

The Association between Cataract Surgery and Cognitive Dysfunction in Cataract Patients - A Population-Based Follow-Up in Taiwan

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Abstract

Purpose: Senile cataract and cognitive dysfunction (senile dementia, cognitive impairment and Alzheimer disease) are commonly coexisting. Some ophthalmologists believe that cataract surgery not only can restore health to a patient's eyes, but also can improve the degenerative state of mind. Since their relationship is not fully understood, this study aims to prospectively investigate the association between cataract surgery and long-term incidence of cognitive dysfunction among older people with senile cataract.

Methods: Retrospective Cohort study design. We used the National Health Insurance Research Database of Taiwan to investigate the relationship between cataract surgery and incidence of cognitive dysfunction in patients with cataract between January 2000 and December 2010. A total of 31401 cataract patients were included. There were 11385 patients receiving cataract surgery and 20016 age-, sex-, and cataract-diagnosis-timing matched controls who were linked to the claim data to identify the first occurrence of a primary or secondary diagnosis of cognitive dysfunction (including cognitive impairment, senile dementia, and Alzheimer disease). We used the

Kaplan-Meier method to estimate the survival curves of cognitive dysfunction. Cox proportional hazards regression modeling was used to determine the association between patients with cataract who received surgery or not and risk for cognitive dysfunction, adjusting for comorbidities, such as hypertension, diabetic mellitus, hyperlipidemia, dysrhythmia, and coronary artery disease.

Results : Over nearly 10 years of follow-up, a total of 505 cataract subjects who received surgery developed cognitive dysfunction, representing a cumulative incidence rate of 4.44% (n = 727; 3.63% in controls). Cataract patients who received surgery had a similar risk of cognitive dysfunction as patients who did not receive surgery (Hazard Ratio (HR): 1.08, 95% Confidence Interval (CI) 0.97-1.21). In the age-stratified analysis, cataract surgery was associated with increased risk of cognitive dysfunction among patients between 65-74 years of age (HR 1.20, 95% CI 1.04-1.39), and the association was null in patients over 75 years (75-84 years, HR 0.948, 95% CI 0.78-1.16; ≥ 85 years, HR 0.727, CI 0.41-1.28).

Conclusion: Overall, cataract patients (>65 years) undergoing surgery had a similar risk of cognitive

dysfunction (HR 1.08, 95% Confidence Interval (CI) 0.97-1.21). Among cataract patients who were 65-74 years of age, the long-term risk for cognitive dysfunction seemed to increase in those who received cataract surgery compared to those who did not. The risk of surgery was not observed in the older age groups (≥ 75 years old). The surgery for cataract led to better vision for patients, but there was no conclusive evidence for improvement in cognitive function. On the other hand, "the necessity for cataract surgery" could be an indicator for aging change, especially for "younger" aged people (< 75 years). The ophthalmologists should be aware of the patients with the potential need for cataract surgery, which could be a sign of cognitive dysfunction. The multiple lines of communication between ophthalmology, neurology and geriatrics are very important.

Keywords: Cataract; Dementia; Cognitive Dysfunction; Alzheimer Disease; Phacoemulsification; Lens Extraction

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Introduction

Cataract is the opacity of the lens in the eye which leads to a decrease in vision. The number of people with blindness due to age-related cataracts is about 20 million and is responsible for 51% of world blindness [1]. In the United States, age-related lens changes have been reported in 42% of the people between the ages of 52 and 64, [2] 60% between the ages 65 and 74, [3] and 91% above the age of 75 [2]. Direct medical costs for cataract treatment have been estimated at \$6.8 billion annually [4]. Another age-related condition is the neurodegenerative disease dementia, which is characterized by slowly progressive memory loss, cognitive impairment, and an impaired ability to manage personal daily activities [5]. The number of cases of dementia worldwide in 2010 was estimated at 35.6 million [6].

Rates increase significantly with age, with dementia affecting 5% of the population older than 65 and 15–30% of those older than 85 [7]. Both cataract and dementia have caused a considerable public health burden worldwide.

