台灣大學醫學院附設醫院內科部主治醫師
專長：內科學、胸腔醫學、肺癌、呼吸道內視鏡

Implication of PD-L1 expression in Lung cancer with driver mutation
PD-L1表現在具有驅動突變之肺癌的應用

PD-L1 expression has been used as a biomarker of immune checkpoint inhibitors (ICIs) targeting PD-1/PD-L1 axis, especially in lung cancers without driver mutations. Tumors with higher PD-L1 expression had a better response to ICI. Otherwise, lung adenocarcinoma with driver oncogenes, such as EGFR, ALK, and ROS1, generally did not respond well to ICI treatments, irrespective of PD-L1 expression. Recently, more studies have investigated the role of PD-L1 in oncogene-driven tumors. Our studies found PD-L1 showed a smaller extent of expression in EGFR and ALK mutated tumors, compared with EGFR/ALK wild type tumors. Pre-treatment PD-L1 in EGFR or ALK mutated tumors had a predictive impact on the efficacy of targeted therapies. Thus, testing PD-L1 in EGFR or ALK+ lung adenocarcinoma could provide important information of prognosis prediction.

A
B
C

郭家佑 醫師 / Chia-Yu Kuo, M.D.

現職：高雄醫學大學附設中和紀念醫院 胸腔內科主治醫師
專長：一般內科、胸腔內科、重症醫學、老人醫學、睡眠醫學、肺癌

Tissue vs liquid biopsy: the advantage, correlation and clinical outcome
液態切片與組織切片之比較-優勢、關連性及臨床預後

Lung adenocarcinoma is the most common type of non-small-cell lung cancer (NSCLC). The identification of epidermal growth factor receptor (EGFR) gene mutation is very important for newly diagnosed advanced/metastatic disease. The traditional method is gene analysis from biopsied tissue. The procedures for tissue biopsy are usually invasive. Liquid biopsy is less invasive and can identify circulating tumor DNA from patients' blood. According to one recent study analyzing the association between plasma genotyping and treatment outcomes of osimertinib in advanced NSCLC patients who failed to the first-line EGFR TKIs therapy, patients with T790M mutation detected by either liquid biopsy or tissue biopsy had similar outcomes. The association between clinical features and detectable ctDNA has been investigated in some previous studies. Recent studies of late-staged NSCLC patients even showed that bone metastasis was the independent factor predicting ctDNA detection.

In a retrospective study to evaluate the predicting factors and clinical outcomes associated with concordant results in liquid/tissue biopsy in newly diagnosed lung adenocarcinoma patients with EGFR mutations in Kaohsiung Medical University Hospital, 80 patients with stage III or IV lung adenocarcinoma were enrolled. 51 patients had EGFR mutation detected in tissue samples, while 33 (65%) of them had concordant results shown in liquid biopsy. Multivariable regression analysis showed that lymph node involvement (adjusted odds ratio [95% CI]: 8.71 [1.88-40.35], p = 0.0057) and bone metastasis (adjusted odds ratio [95% CI]: 9.65 [1.72-54.05], p = 0.0099) were the independent predicting factor for concordant results. Forty of these 51 patients were stage IV and were treated with EGFR tyrosine kinase inhibitors (TKIs). The concordant results in liquid/tissue samples was associated with significantly poorer progression-free survival (PFS) in univariate analysis.

Based on recent studies, in advanced lung adenocarcinoma patients having insufficient tissue sample for EGFR mutation testing, liquid biopsy to determine EGFR mutation status might be particularly useful if they have lymph node involvement and/or bone metastasis. The concordant results in liquid/tissue samples might indicate a larger tumor burden, as evidenced by the presence of lymph involvement, which actually contribute to poorer PFS. The physicians should be more careful while caring for these patients.

黃建勝 醫師 / Chien-Sheng Huang, M.D.

現職：台北榮總外科部胸腔外科主治醫師
專長：胸腔外科手術

Preoperative biopsy and tumor recurrence of stage I adenocarcinoma of the lung
第一期肺腺癌腫瘤復發與手術前施行切片診斷的相關性探討

Lung cancer remains the leading cause of cancer-related deaths worldwide, and many patients in different facilities and countries experience substantial waits for lung cancer diagnosis and subsequent treatment. Tissue diagnosis through small-piece biopsies to confirm the pathological diagnosis of lung cancer is essential for deciding on the most appropriate treatment strategy, especially for patients with advanced non-small cell lung cancer (NSCLC). As therapy for NSCLC patients becomes more individualized, further tissue samples for molecular analysis, in addition to routine histopathological examinations, are being required to decide on the appropriate therapy strategy and for enrollment into clinical trials. Currently, the main treatment methods for early-stage NSCLC is surgical resection. There is still controversy among surgeons about whether preoperative biopsy increases the possibility of recurrence. Accordingly, the relationships between tumor recurrences of pathologic stage I lung adenocarcinoma and preoperative biopsy will be discussed in this study.

謝燿宇 醫師 / Yao-Yu Hsieh, M.D., Ph.D.

現職：衛生福利部雙和醫院血液腫瘤科病房主任
專長：一般血液疾病、急慢性白血病、淋巴惡性腫瘤、癌症免疫療法、癌症標靶治療、癌症化學治療、消化道癌症治療、癌症新藥臨床試驗

Is there difference in first line TKIs? Perspectives from clinical trial to real world data
一線TKI的選擇，從臨床試驗到台灣實際經驗分析

Epidermal growth factor receptor tyrosine kinase inhibitors (EGFRTKIs) have been the standard of care among patient with lung adenocarcinoma harboring epidermal growth factor (EGFR) mutations. In Taiwan, there are three EGFRTKIs, including gefitinib, erlotinib, and afatinib, currently reimbursed by the National Health Insurance (NHI) program as first-line treatment for patients with advanced non–small cell lung cancer (NSCLC) harboring EGFR mutations. Although all of them exhibit superior progression-free survival

(PFS) to traditional chemotherapys, few prospective trials have directly compared these TKIs in the first-line settings. The study was to compare the effectiveness of different epidermal growth factor receptor—tyrosine kinase inhibitors (EGFRTKIs) in patients with advanced non-small-cell lung cancer (NSCLC) and received EGFR-TKIs as first-line therapy. This retrospective cohort study was conducted using data from real-world settings. Patients with stage IIIB and IV NSCLC and first received gefitinib, erlotinib, or afatinib between 2011 and 2015 were included. The date of the first claim for EGFR-TKIs was set as the index date. Study endpoints were all-cause death and treatment failure that was defined when patients added on or switched to chemotherapy or terminal care. A total of 5,940 patients, including 3,982 (67.0%) receiving gefitinib, 1,207 (20.3%) receiving erlotinib, and 751 (12.7%) receiving afatinib, were eligible for this study. The 1-year overall survival (OS) rates for gefitinib, erlotinib, and afatinib groups were 74% (95% confidence interval [CI]: 72–75%), 75% (95% CI: 73–77%), and 80% (95% CI: 77–83%), respectively. Compared to gefitinib, afatinib was associated with a lower risk of allcause death (adjusted hazard ratio [aHR] = 0.82, 95% CI: 0.72–0.93) but not erlotinib (aHR = 0.95, 95% CI: 0.86–1.05). Similar results were also found regarding the effectiveness of treatment. All the three EGFR-TKIs showed no differences for both outcomes among patients with an Eastern Cooperative Oncology Group Performance Score of 2. The real-world data exhibited afatinib was more likely to be used for younger patients in a better condition than other EGFR inhibitors, and observed prolonged OS and treatment effectiveness compared to gefitinib after performing a multivariate Cox regression analysis.

直播室 B

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張立禹 醫師 / Lih-Yu Chang, M.D.

現職：臺大醫院新竹分院胸腔科主治醫師
專長：介入性支氣管鏡檢查／治療、肺結核

The role of intervention technique in diagnosis of tuberculosis
介入性技術在診斷結核病的角色

Although tuberculosis (TB) is one the most important infectious diseases, early diagnosis of TB is still a challenge. For pulmonary tuberculosis, about half of patients have smear-negative sputum or could not product sputum. Bronchoscopy and endobronchial ultrasound are good tools to conquer this problem. These procedures could get adequate microbiology or histology evidences for early diagnosis and drug sensitivity test. Similar problem happens at TB pleurisy. The low smear positive rate and culture positive rate of pleural effusion let TB pleurisy diagnosis difficult. Pleuroscopy resolves this problem by higher yield rate of histology exam and TB culture. Interventional techniques are helpful for early, definite diagnosis.

莊校奇 副教授 / **Hsiao-Chi Chuang, Ph.D.**

現職：臺北醫學大學呼吸治療學系副教授
專長：呼吸道疾病、環境毒理、環境衛生、環境職業醫學

Association between air pollution and tuberculosis infection
空氣污染與結核感染的關聯性

Tuberculosis (TB) is a communicable disease caused by the inhalation of bacillus Mycobacterium TB. TB is spread to others when patients with pulmonary TB expel bacteria. In 2013, an estimated 9 million people developed TB and 1.5 million people died from TB due to the comprehensive WHO strategy for TB control. Air pollutants are considered to influence the development of pulmonary disease by interfering with non-specific and specific lung defences. Many studies conducted over 30 years have shown an association between air pollution and TB. People who are exposed to particulate matter that is less than 10 μm in aerodynamic diameter (PM10) generated from biomass burning are more likely to be infected with Mycobacterium TB and progress to TB disease. Therefore, it is an urgent issue to understand the role of air pollution on TB infection

陳俊谷 主任 / Chun-Ku Chen, M.D., Ph.D.

