### **ORIGINAL ARTICLE**

## Caseload volume-outcome relation for pulmonary embolism treatment: association between physician and hospital caseload volume and 30-day mortality

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Summary. Objective: This study sets out to examine the association between physician and hospital pulmonary embolism (PE) caseload volume and subsequent patient outcomes using 3-year nationwide population-based data in Taiwan. Method: This study used claims data from the 2002-2004 National Health Insurance Research Database. The sample of 2761 PE inpatients was divided into three physician caseload volume groups, <3 cases (low volume), 3–6 cases (medium volume) and  $\geq$ 7 cases (high volume), while the three hospital volume groups were <42 cases (low volume), 42-110 cases (medium volume) and  $\geq 111$  cases (high volume). A conditional logistic regression model was performed to evaluate the effects of caseload volume on 30-day mortality for PE treatment. Results: Patients treated by low case volume physicians had significantly higher mortality rates than those treated by medium case volume (19.0% vs. 13.3%, P < 0.001) or high case volume physicians (19.0% vs. 8.4%, P < 0.001). However, no significant relationship was observed between 30-day morality and hospital caseload volume (P = 0.697). The regression shows that the adjusted odds of 30-day mortality among patients of low case volume physicians were over twice the mortality odds among patients of high case volume physicians (OR = 2.164, P < 0.001), and odds ratios were 1.401 relative to medium case volume physicians' patients (P < 0.05). Conclusion: We conclude that an inverse PE volume-outcome relationship does exist for physicians, but not for hospitals. The skill or experience of an individual physician is a more critical factor than hospital equipment, infrastructure or staffing team in determining PE patient outcomes.

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#### Introduction

Pulmonary embolism (PE) is a highly lethal condition. In the US, the estimated case fatality rate remained as high as 7.7 deaths per 100 patients in 1998 [1] and accounts for 60 000 deaths annually [2], although mortality from PE has decreased significantly during the past two decades [3–6]. PE treatment usually involves anticoagulant medication (such as heparin, low-molecular-weight heparin, and warfarin), and rarely (in severe cases) thrombolysis or surgery. While anticoagulant therapy is considered the mainstay of PE treatment, the treatment prognosis from all methods combined may still depend on a provider's experience, prompt diagnosis and treatment and the size and location of the clot [7,8].

Numerous volume-outcome studies have been conducted on various surgical procedures or medical conditions since the first such study reported by Luft *et al.* in 1979 [9]. The overwhelming majority of these reported that patients treated by providers with higher caseloads had superior treatment outcomes [10]. To our best knowledge, only one study by Aujesky *et al.* has focused on patient outcomes and the number of PE patients treated by particular hospitals, using discharge records from Pennsylvania hospitals [7]. So far no study has sought to examine the simultaneous contribution to patient outcomes of both hospital and physician caseload levels.

Therefore, this study sets out to examine the association between physician and hospital PE caseload volume and subsequent patient outcomes in Taiwan using a 3-year nationwide population-based data set.

### **Research methods**

### Data base

This study used claims data from the 2002–2004 National Health Insurance Research Database (NHIRD) published in

Taiwan by the National Health Research Institute. The NHIRD includes data on every inpatient admission for NHI beneficiaries, over 21 million people (96% of Taiwan's population). Taiwan's NHI provides universal coverage to all citizens, a single plan with generous benefits, low copayments, and free choice of a widely-dispersed network of healthcare providers. Although there are no documented sensitivity and specificity studies for coding accuracy, it is generally believed that the NHI's checks and balances foster accurate coding. Hundreds of studies have been published based on this data set.

The NHIRD was then linked to the 'cause of death' data file with the assistance of the Department of Health (DOH) in Taiwan in order to determine 30-day mortality after PE presentation.