The etiology of cognitive dysfunction (including senile dementia, cognitive impairment, and Alzheimer disease) are multi-factorial and the majority of cases are caused by vascular or senile factors [8]. However, cataract-related visual impairment may be the one of the causes for cognitive decline. There are numerous studies examining the hypothesis that cataract surgery can reduce cognitive decline, though with varying results [9-15].

The true correlation between cataract and cognitive dysfunction in a clinical setting remains unclear. Research shows that there are possible ways to decrease the risk of acquiring dementia, including lifestyle changes and medications [16]. Physical and mental activity may also help to prevent cognitive decline by building up additional connections between neurons which are more resistant to the deterioration seen in dementia [16]. Therefore, most ophthalmologists believe the cataract surgery will not only restore health to a patient's eyes, but also result in better vision, improvement in the quality of life, enhanced awareness of the environment, and perhaps improvement to the confused, degenerative state of mind [17]. The aim of this study was thus to investigate the risk of cognitive dysfunction over a 10-year period in elderly (> 65 years) people with newly diagnosed cataract. The two main questions guiding this study were the following:

- i. Could the cataract surgery decrease the risk of cognitive dysfunction?
- ii. Could the cataract surgery decrease the risk of cognitive dysfunction under the different age and sex stratification?

Methods

Database

Since 1995, the National Health insurance program has provided health care to all Taiwanese residents. This program

had increased its coverage rate from an initial 96.16% to over 99% in 2010. Around 97% of hospitals and 90% of clinics have contracts with the Bureau of National Health Insurance (NHI). (http://www.nhi.gov.tw/webdata/webdata.aspx?menu=17&menu_id=659&webdata_id=2891&WD_ID=897)

The Bureau of NHI provided the administrative data contained in the National Health Insurance Research Database (NHIRD) for research purposes. For individual privacy, the Bureau of NHI made a secret code for each subject and constructed this database, further scrambling this information for the researchers.

Our database included the registration and medical claims of the 1,000,000 randomly sampled individuals from 1997 till 2010. Due to the encrypted information, any individual information could not be obtained linked to identity number. As the National Health Research Institute (NHRI) had addressed the confidentiality assurance issue, a full review of this study was waived by the Institution Review Board of Cheng-Hsin General Hospital.

Study Population

We conducted a cohort study from the Longitudinal Health Insurance Database 2005 (LHID2005). Inclusion criteria were that the subjects were older than 65 years of age with newly diagnosed cataract from 1997 till 2010. And the exclusion criteria were any past history of dementia, head injury, stroke, depression, mood disorder, eye disease or other catastrophic illness which may interfere with our final results

Our study cohort was based on the following inclusion criteria: patients aged over 65 years who visited outpatient clinics from January 1997 with at least 2 consensual diagnoses of cataract according to the International Classification of Diseases (9th Revision, Clinical Modification [ICD-9-CM], code 366.10-366.19) (n=83934). A flow chart of the selection process is illustrated in Figure 1.