現職：臺北榮民總醫院放射線部科主任
專長：胸腔介入診療、心血管影像學、肺部影像學診斷

Quantitative CT imaging and analysis of pulmonary fibrosis
肺纖維化定量電腦斷層影像分析

HRCT is an important imaging modality for diagnosis of fibrosing lung diseases; however, current CT interpretation is more of qualitative and subjective. Using conventional visual CT interpretation to monitoring the disease progression or exploring novel treatment for pulmonary fibrosis is sometimes difficult.

Several methods were used for quantifying fibrotic change of the lung parenchyma. In lung density analysis, mean lung density, were shown to increase with lung fibrosis, while the kurtosis and skew characteristics would decrease. These density profiles were shown to have correlation with disease severity and showed changes with disease progression.

Texture-based analysis takes spatial relationship between voxel into consideration and simulate human perceptual to classify parenchymal changes and could correlate with expert radiologist assessment. The application of machine learning could improve the algorithm performance.

Agnostic features such as pulmonary vessel volume was emerging as an analytic tool, it was shown to outperformed physiological indices and visual CT parameters to predict prognosis by literature.

While there were multiple tools to quantify the lung fibrosis, there were limitations and challenges, the inspiration cooperation, scanning parameters, reconstruction method could affect the analysis result, standardization is important for applying the quantifying analysis in clinical and research field.

李枝新 醫師 / Chih-Hsin Lee, M.D., Ph.D.

現職：台北市立萬芳醫院結核病中心主任、
台北市立萬芳醫院胸腔醫學研究中心主任
專長：胸腔醫學、結核病、醫學資訊、流行病學

Update on Medical Treatment for COVID-19
2019新冠病毒感染的藥物治療進展

The severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection, also known as the Corona Virus Disease-19 (COVID-19) was initially identified in Wuhan, China in December 2019, and emerged as global pandemic with 4,749,813 confirmed cases, and a total of 314,921 deaths globally as of May 18, 2020. Although a high proportional of infected patients recovered spontaneously with minimal symptoms, severe inflammatory response and acute lung injury, in addition to lymphopenia and cytokine release syndrome have been frequently reported as important clinical features of COVID-19 patients, suggesting the pivotal role of immune system homeostasis in pathogenesis process. Definite treatment for COVID-19 is still unclear. However, to minimize the death and morbidities, in-vitro and in-vivo studies repurposing old drugs are on-going to find the potential drugs and optimal dosing strategies. Emerging evidence is growing rapidly through the global collaboration. Among the drugs being tested, the benefit of Remdesivir, Ritonavir/Lopinavir and Hydroxychloroquine are highly anticipated. On the other way, covalescent plasma from the patients who recovered from COVID-19 was also used to treat the patients with severe disease. The present talk will integrate recent progress in treatment of COVID-19 to evaluate the efficacy and adverse events of the corresponding potential drugs. Evidence based on observational studies for host-directed therapy such as angiotensin-II inhibitors will also be reviewed.

謝孟亨 醫師 / Meng-Heng Hsieh, M.D.

現職：長庚醫學中心主治醫師、桃園長庚醫院加護病房主任
專長：支氣管擴張症、氣喘、肺阻塞、肺癌

The assessment of Non-cystic fibrosis (CF) bronchiectasis in adults: focus on clinical phenotypes
成人支氣管擴張症的評估：聚焦於臨床表現型的探討

Bronchiectasis is a rapidly developing field in pulmonology. This emerging epidemic of chronic and progressive immune-infective-inflammatory airway destruction results in a vicious cycle of repeated exacerbations and irreversible damage that now clearly necessitates greater global focus and investment.

Recent progress in assessment of the diseases including two prognostic indices that aid clinical decisions is the bronchiectasis severity index (BSI) and the FACED score. But questions in bronchiectasis, from pathophysiology to long- term management, remain unanswered; the first step in solution might involve the selection of these patients: namely, phenotyping. In practical terms, phenotype describes aspects of the patient that influence clinical decision-making (e.g., the need for close monitoring because of a worse prognosis) or, perhaps more importantly, describes how a patient should be treated based on a specific response to a therapy. Although disease phenotyping in non-CF bronchiectasis is in its infancy, analyses performed to date suggest enormous heterogeneity in disease severity and presentation as well as potential to identify populations with greater likelihood of treatment response and varying prognoses.

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黃萬均 醫師 / Wan-Chun Huang, M.D.

現職：台北醫學大學 – 衛生福利部雙和醫院胸腔內科主治醫師
專長：慢性呼吸道疾病、流行病學、全球衛生

A Global Perspective of Implementing Evidence-Based Strategies for COPD
實施COPD實證策略的全球視角

In the era of an aging global population, the challenge of non-communicable diseases (NCDs) on healthcare systems is enormous. Chronic Obstructive Pulmonary Disease (COPD) is one of the main NCDs, nevertheless receiving little attention and funding in comparison with other major causes of global morbidity and mortality. Over the past decade, there has been a ring trend in the prevalence of COPD. Despite the availability of evidence-based strategies that aim to reduce the burden of COPD, such as the Global Initiative for Chronic Obstructive Lung Disease (GOLD) report, COPD is still expected to become the leading cause of death worldwide. Twenty years following the initiation of GOLD, is the world doing better in combating COPD? In this presentation, I will be sharing how these strategies have been implemented in real-world, how we might be able to address the issues of implementation in the future, and how, as a member of the global community, Taiwan can help.

廖信閔 醫師 / Xin-Ming Liao, M.D.

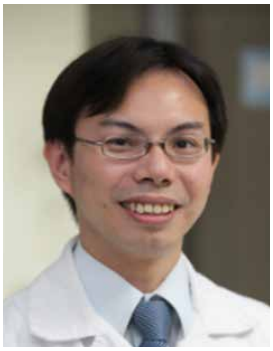
現職：國立成功大學醫學院附設醫院內科部主治醫師
專長：一般內科醫學、胸腔免疫學、重症加護醫學、睡眠醫學、胸腔腫瘤學

Recent advancement in biologic therapy of severe asthma
生物製劑在嚴重氣喘近期的發展

Type 2 severe asthma, one of the phenotypes in the asthma, is associated with type 2 inflammation and affecting more than half of severe asthma patients. The underlying mechanisms of type 2 inflammation in lower airway are driven by type 2 T-helper cells and group 2 innate lymphoid cells (ILC2) through the secretion of various effector cytokines, including interleukin (IL)-4, IL-5, and IL-13. The heterogeneity between individual is a challenge for clinician in diagnosing and treating severe asthma subjects. Due to the advancement of medical science, we now can perform molecular testing to delineate endotype signature of each severe asthma subjects. With knowing the type 2 inflammation-related biomarker profiles (IgE, blood eosinophil count, FeNO) of each patient combining clinical phenotypes evaluation, the corresponding biologics (omalizumab, mepolizumab, benralizumab, dupilumab), in terms of each mechanism of action, can be administered for severe asthma treatment according to GINA guideline recommendation. Accumulated study evidence revealed the clinical benefit of biologic therapy in terms of lowering acute exacerbation, improving quality of life, and corticosteroids sparing effects. In the era of personalized medicine, we should offer optimized treatment for severe asthma subjects not only at the level of pheynotype, but endotype as well.

陳彥甫 醫師 / Yen-Fu Chen, M.D.

現職：臺大醫院雲林分院門診部主任
專長：肺部疾病及感染、一般內科及重症加護疾病、慢性呼吸道疾病（氣喘、慢性阻塞性肺病、特發性肺纖維化）、胸腔腫瘤、肺癌、胸部超音波檢查，支氣管鏡檢查



The role of lung microbiome in chronic lung disease
肺微生物叢基因體在慢性肺部疾病的角色

In past decade, the studies of the lung microbiota using culture-independent techniques (such as 16S rRNA sequencing) have demonstrated that bronchial tree is not sterile in either healthy or chronic lung disease individuals. Increasing evidences showed that the lung microbiome may play a potential role in the pathogenesis of some chronic lung diseases (e.g. COPD), which are relevant to the dysbiosis of lung microbiota. However, most studies are only correlative and conjecture about a causative link between the lung microbiota and chronic lung disease severity/exacerbation. Only few researches investigate the impact of therapeutic interventions (such as macrolide) on lung microbiome and the mechanism of interaction between lung microbiome and the host immunity. Here, we will critical review the role of the lung microbiome in four common chronic lung diseases: COPD, bronchiectasis, IPF and Asthma.

直播室 C

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陳祐易 醫師 / You-YI Chen, M.D.

現職：臺大醫院雲林分院胸腔科主治醫師
專長：一般內科、胸腔內科、支氣管鏡檢查、
胸部超音波檢查、重症醫學

Brief Review and Case Sharing of Orphan ILD
少見之間質性肺病個案分享與文獻回顧

Rare interstitial lung diseases by their nature are difficult to diagnose. The healthcare burden of rare disease is often greater than that of more common diseases. Orphan diseases are those which are not widely researched, those where specific treatments are not available, and those which may only be of limited interest to scientists and doctors. Here we describe and briefly reviewed three different orphan interstitial lung disease including lymphangioleiomyomatosis, Langerhans-cell histiocytosis and pulmonary alveolar proteinosis, the research of these diseases has received much development during the past 10 years.

柯信國 醫師 / Hsin-Kuo Ko, M.D., Ph.D.