### Study sample

Each claim record in the NHIRD provides ICD-9CM codes for one principal procedure, one principal diagnosis, and up to four secondary diagnoses. First, we selected patients hospitalized with a principal diagnosis of pulmonary embolism (ICD-9-CM codes 415.1, 415.11, 415.19 and 673.20-673.24). In order to be sure to include all cases hospitalized for pulmonary embolism treatment, we also selected those with a secondary diagnosis of pulmonary embolism and one of the following principal diagnoses, in accordance with prior studies [7]: respiratory failure (518.81), cardiogenic shock (785.51), cardiac arrest (427.5), secondary pulmonary hypertension (416.8), syncope (780.2), thrombolysis (99.10), and intubation or mechanical ventilation (96.04, 96.05, 96.70-96.72). As those conditions might be related to or even immediate complications of treatment procedures for pulmonary embolism, they may appear as the principal diagnosis in the claim record, with a secondary diagnosis of pulmonary embolism being recorded at discharge. Furthermore, we excluded readmissions in order to limit our study sample to first-time admissions. Ultimately, a total of 2761 patients were included in this study.

# Classification of patients by their physician's and hospital's case volume

Using attending physician and hospital identifiers in the claims, we identified when the same physician or hospital admitted one or more patients for pulmonary embolism treatment during our 3-year study period. Physicians were sorted in ascending order of volume, and volume cut-off points were determined such that the sampled patients were classified into three, approximately equal-sized groups, according to standard practise [11,12]. The sample of 2761 patients was divided into three physician volume groups: <3 cases (hereafter referred to as low volume), 3–6 cases (medium volume) and  $\geq$ 7 cases (high volume), while the three hospital volume groups were <42 cases (low volume), 42–110 cases (medium volume) and  $\geq$ 111 cases (high volume).

### Statistical analysis

The SAS package (Version 9.1; SAS Institute Inc., Cary, NC, USA) was used. The key independent variable of interest was physician caseload volume, while the key dependent variable was '30-day mortality', with 'patient' as the unit of analysis. The outcome measure was dichotomous, irrespective of whether or not pulmonary embolism treatment resulted in 30-day mortality.

We carried out a conditional (fixed-effect) logistic regression model in which observations are conditional on hospitals, in order to partition out systematic hospital-specific variation. This model essentially evaluates the effects of physician-volume on patient-outcomes within each hospital and then averages these effects across hospitals, an approach that eliminates the possibility of confounding by hospital. In addition, this conditional model also uses a clustered method for variance estimation to account for the possibility that patients of each physician have more similar outcomes than patients viewed across physicians.

We adjusted for physician's gender, age, practise location (urban vs. rural), the hospital's accreditation level, patient demographics (age and gender) and patient comorbidities. The hospital accreditation level variable, used as a proxy for both hospital size and clinical service capabilities, classified each hospital as a medical center (with a minimum of 500 beds), a regional hospital (minimum 250 beds) or a district hospital (minimum 20 beds). We adjusted for patients' comorbidites using the Elixhauser Comorbidity Index, which was created in 1997 and has been widely used for risk adjustment in administrative data sets [13-15]. The Elixhauser method of comorbidity measurement uses 30 binary (1 = present and0 = absent) comorbidity measures to account for inpatient morbidity and mortality. Finally, only those covariates that had significant relationships with 30-day mortality were entered into the regression model. A two-sided P value of 0.05 was used.

### Results

Table 1 describes the distribution of the 30-day mortality rate and patient and physician characteristics across physician pulmonary embolism case volume groups. Patients treated by low case volume physicians had significantly higher mortality rates than those treated by medium case volume (19.0% vs. 13.3%, P < 0.001) or high volume physicians (19.0% vs. 8.4%, P < 0.001). However, no significant relationship was observed between 30-day mortality and hospital caseload volume (P = 0.697). Of the total of 2761 first-time pulmonary embolism hospitalizations during the 3-year study period, 1271 (46.0%) were male, and 1263 (45.7%) were <65 years old. The mean age of the patients was 62.5 years, while that of the attending physicians was 42.5 years. Global chi-squared tests show that patients treated by physicians with high caseload volumes were more likely to be older (P < 0.001) and female