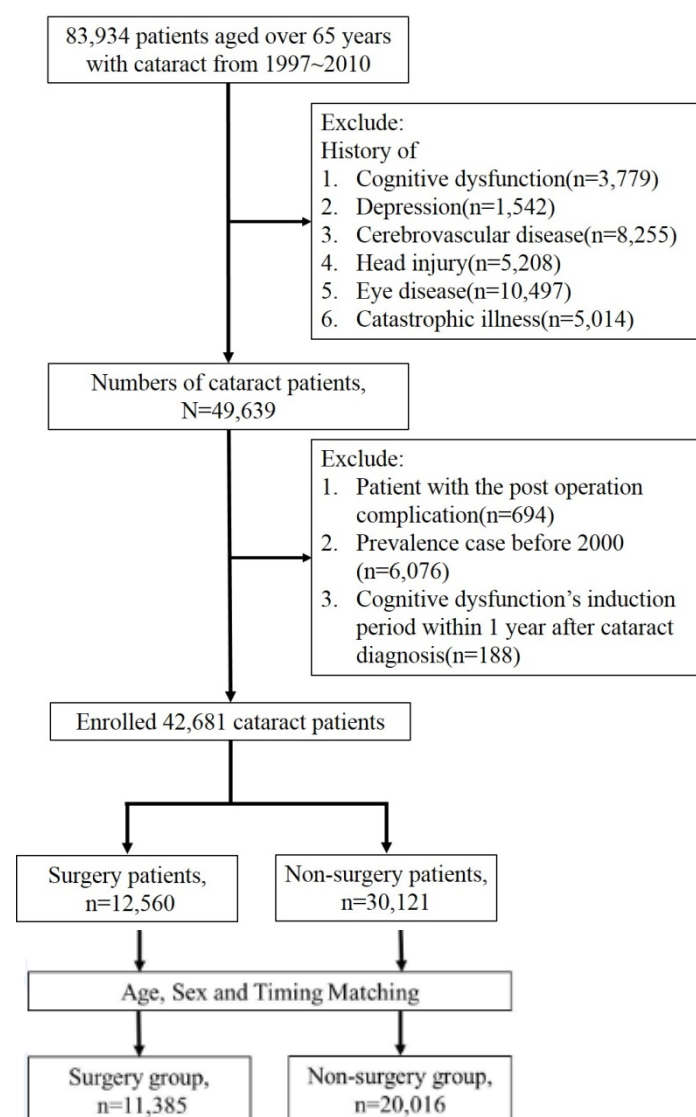


Figure 1: Flowchart of recruitment of subjects from the National Health Insurance Research Database (NHIRD) from 1997 to 2010.

We excluded patients with a previous history of dementia (ICD-9-CM code 290; n=3703), stroke (ICD-9-CM code 433-434; n=7188), intracranial hemorrhage (ICD-9-CM code 430-432; n=1067), cognitive impairment (ICD-9-CM code 331.83; n=0), Alzheimer disease (ICD-9-CM code 331.0; n=76), major depression (ICD-9-CM code 296.2x, 296.3x; n=1542), head injury (ICD-9-CM code 959.01, 850.0-854.1; n=5208), eye disease (e.g., glaucoma ICD-9-CM code 365.xx, n=6114, proliferative diabetic retinopathy ICD-9-CM code 362.02 ,

379.23, 362.53; n=1394; age-related macular degeneration ICD-9-CM code 362.50-362.52; n=2032; optic neuropathy ICD-9-CM code 377; n=557, ocular trauma ICD-9-CM code 870, 871; n=400), or other catastrophic illness (ICD code 140.0-199.1, n=5014)--such as cancer (any type)--that was diagnosed before the first diagnosis of cataract. The present study cohort consisted of 49636 patients with diagnosis of cataract. Our compared cohort was selected from the selected patients who also had outpatient visits and received cataract operation during the same period. From the remaining eligible subjects, we randomly selected an age- sex- and cataract-diagnosed-timing-matched control cohort that was diagnosed as cataract within two months compared to the study group who was also diagnosed as victim of cataract.

In case of poor visual acuity after cataract surgery, we excluded the patients with post operation complications (n=694) such as endophthalmitis (ICD-9-CM code 360.0/360.00-04/360.11-19), retinal detachment (ICD-9-CM code 361.0/361.3/361.8/361.9), cystoid macular edema (ICD-9-CM code 362.53), and lens subluxation (ICD-9-CM code 379.32). To recruit only subjects with newly diagnosed cataracts, we excluded patients who were previously diagnosed with cataract or who had ever received topical anti-cataract drug between January 1997 and December 1999 (n=6076). To control the bias about the induction time for the development of cognitive dysfunction, we excluded the cognitive dysfunction diagnosed within 1 year after the diagnosis of cataract (n=188). In the end, our study included a total of 42681 subjects with newly diagnosed cataract (12560 newly diagnosed cataract patients receiving cataract surgery and 30121 newly diagnosed cataract non-surgery patients).