現職：臺北榮總胸腔部呼吸治療科主治醫師、臺北榮總胸腔加護病房 B 主任
專長：呼吸道疾病、間質性肺病、重症呼吸治療、高壓氧

Rapidly progressive interstitial lung disease (RP-ILD)
快速進展間質性肺纖維化疾病

Interstitial lung diseases (ILDs) are a group of rare respiratory, non-malignant disorders, characterized by varying degrees of damage to the lung parenchyma via inflammation and fibrosis. Patients with certain types of fibrosing interstitial lung disease (ILD) are at risk of developing a progressive phenotype characterized by self-sustaining fibrosis, decline in lung function, worsening quality of life, and early mortality. Terminology recently used to describe these patients with a progressive phenotype as "progressive-fibrosing ILD (PF-ILD)". Here this lecture will be focused on other ILDs that may present a progressive-fibrosing phenotype, namely idiopathic nonspecific interstitial pneumonia, unclassifiable idiopathic interstitial pneumonia, connective tissue disease-associated ILDs (e.g. rheumatoid arthritis-related ILD), fibrotic chronic hypersensitivity pneumonitis, fibrotic chronic sarcoidosis and ILDs related to other occupational exposures. Differential diagnosis of these ILDs can be challenging, and requires detailed consideration of clinical, radiological and histopathological features. Accurate and early diagnosis is crucial to ensure that patients are treated optimally. This lecture also discusses the current knowledge of acute exacerbation of ILDs that may present a progressive-fibrosing phenotype.

王才郁 醫師 / Tsai-Yu Wang, M.D.

現職：林口長庚胸腔內科助理教授級主治醫師
專長：阻塞型睡眠呼吸中止症、氣喘、慢阻肺、肺結核

OSA and COPD: the overlap syndrome
當阻塞型睡眠呼吸中止症遇上慢阻肺

Both COPD and OSA are highly prevalent diseases. The coexistence of both disorders, often referred to as the overlap syndrome, may occur based on chance association alone. However, the survival of patients with overlap syndrome that is not treated with nocturnal positive airway pressure is significantly inferior to that of patients with overlap syndrome that is appropriately treated. Therefore, the recognition of coexisting OSA in patients with COPD has important clinical relevance. The diagnosis of OSA in patients with COPD requires awareness of relevant clinical features, and screening questionnaires may help identify suitable patients for further overnight study.

Patients with COPD and exercise limitation will adopt a more sedentary lifestyle and give up the more strenuous physical activities, which eventually lead to deconditioning and ultimately further exercise limitation. Moreover, exercise limitation is associated with morbidity and mortality. CPAP treatment was associated with an increased walking capacity from baseline from 226.4 ± 95.3 m to 288.6 ± 94.6 m ($P < 0.05$), and decreased urinary catecholamine levels, pre-exercise heart rate, oxygenation, and Borg scale in the patients with overlap syndrome.

Osteoporosis, characterized by a decrease in bone mineral density (BMD), is reported to affect 9%–69% of patients with COPD, indicating that COPD patients have a high risk of developing osteoporosis. The BMD in those with OSA was significantly lower than in those without OSA (−1.99±1.63 versus −1.27±1.14, P=0.045). After multivariate linear regression analysis, the ODI was still an independent factor for BMD. In addition, smaller total lung capacity is significantly associated with higher ODI and lower BMD, which implies that lower BMD might cause severer OSA via decreased total lung capacity.

Some screening questionnaires such as Epworth sleepiness scale (ESS), sleep apnea clinical score (SACS), Berlin questionnaire (BQ), and STOP-BANG questionnaire (SBQ) are used in screening for OSA in patients with COPD. In predicting severe OSA (AHI ≥30), SBQ performed better than others with sensitivity, specificity, and AUC for SBQ >4, 66.1%, 82.1%, and 0.824 respectively. Our simplified screening questionnaire contains BMI, witnessed apnea, snoring and CAD. The sensitivity, specificity and AUC of our simplified screening questionnaire >3 are 70.8%, 90.2% and 0.80 respectively.

Patients with overlap syndrome had worsened exercise capacity, bone mineral density and survival. Screening questionnaires may help identify suitable patients for further overnight study.

蕭慈慧 醫師 / Tsu-Hui Shiao, M.D.

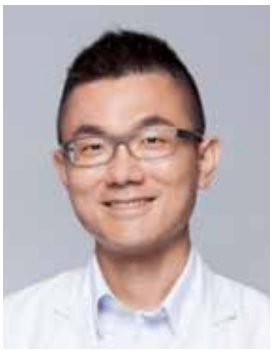
現職：台北榮民總醫院胸腔部主治醫師
專長：胸腔內科

Impulse Oscillometry to Predict the Severity of Obstructive Sleep Apnea
脈衝震盪肺功能預測阻塞性睡眠呼吸中止症的嚴重性

Obstructive sleep apnea (OSA) is a disorder with repetitive upper airway obstruction during sleep. Polysomnography is the standard diagnostic method of OSA but also time consuming and expensive. Impulse oscillometry(IOS) is a forced oscillation technique that measures pulmonary mechanics during tidal breathing. It has been used to study various respiratory disorders especially small airway disease. Since IOS has the capability to distinguish upper airway function from that of lower airways, the application of IOS to diagnosis and management of OSA would be a focus of interest in the field of sleep disordered breathing. Several studies had been conducted with using IOS measurements in OSA patients. In this brief talk, we will discuss the rationale on IOS measurements, previous data of IOS on OSA patients and its potential role on OSA screening.

曾健華 醫師 / Chien-Hua Tseng, M.D., Ph.D.

現職：雙和醫院重症醫學科主任
專長：胸腔重症醫學、流行病學、生物統計、呼吸道疾病

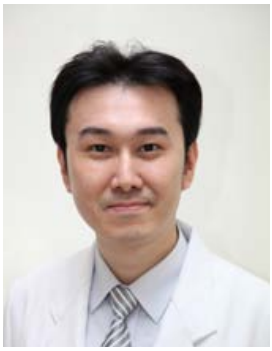


Indoor air pollution: an overview
概觀室內空氣汙染

Indoor air quality problems are problems with how clean the air we breathe is in buildings such as homes, schools, and workplaces. Poor or inadequate indoor air quality can cause breathing problems and other medical issues. Because we spend so much time indoors, including at work.

蘇一峰 醫師 / Yi-Fong Su, M.D.

現職：市立聯合陽明醫院胸腔內科主治醫師
專長：胸腔呼吸內科學、呼吸道疾病，胸腔重症醫學，肺癌胸腔腫瘤，呼吸道感染症，肺結核，呼吸睡眠疾病



E-cigarette and vaping: help or harm?
使用電子菸的好處與壞處

E-cigarettes are sometimes called "e-cigs," "vapes," and "electronic nicotine delivery systems (ENDS)." Some e-cigarettes look like regular cigarettes, cigars, or pipes. Some look like USB flash drives, pens, and other everyday items.

Most e-cigarettes contain nicotine, which is addictive and toxic to developing fetuses. E-cigarette aerosol can contain chemicals that are harmful to the lungs. And youth e-cigarette use is associated with the use of other tobacco products, including cigarettes. E-cigarettes are not currently approved by the FDA as a quit smoking aid.

In the United States, youth are more likely than adults to use e-cigarettes. In 2019, over 5 million U.S. middle and high school students used e-cigarettes in the past 30 days, including 10.5% of middle school students and 27.5% of high school students.

EVALI (e-cigarette or vaping product use associated lung injury) is an inflammatory response in the lungs triggered by inhaled substances. EVALI was initially recognized in the summer of 2019. As of February 18, 2020, a total of 2,807 hospitalized EVALI cases or deaths have been reported in the United States. The key risk factor for EVALI is use of an e-cigarette or similar product; no single constituent has been identified that is common to all cases. Until the exact mechanism of EVALI is known, it is reasonable to advise patients with EVALI to completely avoid vaping in the future.

孫文榮 主任 / Wen-Jung Sun, M.D., Ph.D.

現職：台北市立聯合醫院社區醫學部部主任 / 社區安寧發展中心主任 / 全觀心理健康中心主任 / 中興院區家庭醫學科主任
專長：社區醫學、家庭醫學、安寧緩和醫學、公共衛生學、流行病學、生物統計學、預立醫療照護諮商

Shared Decision Making in Patients with Terminal Lung Disease
末期肺疾患病人的醫病共享決策

The term "Shared Decision Making" (SDM) was first used in the United States' patient-centered care common well-being program in 1982 to promote the mutual respect and communication between medical staffs and patients. In 1997, Charles proposed an operational definition, it stats that both physician and patient must participate, the physician should propose a variety of empirical information on the disposition, the patient should propose the personal preferences and values, and all the above information shall be exchanged and discussed; thus, the most proper and the best treatment options can be found.

Shared Decision-making is a patient-centered clinical medical execution process, with knowledge, communication and respect, designed to enable medical personnel and patients to share existing empirical medical outcomes, combine with patients' own preferences and values, provide patients with all considerable options, and the involvement of clinicians and patients in medical care, reach consensus on medical decision-making, and finally support patients to make medical decisions that fit their preferences.

For patients with terminal lung disease, Shared Decision-making can improve mutual awareness of the entire care process, make the patients and their families to be more aware of the treatment options, and reduce medical disputes caused by poor communication. Therefore, clinical staff ought to learn this skill, and should further integrate it into their professional skills, in order to provide a true people-centered and holistic care.