		Hospital pulmonary embolism volume			
Variable	All	Low (<42)	Medium (42–110)	High (≥111)	P value
No. (%) patients	2761	919 (33.3)	948 (34.3)	894 (32.4)	
30-day mortality rate (%)	14.1	14.7	14.1	13.3	0.697
Patient characteristics					
Gender, $n$ (%)					0.342
Male	1271 (46.0)	441 (48.0)	519 (54.8)	493 (55.2)	
Female	1490 (54.0)	478 (52.0)	429 (45.2)	401 (44.8)	
Age (years), $n$ (%)					0.237
< 65	1263 (45.7)	431 (46.9)	423 (44.6)	409 (45.7)	
65–74	665 (24.1)	199 (21.7)	249 (26.3)	217 (24.3)	
>74	833 (30.2)	289 (31.5)	276 (29.1)	268 (30.0)	
		Physician pulmonary embolism case volume			
		Low (<3)	Medium (3–6)	High (≥6)	
30-day mortality rate (%)	14.1	19.0	13.3	8.4	< 0.001
Patient characteristics					
No. (%) patients	2761	1066 (38.6)	875 (31.7)	820 (29.7)	
Age, mean (SD), years	62.5 (18.2)	60.2 (19.3)	62.8 (18.3)	65.2 (16.2)	< 0.001
No. (%) female	54.0	50.3	56.0	56.6	0.008
Age (years), $n$ (%)					< 0.001
< 65	1263 (45.7)	539 (50.6)	388 (44.3)	336 (41.0)	
65–74	665 (24.1)	246 (23.1)	218 (24.9)	201 (24.5)	
>74	833 (30.2)	281 (26.4)	269 (30.7)	283 (34.5)	
Physician characteristics		. ,		. ,	
No. (%) physicians	1229	880 (71.6)	258 (21.0)	91 (7.4)	
Age, mean (SD), years	42.5 (7.1)	42.6 (7.5)	42.4 (6.2)	42.1 (6.0)	0.762
No. (%) female	5.8	7.1	2.7	2.2	0.010
Age (years), $n$ (%)					0.053
≤40	880 (71.6)	400 (45.5)	109 (42.3)	44 (48.4)	
41-50	258 (21.0)	345 (39.2)	124 (48.1)	34 (37.4)	
≥51	91 (7.4)	135 (15.3)	25 (9.7)	13 (14.3)	
No. (%) practise location		(		- 🔨 2	0.117
Urban	816 (66.4)	571 (64.9)	177 (68.6)	68 (74.7)	
Rural	413 (41.3)	309 (35.1)	81 (31.4)	23 (25.3)	

Table 1 30-day mortality rate and patient and physician characteristics across physician pulmonary embolism caseload volume groups (n = 2761)

(P = 0.008). No significant relationships were found in the physician distributions in terms of age and practise location across caseload volume groups.

Table 2 presents the distributions of 30-day mortality after treatment for pulmonary embolism according to patient gender, age and comorbidites. Global chi-squared analyses showed that there were significant differences in 30-day mortality associated with age (P < 0.001), congestive heart failure (P = 0.031), hypertension (P < 0.001), coagulopathy (P < 0.001), renal failure (P = 0.009), solid tumors without metastasis (P < 0.001) and metastatic cancer (P < 0.001).

Table 3 also presents the regression results of conditional logistic regression modeling, showing that the adjusted odds of 30-day mortality among patients of low case volume physicians were over twice the mortality odds among patients of high case volume physicians, (OR = 2.164, reciprocal of 0.462, P < 0.001), and odds ratios were 1.401 relative to patients of medium case volume physicians (P < 0.05). These results support an independent effect of physician's experience on mortality, regardless of the hospital in which they practise. In

addition, as expected, the odds of 30-day mortality increased with physicians' age and with patients' comorbidities.

### Discussion

We used a 3-year nationwide population-based data set to study caseload volumes and outcomes for PE treatment. We found a significant inverse relationship between physician caseload volume and 30-day mortality, after adjusting for characteristics of physicians, patients and hospitals and the clustering effect among physicians. Patients treated by low case volume physicians have twice the 30-day mortality odds of those treated by high case volume physicians. However, hospital PE caseload volume was not a significant predictor of 30-day mortality. That is consistent with the conclusions of some prior studies which reported that physician caseload volume is a more significant factor than hospital caseload volume in predicting patient outcomes for specific treatment procedures, and that hospital volume is only marginally or not at all related to outcomes [16–19].