Ascertainment of cognitive dysfunction

We defined cognitive dysfunction as any of dementia, cognitive impairment, or Alzheimer disease; we individually tracked all subjects in both the study and control cohorts from the date of cataract surgery for 10 years. We retrieved records for subjects who were diagnosed for the first time with dementia (ICD-9 CM code 290) or Alzheimer disease (ICD-9 CM code

331.0) or cognitive impairment (ICD-9 CM code 331.83) after confirming the diagnosis through the presence of 2 consensual diagnoses and with an induction period of at least one year. To increase confidence of the final diagnosis, we combined Alzheimer disease or senile dementia with the drug using including donepezil, rivastigmine, galantamine, memantine). The incidence of cognitive dysfunction was recorded after the index visit of each individual.

Information on other covariates

We also identified the baseline comorbidities of all subjects, including hypertension (ICD-9-CM code 401), diabetes mellitus (ICD-9-CM code 250.xx), hyperlipidemia (ICD-9-CM code 272.xx), dysrhythmia (ICD-9-CM code 427.xx, 785.1, 785.1) and coronary artery disease (ICD-9-CM code 410.xx-414.xx) from data gathered from ambulatory care claims with at least 2 consensual diagnoses between 2000 and 2010. We tracked these particular comorbidities because they might have been involved in the development of cognitive dysfunction.

Statistical analysis

All statistical analyses and survival curve plots were performed using SAS version 9.2. Pearson chi-square tests were performed to examine the differences in categorical data, including the state of comorbidities between study and age- and sex-matched groups. We conducted a survival analysis (time to cognitive dysfunction) using the Kaplan-Meier method and the log-rank test to compare the survival distributions between the two groups. The Cox proportional hazards regression model was used to analyze the Hazard Ratio (HR) for incidence of cognitive dysfunction among patients with newly diagnosed cataract who received cataract surgery or not, adjusting for potential confounders. To evaluate the Hazard Ratio (HR) for cataract patients with and without treatment for cataract under the age stratification and sex subgroup analysis in the development of cognitive dysfunction, multivariate hazards regression was used. A p value of <0.05 was considered statistically significant.

Results

The mean age of the final cataract cohort and surgery cohort was 74.38±5.88 and 74.40±5.87 years, respectively. In the cataract surgery study cohort, subjects with onset ages of 65-74 years (younger-aged) accounted for 58.05% of the cohort, while subjects with onset ages of ≥75 years (older-aged) accounted for the remaining 41.95%. Men consisted of 45.0% of both study and control cohorts (Table 1). The presence of hypertension, diabetes, hyperlipidemia, dysrhythmia and coronary artery disease at baseline was significantly higher in patients with cataract surgery than in the control group (Table 1). After a median follow up of 4.65±3.1 years, there were 1232

cases of cognitive dysfunction (36 cases of dementia combined with drugs, 104 cases of Alzheimer disease, and 1128 cases of dementia). The 10-year cumulative incidence rates of cognitive dysfunction were 505 (4.44%) (Including 469 cases (4.12%) of dementia, 36 cases (0.32%) of Alzheimer disease, and 16 cases (0.14%) of dementia with drugs) in the surgery group and 727 (3.63%) (Including 659 cases (3.29%) of dementia, 68 cases (0.34%) of Alzheimer disease, 20 cases (0.10%) of dementia with drugs) in the non-surgery group. The mean incident time of developing cognitive dysfunction in the surgery group was 4.05±2.53 years (3.42±2.31 years in the non-surgery group).

Table 1: Comparisons of characteristics among the sampled cataract patients

	Surgery		
	With	Without	
Variables	N=11385	N=20016	p-value
Age (years)*	74.40±5.87	74.38±5.88	0.7154
Male	5127 (45.03%)	9003 (44.98%)	0.9264
Follow up times (years)	4.64±3.08	4.66±3.09	0.6500
History of			
Hypertension	6916 (60.75%)	9988 (49.90%)	<0.0001
Diabetes mellitus	3819 (33.54%)	6046 (30.21%)	<0.0001
Hyperlipidemia	4613 (40.52%)	7655 (38.24%)	<0.0001
Coronary artery disease	2357 (20.70%)	3409 (17.03%)	<0.0001
Dysrhythmia	2982 (26.19%)	4717 (23.57%)	<0.0001
Time to Cognitive dysfunction (years)	4.05±2.53	3.42±2.31	<0.0001
Cognitive dysfunction	505 (4.44%)	727 (3.63%)	0.0004
Dementia	469 (4.12%)	659 (3.29%)	0.0003
Alzheimer	36 (0.32%)	68 (0.34%)	0.7273
Dementia combine drug	16 (0.14%)	20 (0.10%)	0.3066
*Mean ± SD			