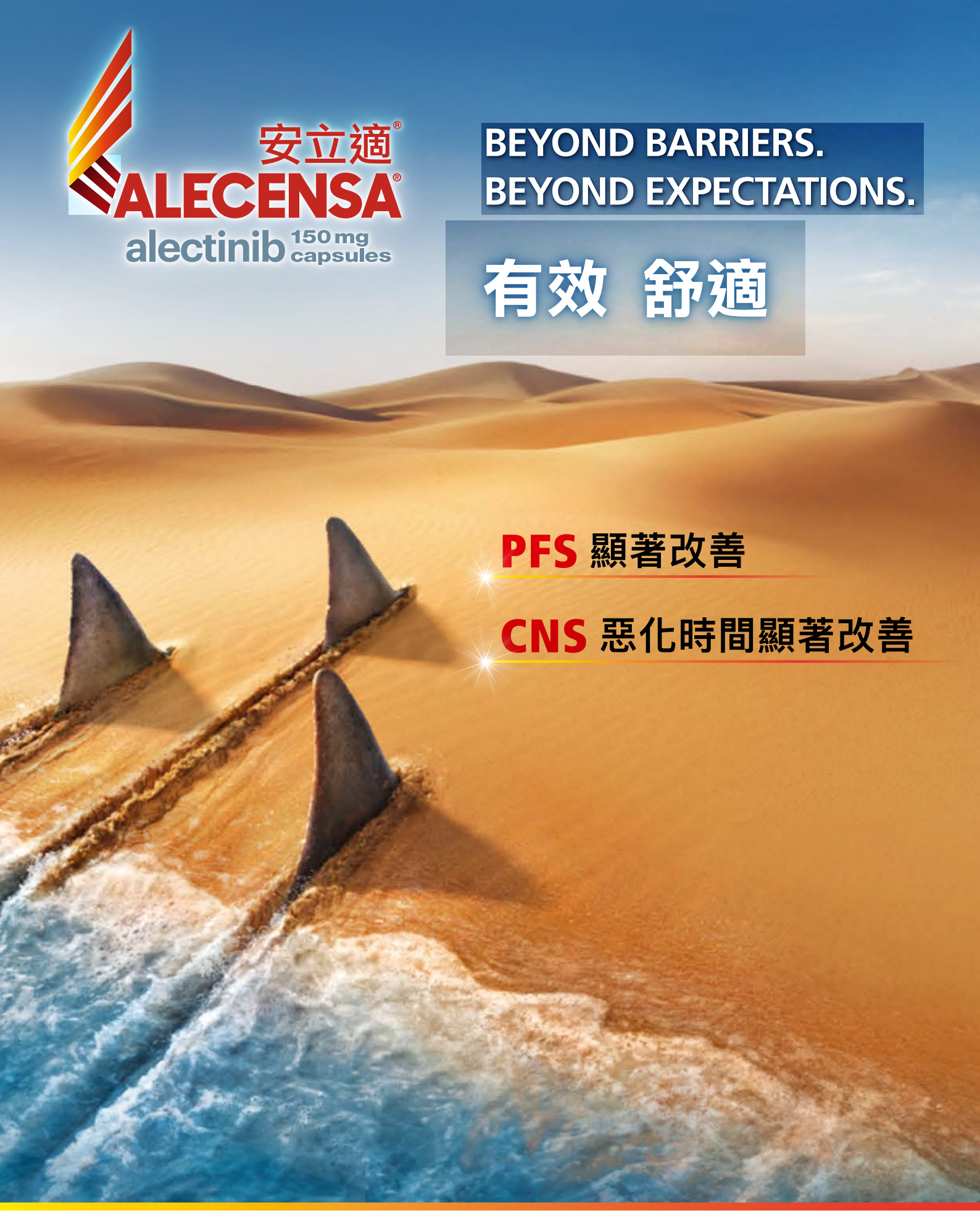
藍胄進 醫師 / Chou-Chin Lan, M.D., Ph.D.

現職：台北慈濟醫院胸腔科主任暨內科副部長
專長：胸腔復原運動、運動心肺功能、重症醫學

The role of pulmonary rehabilitation in palliative care
肺復原在緩和醫療中的角色

Patients with advanced pulmonary diseases often experience distressing physical and psychological symptoms, such as dyspnea, anxiety, fatigue and etc. They often suffered from poor quality of life and limited daily activity. These are strong indicators for expert multidisciplinary palliative care incorporating the management of symptoms. Pulmonary rehabilitation aims to improve health-related quality of life, increase exercise capacity and daily activity. Pulmonary rehabilitation encompasses tailored therapies that aim to help the physical and psychological health of the patients. It is therefore an important consideration for patients receiving palliative care. Early integration of palliative care with primary care, respiratory care and pulmonary rehabilitation, with referral based on the complexity of symptoms, rather than prognosis, can improve patient and carer outcomes. Combine pharmacological and non-pharmacological managements for patients with advanced pulmonary diseases are optimal strategy for increasing their quality of life and care outcomes.

This lecture will focus on non-pharmacological pulmonary rehabilitation for patients receiving palliative care and will address specific respiratory conditions of: chronic obstructive pulmonary disease (COPD), cancer, and interstitial lung disease.



ALECENSA® 處方說明

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警語：可能不良反應包括肝毒性、間質性肺病 (ILD) / 肺炎、腎功能不全、心搏徐緩、嚴重肌痛與肌酸磷酸激酶 (CPK) 升高、胚胎－胎兒毒性等。

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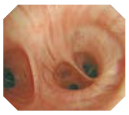

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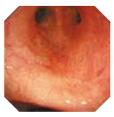

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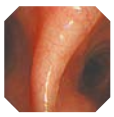

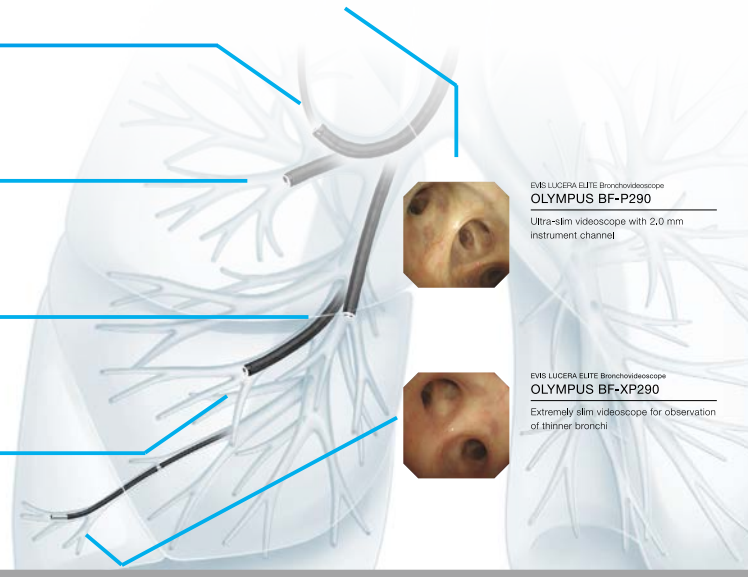

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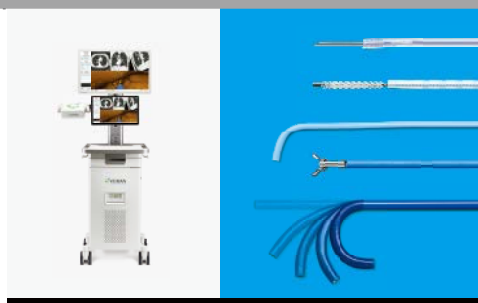

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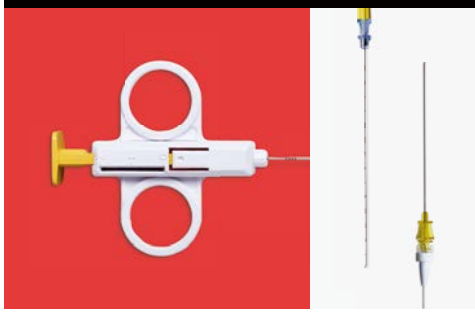

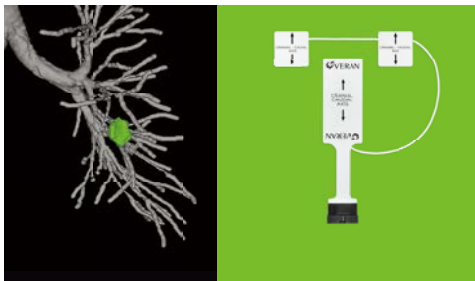

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靜脈注射劑 50 mg/mL

第一且唯一獲得核准於無法切除的第三期非小細胞肺癌病人

相較於安慰劑，經試驗證實，可延長無惡化存活期(PFS)多達三倍且降低三成以上死亡風險[1,3]

對象為局部晚期、無法手術切除的非小細胞肺癌，且接受放射治療合併含鉑化療後病情未惡化的病人[1]