**Table 2** Distributions of 30-day mortality after pulmonary embolism by patient characteristics and comorbidities (n = 12 369)

### Table 2 (Continued)

	30-day mortality		
Variable	Yes, <i>n</i> (row %)	No, <i>n</i> (row %)	P value
	. ,	. ,	
Overall Gender	308 (14.1)	2373 (85.9)	0.14
Male	192 (15.1)	1079 (84.9)	0.14
Female	192 (13.1)	1294 (86.8)	
Age (years)	· · · ·		< 0.00
< 65	143 (11.3)	1120 (88.7)	
65–74	107 (16.1)	558 (83.9)	
>74	138 (16.6)	695 (83.4)	
Cardiac arrhythmias	20 (14 0)	1(1(05.2))	0.755
Yes No	28 (14.8) 360 (14.0)	161 (85.2) 2212 (86.0)	
Congestive heart failure	500 (14.0)	2212 (80.0)	0.03
Yes	59 (17.9)	270 (82.1)	0.05
No	329 (13.5)	2103 (86.5)	
Valvular disease	· /		0.198
Yes	8 (9.3)	78 (90.7)	
No	380 (14.2)	2295 (85.8)	
Pulmonary circulation disorders	aa	101 (0 : -	0.549
Yes	33 (15.4)	181 (84.6)	
No Denia hana lana analana dia andana	355 (13.9)	2192 (86.1)	
Peripheral vascular disorders Yes	N/A	10 (100.0)	-
No	388 (14.1)	2363 (85.9)	
Hypertension	500 (14.1)	2303 (03.7)	< 0.001
Yes	54 (8.9)	550 (91.1)	0100
No	334 (15.5)	1823 (84.5)	
Paralysis			0.337
Yes	5 (20.8)	19 (79.2)	
No	383 (14.0)	2354 (86.0)	
Coagulopathy			< 0.00
Yes	13 (38.2)	21 (61.7)	
No Other neurological disorders	375 (13.8)	2352 (86.3)	0.27
Other neurological disorders Yes	11 (19.0)	47 (81.0)	0.277
No	377 (14.0)	2326 (86.0)	
Chronic pulmonary disease	577 (14.0)	2520 (00.0)	0.958
Yes	42 (14.0)	259 (86.0)	0.500
No	346 (14.1)	2114 (85.9)	
Diabetes, uncomplicated			0.129
Yes	52 (16.9)	256 (83.1)	
No	336 (13.7)	2117 (86.3)	
Diabetes, complicated			0.568
Yes	16 (16.0)	84 (84.0)	
No	372 (14.0)	2289 (86.0)	0.550
Hypothyroidism Yes	7 (11.5)	54 (88.5)	0.558
No	381 (14.1)	2319 (85.9)	
Renal failure	201 (17.1)	(00.0)	0.009
Yes	16 (25.4)	47 (74.6)	0.00
No	372 (13.8)	2326 (86.2)	
Liver disease	. ,	~ /	0.493
Yes	17 (16.4)	87 (83.6)	
No	371 (14.0)	2286 (86.0)	
Peptic ulcer disease excluding			0.117
Bleeding			
Yes	6 (7.9)	70 (92.1)	
No Solid tumor without matastasis	382 (14.2)	2303 (85.8)	< 0.001
Solid tumor without metastasis Yes	16 (27 7)	120 (72.2)	< 0.00
1 55	46 (27.7)	120 (72.3)	