The Kaplan-Meier survival curves shown in Figure 2 demonstrate significantly lower dementia-free survival rates in

the cataract-surgery cohort as compared with the non-surgery subjects (log-rank test p<0.001).

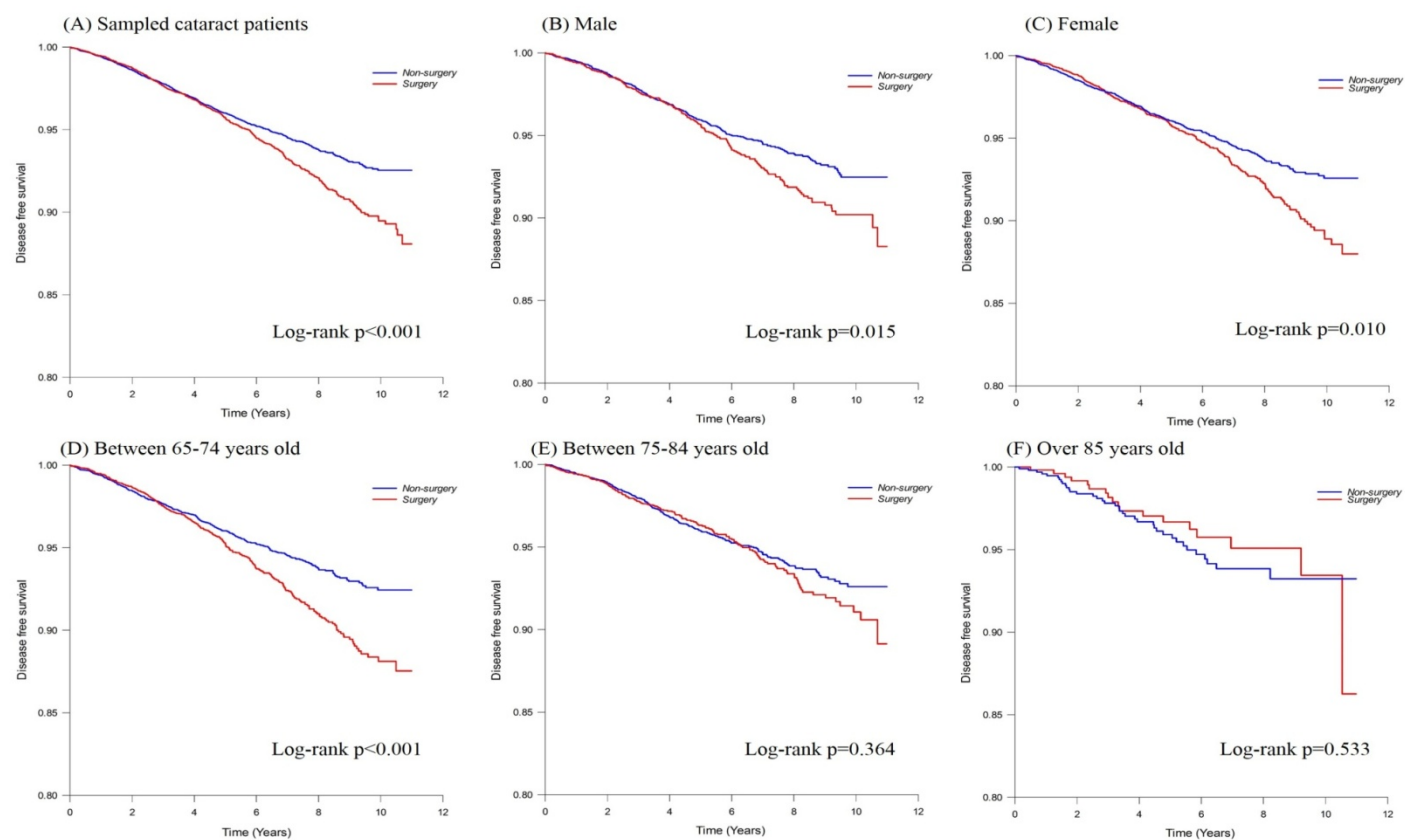


Figure 2: Kaplan-Meier estimates of the probability of cognitive dysfunction free survival in cataract patients.