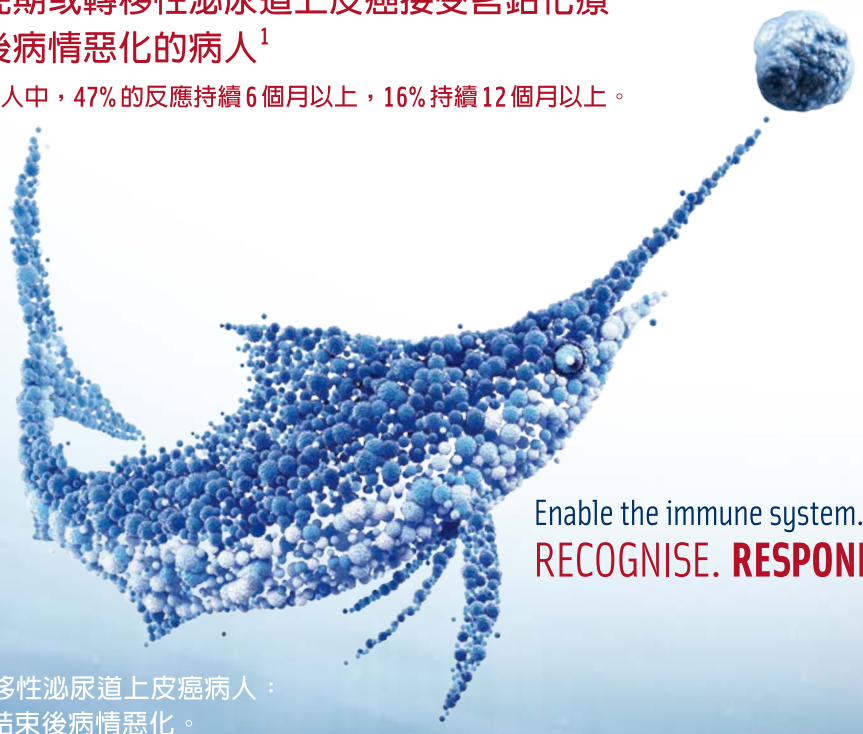
* PFS, stratified HR=0.52; 95% CI, 0.42-0.65; P<0.001 * OS, stratified HR=0.68, 99.73% CI, 0.47-0.997, P=0.0025

泌尿道上皮癌的治療新選擇

臨床試驗顯示可提供病人快速且持續反應的潛力[4]

對象為患有局部晚期或轉移性泌尿道上皮癌接受含鉑化療期間或治療結束後病情惡化的病人[1]

* 總計32名達到反應的病人中，47%的反應持續6個月以上，16%持續12個月以上。



Enable the immune system.
RECOGNISE. **RESPOND.**[2]

適應症

泌尿道上皮癌

治療下列患有局部晚期或轉移性泌尿道上皮癌病人：
- 接受含鉑化療期間或治療結束後病情惡化。
- 於使用含鉑化療進行術前輔助治療，或輔助治療12個月內病情惡化。

本適應症為根據腫瘤反應率及反應持續時間獲得加速核准，此適應症仍須執行確認性試驗以證明其臨床效益。

局部晚期非小細胞肺癌(NSCLC)

治療患有局部晚期、無法手術切除的非小細胞肺癌，且接受放射治療合併含鉑化療後病情未惡化的病人。

抑癌寧注射劑 IMFINZI Injection 50 mg/ml

【適應症】泌尿道上皮癌 治療下列患有局部晚期或轉移性泌尿道上皮癌病人：●接受含鉑化療期間或治療結束後病情惡化。●於使用含鉑化療進行術前輔助治療，或輔助治療12個月內病情惡化。本適應症為根據腫瘤反應率及反應持續時間獲得加速核准，此適應症仍須執行確認性試驗以證明其臨床效益。局部晚期非小細胞肺癌(NSCLC)治療患有局部晚期、無法手術切除的非小細胞肺癌，且接受放射治療合併含鉑化療後病情未惡化的病人。【用法用量】IMFINZI的建議劑量為每公斤體重10毫克，靜脈輸注60分鐘，每兩週一次，直至疾病惡化或發生無法耐受的毒性為止。說明：連續使用本品一年以上的利益風險平衡尚未確認。【禁忌】無。【警語及注意事項】●免疫介導性肺炎，IMFINZI可能引起免疫介導性肺炎，其定義為需要使用皮質類固醇者。已有致死病例之報導。●免疫介導性肝炎，IMFINZI可能引起免疫介導性肝炎，其定義為需要使用皮質類固醇者。已有致死病例之報導。●免疫介導性結腸炎，IMFINZI可能引起免疫介導性結腸炎，其定義為需要使用皮質類固醇者。●免疫介導性內分泌病變，IMFINZI可能引起免疫介導性內分泌病變，包括甲狀腺疾病、腎上腺功能不全、第一型糖尿病和腦下垂體炎/腦下垂體低能症。●免疫介導性腎炎，IMFINZI可能引起免疫介導性腎炎，其定義為有腎功能不全的證據，需要使用皮質類固醇者。已出現致死病例。●免疫介導性皮膚反應，IMFINZI可能引起免疫介導性皮疹：使用本類藥物其他產品曾經發生大皰性皮炎(bullous dermatitis)，史蒂文生－強生氏症候群(SJS)/毒性表皮溶解症(TEN)。●其他免疫介導性不良反應：IMFINZI可能引起嚴重和致死的免疫介導性不良反應。●感染，IMFINZI可能引起嚴重的感染，包括致死病例。●輸注相關反應，IMFINZI可能引起嚴重或危及生命的輸液相關反應。【常見不良反應】PACIFIC研究，最常見的不良反應(≥20%的病人發生)為咳嗽、疲勞、肺炎(pneumonitis)或放射性肺炎、上呼吸道感染、呼吸困難和皮疹。試驗1108，最常見的不良反應(≥15%)為倦怠(39%)、肌肉骨骼疼痛(24%)、便秘(21%)、食慾降低(19%)、噁心(16%)、周邊水腫(15%)、泌尿道感染(15%)。【使用前詳閱說明書警語及注意事項，詳細仿單資料備索】【僅限醫藥專業人員參考：處方藥物請參考衛生福利部核准仿單說明書】

Ref: 1. 衛福部核准仿單說明書 2. Stewart R, Morrow M, Hammond SA, et al. Identification and characterization of MEDI4736, an antagonistic anti-PD-L1 monoclonal antibody. *Cancer Immunol Res.* 2015;3(9):1052-1062. 3. Antonia et al. 2018 NEJM DOI: 10.1056/NEJMoa1809697. 4. Powles T, et al. 2017 JAMA Oncol.; 3 (9): e1-e10

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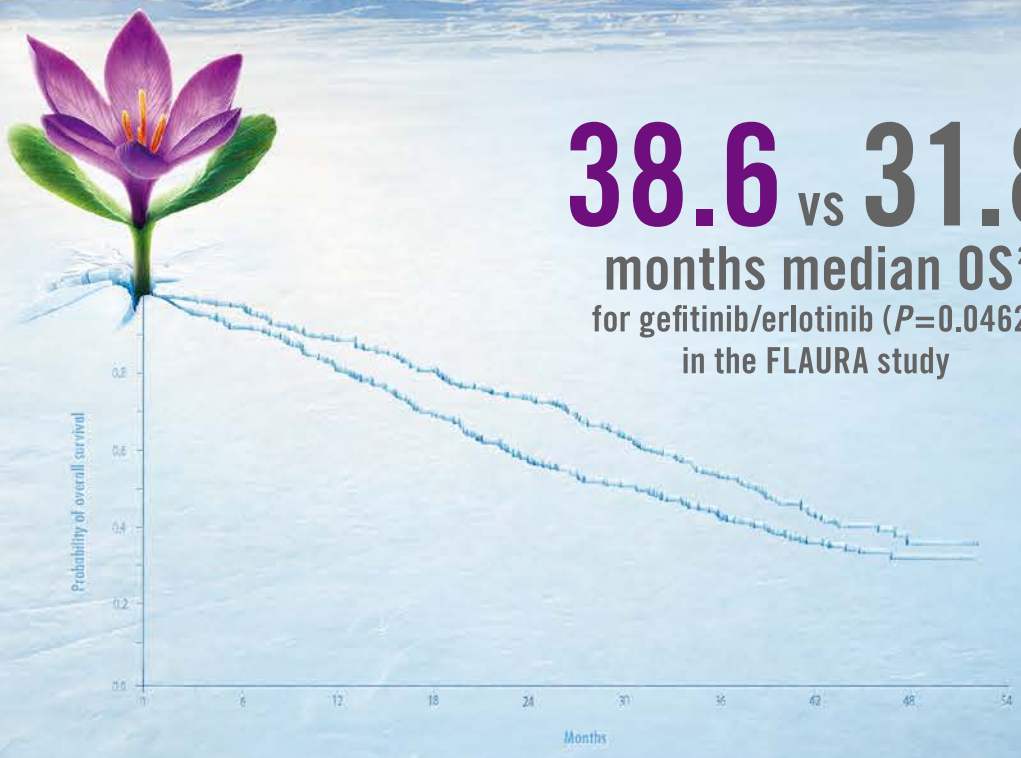

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REFERENCES:

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使肺泰125優氟吸入劑
使肺泰250優氟吸入劑
-適應症或用途：SERETIDE適用於可逆性呼吸道阻塞疾病(ROAD)之常規治療，包括適合使用支氣管擴張劑及皮質類固醇組合療法之患有氣喘的兒童與成人。這可能包括：正在使用長效乙型作用劑(β2-agonist)及吸入型皮質類固醇之有效維持劑量的患者。正在接受吸入型皮質類固醇療法，而仍有症狀之患者。接受支氣管擴張劑之常規治療，而需要吸入型皮質類固醇之患者。SERETIDE Accuhaler 250 及 Seretide Evohaler 125、250 另適用於嚴重慢性阻塞性肺部疾病(FEV1<50%預測值，FEV1/ FVC<70%)之維持治療，包括慢性支氣管炎和肺氣腫；SERETIDE Accuhaler 50/500 適用於成人及十二歲以上青少年之嚴重氣喘及中至重度(FEV1 < 60%)慢性阻塞性肺部疾病之維持性治療。