	30-day mortality		
Variable	Yes, <i>n</i> (row %)	No, <i>n</i> (row %)	P value
No	342 (13.2)	2253 (86.8)	
Rheumatoid arthritis			0.73
Yes	8 (15.7)	43 (84.3)	
No	380 (14.0)	2330 (86.0)	
Fluid and electrolyte disorders			0.060
Yes	16 (21.3)	59 (78.7)	
No	372 (13.9)	2314 (86.2)	
Deficiency anemias		× /	0.26
Yes	9 (10.0)	81 (90.0)	
No	379 (14.2)	2292 (85.8)	
Alcohol abuse	1 (20.0)	4 (80.0)	0.702
Yes	- ()	(0010)	
No	387 (14.0)	2369 (86.0)	
Psychoses	207 (1110)	2009 (0010)	0.65
Yes	3 (11.1)	24 (88.9)	0.05
No	385 (14.1)	2349 (85.9)	
Depression	565 (14.1)	2545 (05.5)	0.274
Yes	3 (25.0)	9 (75.0)	0.27
No	385 (14.0)	2364 (86.0)	
AIDS	385 (14.0)	2304 (80.0)	
Yes	N/A	N/A	_
No	/	/	
	388 (14.1)	2373 (85.9)	0.20
Lymphoma Yes	2 (28 ()	5 (71 4)	0.26
	2(28.6)	5 (71.4)	
No	386 (14.0)	2368 (86.0)	< 0.00
Metastatic cancer	<b>22</b> (21 0)	10 ((0.0)	< 0.00
Yes	23 (31.9)	49 (68.0)	
No	385 (13.6)	2324 (86.4)	
Obesity			-
Yes	N.A	N/A	
No	388 (14.1)	2373 (85.9)	
Weight loss			0.799
Yes	1 (11.1)	8 (88.9)	
No	387 (14.1)	2365 (85.9)	
Drug abuse			0.25
Yes	4 (8.3)	44 (91.7)	
No	384 (14.2)	2329 (85.8)	
Blood loss anemia	N/A	N/A	—
Yes No	388 (14.1)	2373 (85.9)	

Our finding does not concur with the conclusions drawn in an earlier study by Aujesky *et al.* [7]. Using discharge records from Pennsylvania hospitals between 2000 and 2002, they found that the odds of 30-day mortality were marginally but significantly lower at the hospitals with higher caseload volumes than at the very low caseload volume hospitals (P = 0.049), although they did not observe any significant difference in the unadjusted odds of 30-day mortality by hospital volume (P = 0.100).

One likely reason for the departure of our findings on PE treatment outcomes is the potential confounding effect of the health insurance system in the US, which has a tendency to limit a patient's choice to certain providers, using a gatekeeper or referral system. Primary care physicians acting as gatekeepers are likely to send patients to hospitals with established

Caseload volume a	nd mortality for	<sup>,</sup> pulmonary e	embolism treatment	1711
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Table 3 Crude and adjusted odds ratios for 30-day	mortalities,	by
physician pulmonary embolism case volume		

Variables Physician pulmonary of	95% CI	95% CI
Physician pulmonary e		-
	embolism case volume	
< 3	1.000	1.000
3–6	0.714* (0.545-0.935)	0.737* (0.557-0.974)
≥7	0.462*** (0.332-0.642)	0.508*** (0.362-0.714)
Physician characteristi	cs	
Physician age (years	5)	
< 41	0.945 (0.733-1.218)	0.986 (0.761-1.277)
41-50	1.000	1.000
> 50	0.751 (0.508-1.109)	0.827 (0.554-1.235)
Physician gender		
Male	1.061 (0.601-1.872)	1.176 (0.648-2.134)
Female	1.000	1.000
Practise location		
Urban	0.962 (0.744-1.243)	0.991 (0.760-1.292)
Rural	1.000	1.000
Patient characteristics		
Patient age (years)		
< 65	1.000	1.000
65–74	1.479** (1.127-1.941)	1.672*** (1.260-2.220
> 74	1.521** (1.146-2.019)	1.709*** (1.274-2.292
Congestive	1.390 (0.998-1.936)	1.404 (0.998-1.976)
heart failure		
Hypertension	0.519***(0.376-0.716)	0.518*** (0.371-0.723
Coagulopathy	3.819***(1.836-7.943)	4.401*** (2.051-9.444
Renal failure	2.109**(1.139-3.902)	2.075* (1.094-3.936)
Solid tumor	2.332***(1.590-3.420)	1.706* (1.096-2.655)
without		
metastasis		
Metastatic cancer	3.026***(1.773-5.164)	2.132* (1.159-3.920)
Hospital characteristic	S	
Hospital level		
Medical center	1102 (0.878-1.382)	1.073 (0.848-1.359)
Regional hospital	1.000	1.000
District hospital	1.410 (0.942-2.109)	1.203 (0.791–1.829)