The crude and adjusted HRs for cognitive dysfunction is presented in Table 2. In the univariable analysis, receiving cataract surgery was associated with increased risk of cognitive dysfunction (HR 1.23, 95% CI: 1.10-1.37). After adjusting for the potential confounders, receiving surgery was not associated with risk for further development of cognitive dysfunction (HR 1.08, 95% CI: 0.97-1.21). Other significant risk factors for cognitive dysfunction were hypertension (HR 2.06, 95% CI

1.81-2.34), diabetic mellitus (HR 1.20, 95% CI 1.05-1.36), coronary artery disease (HR 1.33, 95% CI 1.16-1.54), and dysrhythmias (HR 1.39, 95% CI 1.22-1.58). Newly diagnosed cataract patients who had comorbidities of hypertension, diabetic mellitus, coronary artery disease, and dysrhythmia were found to have a substantially increased risk of developing cognitive dysfunction. There was no sex difference between these two groups (male HR 1.02, 95% CI 0.91-1.14).

Table 2: Univariate and multivariate hazard ratios for cognitive dysfunction among the sampled cataract patients

			Crude HR ^a		Model ^b	
Variables	Case numbers	person-years	HR	95%CI	HR	95%CI
Surgery						
No	727	93221	1.000		1.000	
Yes	505	52836	1.226	1.10-1.37	1.083	0.97-1.21
Age (years)			0.986	0.98-0.99	0.986	0.98-0.99
Gender						

Female	683	80843	1.000		1.000	
Male	549	65213	0.998	0.89-1.12	1.021	0.91-1.14
History of Hypertension						
Without	400	77005	1.000		1.000	
With	832	69052	2.403	2.13-2.71	2.058	1.81-2.34
Diabetes mellitus						
Without	809	107777	1.000		1.000	
With	423	38279	1.518	1.35-1.71	1.195	1.05-1.36
Hyperlipidemia						
Without	773	100523	1.000		1.000	
With	459	45534	1.368	1.22-1.54	0.952	0.84-1.08
Coronary artery disease						
Without	945	125197	1.000		1.000	
With	287	20860	1.895	1.66-2.16	1.334	1.16-1.54
Dysrhythmia						
Without	869	117808	1.000		1.000	
With	363	28248	1.812	1.60-2.05	1.385	1.22-1.58

- Hazard ratio was calculated by using stratified Cox proportional regression
- Adjusted for age, gender and comorbidity

Table 3 provides the crude and adjusted HR for risk of cognitive dysfunction among subjects newly diagnosed as cataract who received surgery in different subgroups according to age (65-74, or 75-84, or ≥ 85 years) and sex stratification. After adjusting for potential confounders, the data in the table clearly demonstrate that the risk of developing cognitive dysfunction as associated with newly diagnosed cataract receiving surgery was higher for patients with an onset from a younger age (65-74 years; HR=1.2, 95% CI, 1.04-1.39). Otherwise, there was no significant effect modification between sex (male, HR 1.10, 95% CI 0.93-1.31; female, HR 1.066, 95% CI, 0.91-1.24; P=0.79) and elderly age (75-84 years, HR 0.95, 95% CI 0.78-1.16; ≥ 85 years, HR 0.727, 95% CI 0.41-1.28; P<0.0001). From using age stratification, it was found that cataract surgery becomes a protective factor in elderly (>75 years). But the result is non-significant due to the confidence interval including one.