-建議劑量：
-使肺泰100/250/500準納乾粉吸入劑：
成人及十二歲以上之青少年：每日兩次，每次吸一單位劑量-使肺泰100準納乾粉吸入劑：
四歲以上之兒童：每日兩次，每次吸一下(50 mcg Salmeterol及100 mcg fluticasone propionate)。
-使肺泰50/125/250優氟吸入劑：
成人及十二歲以上之青少年：每日兩次，每次吸二單位劑量-使肺泰50優氟吸入劑：
四歲以上之兒童：每日兩次，每次吸二下(25 mcg Salmeterol及50 mcg fluticasone propionate)。
目前還沒有四歲以下幼兒使用SERETIDE的資料。
-特殊患者群：
老年患者，以及肝功能或腎功能不全之患者，使用SERETIDE時不需要調整劑量。
-投藥方式：口腔吸入劑

-成分：使肺泰準納乾粉吸入劑
50 mcg Salmeterol (as xinafoate)及100/250/500 mcg fluticasone propionate
使肺泰優氟吸入 劑：
25 mcg Salmeterol (as xinafoate)及50/125/250 mcg fluticasone propionate
-禁忌症：禁止使用於對本劑任何一種成分有過敏史之患者
-注意事項：甲狀腺毒症，低血鉀或糖尿病的病人使用前需事先告知醫師
-副作用：頭痛、肌肉痙攣、關節痛、口腔與喉嚨的念珠菌病、肺炎(慢性阻塞性肺部疾病患者)
-不良事件通報程序：通報電話：(02) 23126836/
郵箱：oax40892@gsk.com
詳細處方資訊備索
葛蘭素史克藥廠100台北市中正區忠孝西路一段66號24樓

警語：可能出現頭痛，關節痛或口腔及喉嚨念珠菌等副作用

PM-TW-FPS-EDTL-190007 Sept 2019

北市衛藥廣字第109010306號
僅限醫療專業人員使用

SERETIDE
salmeterol/fluticasone propionate

比樂舒活®錠
PIRESPA®
Pirfenidone 200mg Tablets

Help Hold IPF Disease Progression

PIRESPA® Tablets 200 mg
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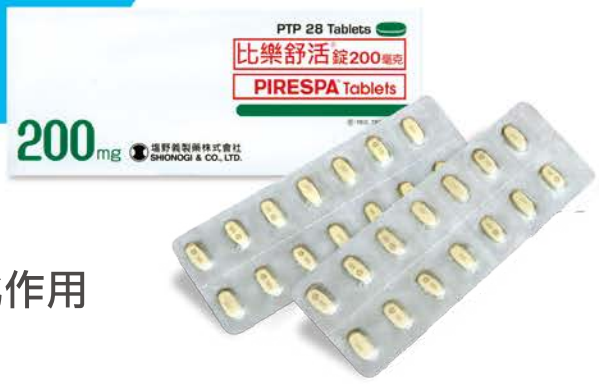

- 有效抑制肺活量下降
- 可抑制 TGF-β1, b-FGF, PDGF 等纖維化作用

使用前請詳閱說明書警語及注意事項，詳細處方資料備索

衛部藥輸字第026734號 健保碼:BC26734100
北市衛藥廣字第108090135號
PRS-ADV-1-TS-201909

SHIONOGI 台灣塩野義製藥

台灣塩野義製藥股份有限公司
地址：台北市南京東路二段2號4樓
電話：(02)2551-6336

TECENTRIQ®

atezolizumab Anti-PD-L1 癌症免疫療法

適應症：局部晚期或轉移性非小細胞肺癌[1]

1L：與 bevacizumab、paclitaxel 和 carboplatin 併用，做為轉移性，不具有 EGFR 或 ALK 腫瘤基因異常之非鱗狀非小細胞肺癌的第一線治療藥物。

2L：單獨使用，適用於治療接受含鉑化學治療後，疾病惡化之局部晚期或轉移性非小細胞肺癌患者。患者若具有 EGFR 或 ALK 腫瘤基因異常，則須先經 EGFR 或 ALK 抑制劑治療，若治療後疾病惡化方可使用 Tecentriq。

EGFR=Epidermal growth factor receptor; ALK=Anaplastic lymphoma kinase; PD-L1=Programmed death-ligand 1. DOR=Duration of response; HR=Hazard ratio.

References: 1. TECENTRIQ® (atezolizumab) 藥品仿單 (衛部菌疫輸字第 001050 號).

羅氏大藥廠股份有限公司
台北市信義區松仁路100號40樓
電話：(02)2713-0630，2715-3111
網址：http://www.roche.com.tw/

衛部菌疫輸字第 001050 號
本藥限由醫師使用
使用前詳閱說明書警語及注意事項
北市衛藥廣字第　　號

PM-TW-2094-03-2019

Roche

雙靶箭合璧 握希望

AVASTIN® 併用 Tarceva® 可作為 EGFR 突變的非小細胞肺癌 第一線治療

適應症：
轉移性大腸直腸癌(mCRC): 1) 與5-fluorouracil化學療法合併使用可以作為第一線治療。2) 與5-fluorouracil/leucovorin/oxaliplatin化學療法合併使用可以作為先前接受過以fluoropyrimidine為基礎的化學療法無效，且未曾接受過Avastin治療的病人的治療。3) 與含有fluoropyrimidine-irinotecan-或fluoropyrimidineoxaliplatin-為基礎的化學療法合併使用，可以做為第一線已接受過以Avastin併用化療後惡化之轉移性大腸或直腸癌病人的第二線治療。[不適用於高風險的二期以及三期大腸癌輔助性療法]
轉移性乳癌(mBC): 與paclitaxel合併使用可以做為HER2(-)轉移性乳癌病人的第一線治療。[不適用於anthracycline及taxane治療轉移性乳癌又出現疾病進展的病人。]
惡性神經膠質瘤(WHO第4級) - 神經膠母細胞瘤: 單獲使用可用於治療曾接受標準放射線治療，且含Temozolomide在內之化學藥物治療失敗之多型性神經膠母細胞瘤復發之成人病人。
晚期、轉移性或復發性非鱗狀非小細胞肺癌(NSCLC): 1) 與carboplatin及paclitaxel合併使用可以作為第一線治療。2) 併用erlotinib可作為EGFR活化性突變的非鱗狀非小細胞肺癌病人的第一線治療。
卵巢上皮細胞、輸卵管或原發性腹膜癌(Epithelial Ovarian, Fallopian Tube or Primary Peritoneal Cancer): 1) 與carboplatin及paclitaxel合併使用後著著單獨使用Avastin，可以做為第三期或第四期病人接受初次手術切除後之治療。2) 與carboplatin及gemcitabine合併使用，可以做為曾接受過第一線含鉑類藥物化學治療間隔至少6個月再復發，且未曾接受過bevacizumab或VEGF抑制劑或VEGF receptor-targeted agents治療之復發性病人之治療。3) 與carboplatin及paclitaxel合併使用，接著單獨使用Avastin治療，可以做為對含鉑藥物具感受性之復發性病人的治療。4) 併用paclitaxel、topotecan或pegylated liposomal doxorubicin可以做為接受過含鉑類藥物化療治療後6個月內再復發、之前接受不超過2種化療療程且未曾接受過bevacizumab或VEGF抑制劑或VEGF receptor-targeted agents治療之病人的治療。
持續性、復發性或轉移性之子宮頸癌(Persistent, Recurrent, or MetastaticCervical Cancer): 1) 與paclitaxel及cisplatin合併使用。2) 與paclitaxel及topotecan合併使用，可用於無法接受含鉑類藥物治療之病人。

使用劑量：
轉移性大腸直腸癌(mCRC): 1) 第一線治療：5毫克/公斤(體重)，每兩週一次；或7.5毫克/公斤(體重)，每三週一次。2) 第二線治療：10毫克/公斤(體重)，每兩週一次；15毫克/公斤(體重)，每三週一次。3) 當用於治療第一線已接受過以Avastin的治療法後惡化的第二線治療，應與含有fluoropyrimidine-irinotecan-或fluoropyrimidine-oxaliplatin-為基礎的化學療法合併使用，投與Avastin 5毫克/公斤(體重)，每兩週一次或7.5毫克/公斤(體重)，每三週一次。
轉移性乳癌(mBC): 靜脈輸注給予10毫克/公斤(體重)，每兩週一次。
惡性神經膠質瘤(WHO第4級) - 神經膠母細胞瘤: 靜脈輸注給予10毫克/公斤(體重)，每兩週一次；或15毫克/公斤(體重)，每三週一次。
晚期、轉移性或復發性非鱗狀非小細胞肺癌(NSCLC): 1) 合併使用含鉑類化學療法的第一線治療：靜脈輸注15毫克/公斤(體重)，每三週一次。合併使用鉑類化學療法六個治療週期，接著單獨使用Avastin治療，直到疾病惡化為止。2) 合併使用erlotinib：靜脈輸注15毫克/公斤(體重)，每三週一次。建議應持續以Avastin併用erlotinib治療至潛在疾病發生惡化為止。
卵巢上皮細胞、輸卵管或原發性腹膜癌(Epithelial Ovarian, Fallopian Tube or Primary Peritoneal Cancer): 1) 第三期或第四期疾病初次手術切除後之治療：靜脈輸注給予15毫克/公斤 (體重)，每三週一次。Avastin與carboplatin及paclitaxel合併使用至多六個治療週期，接著單獨使用Avastin治療直到疾病惡化為止。卸或。Avastin與carboplatin及gemcitabine合併使用六個治療週期，最多用到十個治療週期，接著單獨使用Avastin治療直到疾病惡化為止。對鉑類化療具抗藥性：與paclitaxel、topotecan (每週投予)或pegylated liposomal doxorubicin任一藥劑併用時Avastin投與劑量為10毫克/公斤(體重)，每2週1次以IV輸注。若topotecan為每3週的第1至5天投予時，與其併用之Avastin投與劑量為15毫克/公斤(體重)，每3週1次以IV輸注。建議持續治療直到疾病惡化或無法忍受的毒性產生為止。
子宮頸癌(Cervical Cancer)：可與下列其中一種化學治療方式併用：Paclitaxel加上cisplatin或paclitaxel加上topotecan。建議劑量為15毫克/每公斤(體重)，每三週給藥一次，以靜脈輸注方式給藥。