\*P < 0.05; \*\*P < 0.01; \*\*\*P < 0.001.

reputations for better outcomes. Furthermore, the HMO system in the US not only limits patients' choice of doctors, but also doctors' choice of hospitals, further confounding volume-outcome results. Aujesky *et al.*'s study focused only on hospital caseload volume, rather than physician caseload volume [7], a study design that does not reflect the fact that patient outcomes for PE have been optimized through the development of physician-level strategies.

Conditional logistic regression analysis was used to eliminate potential confounding by unmeasured hospital variables. It essentially evaluates the volume-outcome association among physicians within each hospital and then averages these effects across hospitals. Therefore, our finding suggests that there is truly an independent effect of physicians' experience on outcomes, regardless of the hospital in which they practise. Even within the same department, physicians may be employing a variety of skills or procedures to treat PE, which could well lead to very different patient outcomes by physician.

The underlying mechanisms contributing to the volumeoutcome association in PE treatment found in our study remain unclear. Prior literature proposes the hypotheses of

'practise makes perfect' and 'self-referral' to explain such results [20]. According to the 'practise makes perfect' hypothesis, a larger volume of PE patients allows physicians to develop better skills for managing PE. These skills could include earlier diagnosis through CT pulmonary angiography (CTPA), earlier treatment (such as early anticoagulation therapy or rapid fibrinolytic therapy for patients with massive or unstable PE), closer monitoring of hemodynamic stability to prevent PE recurrence, better patient education, and so on. Besides, high caseload volume physicians may benefit from greater resources in their practise, such as housestaff, nurses and pharmacists working with them to deliver expertise. Although we believe that 'practise makes perfect' plays an important role in our results, we are unable to demonstrate through our crosssectional study whether the volume-outcome relationship can be fully explained by this hypothesis.

The second hypothesis, 'selective-referral', suggests that patients are inclined to selectively gravitate towards physicians with superior outcomes; thus these physicians develop a high volume of PE patients. However, PE occurs very suddenly and demands immediate medical attention. Although patient selfreferral implies that those with less severe disease can plausibly 'shop for' a physician with a 'good reputation' regardless of distance, or can opt for a geographically distant hospital with a 'good reputation', in reality, patients facing acute life-threatening situations generally settle for the nearest hospital. Therefore, selective referral may not be one of the major factors explaining the inverse relationship between patient outcomes and physician PE caseload volumes observed in Taiwan.

This study has a couple of caveats. First of all, some information, such as the interval between onset and diagnosis, Wells scores, blood pressure, pulse and respiratory rates, is not available through the NHIRD. Secondly, although this study has adjusted for patient comorbidities using the Elixhauser Comorbidity Index, the administrative data base we used is extremely limited in its ability to account for differences in PE severity among patients. Further studies using more sophisticated risk-adjusted methods (such as the severity classifications proposed by Aujesky *et al.* [21]) will be necessary to adequately confirm physician volume-outcome results found by this study.

Despite these limitations, we found that after adjusting for patient, physician and hospital characteristics and for clustering effects among physicians, an inverse PE volume-outcome relationship does exist for physicians in Taiwan, but not for hospitals. There are several policy implications. First, we ought to regionalize care so more patients are cared for by the most experienced practitioners, anywhere they may be. Second, payers should consider reimbursing for consultations that include a second experienced attending physician, until each physician completes a threshold critical number of PE cases. Third, future research should be initiated to identify differences in clinical approaches and techniques that distinguish high case volume physicians with superior outcomes from low case volume physicians with inferior outcomes; the results of such studies could help the latter improve the quality of their PE patient care.

### **Disclosure of Conflict of Interests**

The authors state that they have no conflict of interest.

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