使用方法：
用於靜脈(i.v.)輸注之透明至稍微乳白、無色至淡棕色的無菌溶液。調配後供輸注之溶液。Avastin並非用於眼球玻璃體內之配方。

禁忌症：
已知會對下列東西過敏的病人禁止使用Avastin：1) 本產品的任何成份。2) 中國倉鼠卵巢細胞製劑或其他基因重組之人類或人化的抗體。

警語：
胃腸穿孔、外科手術和傷口癒合的併發症及出血胃腸穿孔：使用Avastin治療的病人發生胃腸穿孔(有些是致命的)的發生率為0.3–3.2%。發生胃腸穿孔應停止使用Avastin。外科手術和傷口癒合的併發症：在使用Avastin治療的病人有較高傷口癒合和外科手術併發症的發生率，包括嚴重和致命的併發症的發生率。病人有傷口裂開現象時應停止使用Avastin。目前仍未知為避免減弱傷口癒合能力並減少傷口裂開的風險所需停用Avastin至進行選擇性手術的適當時間間隔。在進行選擇性手術前至少28天，應暫停使用Avastin。在手術後至少28天且手術傷口完全癒合後再開始進行Avastin的治療。出血：在使用Avastin的病人發生嚴重或致命的出血(包括咳血、胃腸出血、神經系統出血、鼻出血和陰道出血)較頻繁(最高達5倍)。對於有嚴重出血或最近曾發生咳血的病人，不可投予Avastin來治療。

副作用：
出血：在針對各種不同適應症的所有臨床試驗中，所有以Avastin治療的病人有0.4%至6.9%發生NCI-CTC 3級至5級出血事件，而化學療法對照組病人則僅有0至4.5%的發生率。在Avastin臨床試驗中所出現的出血事件主要是腫瘤相關的出血及輕微的黏膜皮膚出血 (如鼻出血)。
高血壓：以Avastin治療的病人，其整體高血壓(所有級別)的發生率達42.1%，相較於對照組達14%。以Avastin治療的病人其NCI-CTC 3級和4級高血壓的整體發生率，為0.4%到17.9%。第4級高血壓(高血壓危象)的發生率，在以Avastin治療的病人中達1.0%，而單獨使用相同化學療法的病人達0.2%。高血壓一般都是以口服降血壓藥物予以適當的控制，例如血管收縮素轉換酶抑制劑，利尿劑及鈣離子通道阻斷劑，很少會造成Avastin停藥或住院。
蛋白尿：在臨床試驗中，有0.7%到54.7%接受Avastin治療的病人曾有蛋白尿的報告。蛋白尿的嚴重程度由無臨床症狀、短暫性、輕微的蛋白尿到腎病症候群都有。8.1%的治療組病人發生3級蛋白尿。4級蛋白尿(腎病症候群)發生在治療病人中達1.4%。有高血壓病史的病人在以Avastin治療時，發生蛋白尿的危險性較高。有證據顯示1級蛋白尿可能和Avastin的劑量有關。建議在以Avastin治療前進行蛋白尿的檢驗。在大多數的研究中，尿蛋白值≥2公克/24小時會導致Avastin的停用，直到恢復到<2公克/24小時為止。
過敏、輸注反應：在一些臨床試驗中，相較於單獨使用化學療法的病人，使用Avastin併用化學療法的病人較常有過敏性(anaphylactic)及類過敏性(anaphylactoidtype)反應的報告。Avastin在一些臨床試驗中這類反應的發生率是常見的(bevacizumab治療之病人的發生率達5%)。

衛署菌疫輸字第000807號

適應症:
Tarceva適用於具有EGFR-TK突變之局部侵犯性或轉移性之非小細胞肺癌(NSCLC)病患之第一線及維持治療，或先前已接受過化學治療後，但仍局部惡化或轉移之肺腺癌病患之第二線用藥。

使用劑量及使用方法:
須先接受EGFR突變檢測，每日建議劑量為150毫克，並應於進食前至少1小時或進食後2小時服用。
病人應持續接受治療，直到出現疾病惡化的現象或無法接受的毒性為止。

禁忌症:
無。

警語及不良反應:
肺毒性：在使用Tarceva治療NSCLC或其它晚期實體腫瘤的病人中，曾有發生嚴重類間質性肺病事件(ILD-like events) (包括死亡)的病例報告，但並不常見。

肝毒性：使用Tarceva期間，曾有肝衰竭及肝腎症候群(包括死亡)的病例報告，尤其在基準期有肝功能受損的病人。因此，建議定期進行肝功能檢測(轉胺酶、膽紅素及鹼性磷酸酶)。

腎衰竭：曾有肝腎症候群、急性腎衰竭(包括死亡)及腎功能不全的報告，建議定期監測可能產生脫水的病人之腎功能和血清電解質。

腸胃穿孔：曾有使用Tarceva病人胃腸穿孔(包括死亡)的報告。併用抗血管新生劑、皮質類固醇、NSAIDs、及/或含taxane的化學療法的病人，或有胃潰瘍或腎室疾病病史的病人的風險較高。對於產生胃腸穿孔的病人，應永久停用Tarceva。

大水泡性及脫皮性皮膚疾病：曾有大水泡性、發泡性及脫皮性皮膚疾病的報告，包括可能是Stevens-Johnson syndrome /毒性表皮壞死溶解症(toxic epidermal necrolysis)的病例，在某些病例中是致死的。對於產生嚴重大水泡性、發泡性及脫皮性皮膚疾病的病人，應中斷或停止使用Tarceva治療。

眼睛疾病：曾有在使用Tarceva時發生角膜穿孔及潰瘍的報告。

衛署菌疫輸字第025071號

北市衛藥廣字第109010145號
使用前請詳閱說明書警語及注意事項
PM-TW-3456-12-2019

羅氏大藥廠股份有限公司
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PM-TW-2216-03-2019

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+

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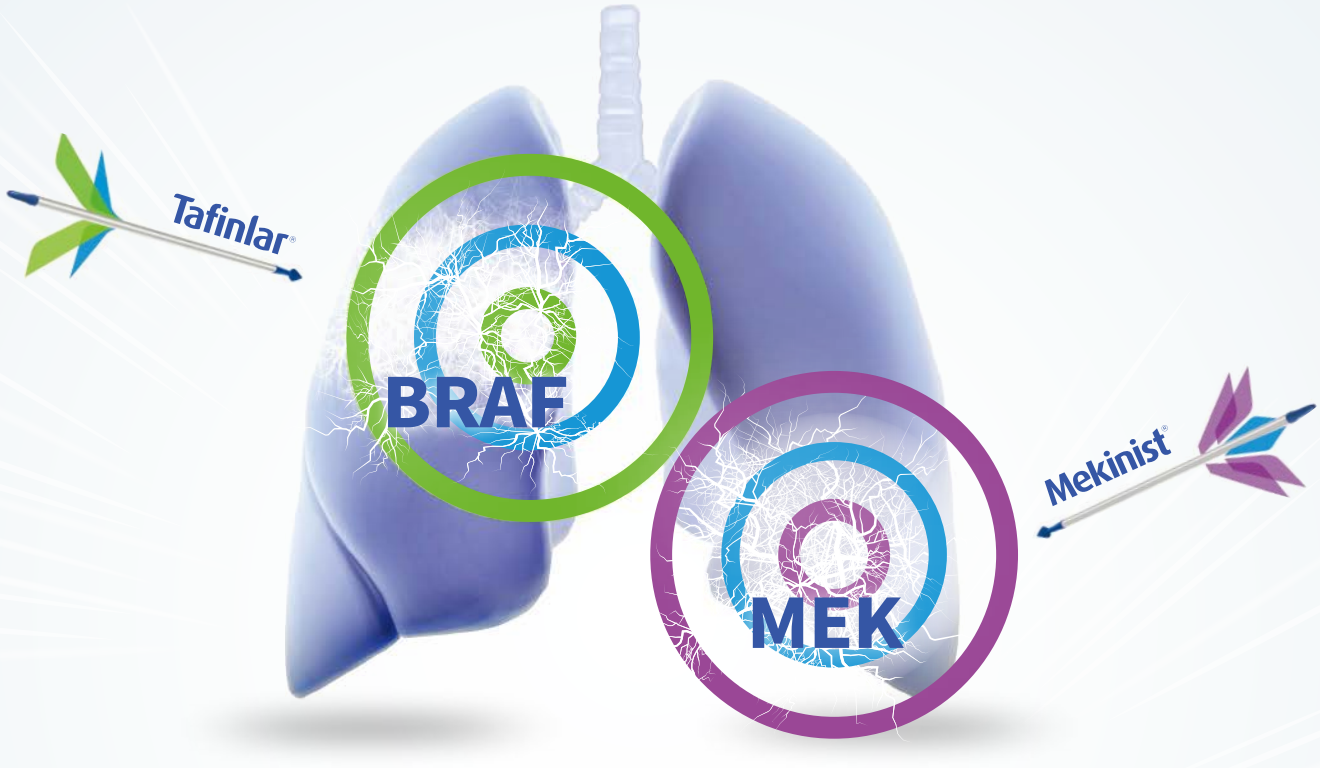
針對 BRAF V600 突變之晚期非小細胞肺癌的

雙標靶治療



Identify the Target . Take Action.

泰伏樂® 膠囊 50 毫克 TAFINLAR® Capsules 50 mg 衛部藥輸字第 026578 號
泰伏樂® 膠囊 75 毫克 TAFINLAR® Capsules 75 mg 衛部藥輸字第 026579 號
【適應症】黑色素瘤：Dabrafenib 單一療法或與 trametinib 併用，可用於治療 BRAF V600 突變陽性且無法以手術切除或轉移性的成人黑色素瘤。黑色素瘤的輔助治療：Dabrafenib 與 trametinib 併用，可用於治療 BRAF V600 突變且完全切除後之第 III 期黑色素瘤病人的術後輔助治療。非小細胞肺癌：Dabrafenib 與 trametinib 併用，可用於治療 BRAF V600 突變之晚期非小細胞肺癌成人病人。**【使用方法】**使用 dabrafenib 之前，必須先經過確效的檢測方法確認病人的腫瘤發生 BRAF V600 突變。應持續治療至病人無法再獲得效益或出現無法接受的毒性反應為止。如果漏服一劑 dabrafenib，且距離服用下一劑的時間不到 6 小時，則不可補服該劑藥物。當 dabrafenib 與 trametinib 併用時，若漏服一劑 trametinib，只有在距離下一次 trametinib 服藥時間超過 12 小時的情況下，方可服用該劑 trametinib。**【劑量】**Dabrafenib 不論單一療法或與 trametinib 併用，建議劑量皆為 150 毫克 (兩顆 75 毫克膠囊) 每日兩次 (相當於每日總劑量 300 毫克)。Dabrafenib 應於餐前至少 1 小時或餐後至少 2 小時服用，兩劑之間並應間隔 12 小時左右。Dabrafenib 應於每天的相同時間服用，以提高病人的順從性。當與 dabrafenib 併用，trametinib 的建議劑量為 2 毫克每日一次(QD)。**【禁忌】**對活性成分或任何賦形劑過敏。賦形劑: Microcrystalline cellulose，Magnesium stearate，Colloidal silicon dioxide，Hypromellose Capsules Size 2 Swedish Orange (50 毫克)，Hypromellose Capsules Size 1 Pink (75 毫克)，Hypromellose Capsules 含有 Red iron oxide，Titanium dioxide，Hypromellose，Water。**【警語】**在使用 BRAF 抑制劑時惡化的黑色素瘤病人中，併用 dabrafenib 與 trametinib，資料顯示療效在這類病人中較低。過去未曾在 BRAF V600 突變陽性黑色素瘤已轉移至腦部的病人中，評估 trametinib 與 dabrafenib 併用療法的安全性及療效。新發生惡性腫瘤。新發生表皮鱗狀細胞癌 (cuSCC)。新發生原發性黑色素瘤。新發生非皮膚性惡性腫瘤。出血。視力損傷。發燒。LVEF 降低 / 左心室功能異常。腎衰竭。肝臟反應。高血壓。間質性肺病(ILD)/ 肺炎。紅疹。橫紋肌溶解症。胰臟炎。深部靜脈栓塞(DVT)/ 肺栓塞 (PE)。胃腸道異常。Dabrafenib 為 CYP2C8 與 CYP3A4 的作用受質，應盡可能避免使用這些酵素的強效誘導劑。會升高胃中 pH 值的藥物可能會降低 dabrafenib 的生體可用率，因此應盡可能避免使用。Dabrafenib 是一種可能會使許多常用藥物療效喪失的代謝酵素誘導劑，因此，在開始使用 dabrafenib 治療時，一定要進行藥物使用審查(DUR)。**【極常見副作用】**Dabrafenib 單一使用：乳突瘤，食慾降低，頭痛，咳嗽，噁心，嘔吐，腹瀉，皮膚角化過度，禿髮，皮疹，掌蹠紅斑觸痛症候群 (Palmar-plantar erythrodysaesthesia syndrome)，關節痛，肌痛，四肢疼痛，發燒，疲倦，發冷，無力。Dabrafenib，Trametinib 合併使用：泌尿道感染，鼻咽炎，嗜中性球減少，食慾降低，頭痛，暈眩，高血壓，出血，咳嗽，腹痛，便祕，腹瀉，噁心，嘔吐，皮膚乾燥，搔癢，紅疹，紅斑，痤瘡性皮膚炎，關節痛，肌痛，四肢疼痛，肌肉痙攣，疲倦，發冷，無力，周邊水腫，發燒，類流感疾病，丙胺酸轉胺酶升高，天冬胺酸轉胺酶升高。
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麥欣寬® 膜衣錠 0.5 毫克 Mekinist® Film-Coated Tablets 0.5mg 衛部藥輸字第 026817 號
麥欣寬® 膜衣錠 2 毫克 Mekinist® Film-Coated Tablets 2 mg 衛部藥輸字第 026818 號
【適應症】黑色素瘤：Trametinib 單一療法或與 dabrafenib 併用，可用於治療罹患發生 BRAF V600 突變且無法切除或有轉移現象之成人黑色素瘤。在先前接受 BRAF 抑制劑療法時惡化的病人中，trametinib 單一療法並未展現出臨床活性。黑色素瘤的輔助治療：Trametinib 與 dabrafenib 併用，可用於治療 BRAF V600 突變且經完全切除後之第 III 期黑色素瘤病人的術後輔助治療。非小細胞肺癌：Dabrafenib 與 trametinib 併用，可用於治療 BRAF V600 突變之晚期非小細胞肺癌成人病人。**【使用方法】**使用 trametinib 之前，病人必須先接受已確效的檢測方法，以確認帶有 BRAF V600 突變。一般建議病人應持續接受 trametinib 的治療，直到病人無法再獲得效益或出現無法接受的毒性反應為止。**【劑量】**Trametinib 不論單一療法或與 dabrafenib 併用，建議劑量皆為 2 毫克每日一次。當與 trametinib 併用，dabrafenib 的建議劑量為 150 毫克每日兩次。**【禁忌】**對活性物質或任一賦形劑過敏者：Mannitol，Microcrystalline cellulose，Hypromellose 2910，Croscarmellose sodium，Magnesium stearate，Sodium lauryl sulfate，Colloidal silicon dioxide，Opadry Yellow 03B120006，Opadry Pink YS-1-14762-A【警語】黑色素瘤為 BRAF V600 突變檢測陰性的病人使用 trametinib 的安全性及療效未曾被評估。依據跨試驗比較的結果，整體存活期及無惡化存活期資料似乎在 trametinib 與 BRAF 抑制劑之間展現出相近的有效性，不過整體反應率在接受 trametinib 治療的病人中，比接受 BRAF 抑制劑治療的病人來得低。在曾經使用 BRAF 抑制劑時惡化的病人中， 併用 trametinib 與 dabrafenib，資料顯示併用療法的療效在這類病人中較低。過去未曾在 BRAF V600 突變陽性黑色素瘤已轉移至腦部的病人中，評估 trametinib 與 dabrafenib 併用療法的安全性及療效。當 trametinib 與 dabrafenib 併用時，有可能發生新的皮膚或非皮膚惡性腫瘤，皮膚鱗狀上皮細胞癌 (cuSCC)，新發生的原發性黑色素瘤。在 RAS 突變存在的情況下，使用 dabrafenib 治療可能會導致發生非皮膚性惡性腫瘤的風險升高。對於發生 RAS 突變陽性之惡性腫瘤病人，當 trametinib 與 dabrafenib 合併使用時，trametinib 不須因此調整劑量。出血。LVEF 下降 / 左心室功能障礙。發燒。高血壓。間質性肺病(ILD)/ 肺炎。視覺障礙。皮疹。橫紋肌溶解症。腎衰竭。胰臟炎。QT 間期延長。肝臟相關事件。肝功能不全。深層靜脈血栓 (DVT)/ 肺栓塞 (PE)。胃腸異常。**【極常見副作用】**Trametinib 單一治療：高血壓，出血，咳嗽，呼吸困難，腹瀉，噁心，嘔吐，便祕，腹痛，口乾，皮疹，痤瘡樣皮膚炎，皮膚乾燥，皮膚搔癢，掉髮，疲倦，週邊水腫，發熱，天門冬胺酸轉胺酶增加。Dabrafenib 與 Trametinib 並用：泌尿道感染，咽喉炎，鼻咽炎，嗜中性球減少，食慾降低，頭痛，暈眩，高血壓，出血，咳嗽，腹痛，便祕，腹瀉，噁心，嘔吐，丙胺酸轉胺酶上升(ALT)，天門冬胺酸轉胺酶上升 (AST)，皮膚乾燥，搔癢，紅疹，紅斑，痤瘡性皮膚炎，關節痛，肌痛，四肢疼痛，肌肉痙攣，疲倦，發冷，無力，周邊水腫，發燒，類流感疾病。【衛福部食藥署網站】https://www.fda.gov.tw
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NOVARTIS
台灣諾華股份有限公司
10480台北市中山區民生東路3段2號8樓
http://www.novartis.com.tw
電話：(02)2322-7777
傳真：(02)2322-7993
免費諮詢專線
0800-880-870

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僅供醫療專業人員使用

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台灣胸腔暨重症加護醫學會

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