Table 3: Univariate and multivariate hazard ratios for cognitive dysfunction stratified by gender and age

	Crude ^a		Model 2 ^b	
Variables	HR	95%CI	HR	95%CI
Male				
Surgery	1.233	1.04-1.46	1.102	0.93-1.31
Female				
Surgery	1.220	1.05-1.42	1.066	0.91-1.24
p value		0.93		0.79
Between 65-74 years old				
Surgery	1.342	1.16-1.55	1.198	1.04-1.39
Between 75-84 years old				
Surgery	1.095	0.90-1.33	0.948	0.78-1.16
Over 85 years old				
Surgery	0.837	0.48-1.47	0.727	0.41-1.28
p value		0.033		<0.0001

- a. Hazard ratio was calculated by using Cox proportional regression
- b. Adjusted for age and gender and comorbidity

N_{Male}: 14130, N_{Female}: 17271, N_{Between 65-74 years old}: 18229, N_{Between 75-84 years old}: 11551, N_{Over 85 years old}: 1621

Discussion

Our study revealed an increased risk of cognitive dysfunction among 65-74 year-old patients with newly diagnosed cataract receiving surgery over a 10-year period. However, the association was not found in the older group (>75 years). Furthermore, we also found that overall cataract patients (>65 years) undergoing surgery had a similar risk of cognitive dysfunction (HR 1.08, 95% Confidence Interval (CI) 0.97-1.21). Cataract surgery seems to delay the incident time of cognitive dysfunction. The mean incident time of cognitive dysfunction in the surgery group was 4.05 ± 2.53 years (non-surgery group: 3.42 ± 2.31 years). In line with previous studies, the patients in this study with hypertension, diabetic mellitus, coronary artery disease and dysrhythmia were at a higher risk of developing cognitive dysfunction.

Most studies have not reached a consensus about whether cataract surgery can improve cognitive function or reduce the risk of cognitive dysfunction. Since we hypothesize that cognitive decline and visual impairment may be related, amelioration of visual decline, such as by cataract surgery, may thus improve cognitive function and visual disturbance [16]. However, the results of the cross-sectional epidemiological survey from Grodstein and colleagues [11] show no beneficial effect of cataract surgery on cognitive impairment or dementia. In addition, Anstey and associates [9] demonstrated the non-difference in cognitive performance for either the surgery or control group in a randomized control study. Those are large sample design and well-organized studies. Other reports supporting the hypothesis that cataract surgery may improve cognitive function are small in patient number or suffer from improper study design or are uninterpretable in terms of learning effect [10, 12, 13, 15]. In our study, the overall adjusted HR was 1.083 (95% CI: 0.97-1.21) for people older

than 65 years receiving cataract surgery. This means that cataract surgery has no effect on preventing cognitive dysfunction. When taking into account age stratification, the slight effect of cataract surgery was only evident for the 65-74 year-old age group, with no clinically significant effect for the >75 year-old groups. Our results are therefore compatible with previous large-scale studies [9, 11]. Although the visual function, mood, and visual quality of life may be improved after cataract surgery, at present, there is no definite evidence indicating that cataract surgery can improve cognitive functioning.

There could be many factors affecting the results of our study. Nevertheless, no previous study has shown the effect of cataract surgery on the development of cognitive dysfunction. It is reasonable to explain the association of cataract surgery and cognitive dysfunction in so far as for patients with cataract they need to receive surgery, these may be indicators of aging and cognitive dysfunction. The results of earlier epidemiological studies suggested that senile cataract is a marker of generalized tissue aging and even higher mortality rate, especially in younger patients [9, 18-26]. Shinohara and colleagues reported that many cataractogenic stressors induce Endoplasmic Reticulum (ER) stress, which in turn induces the Unfolded Protein Response (UPR) [27-29]. The UPR up-regulates caspases and produces Reactive Oxygen Species (ROS) in Lens Epithelial Cells (LECs). These caspases and the ROS can lead to serious cellular damage, cell death, cataract, and Alzheimer disease [27-30]. Since Alzheimer disease, Parkinson disease and cataract are all caused by protein aggregations, e.g., mutant presenilin and β -amyloid protein are found in the lens and brain, both cataract and senile dementia/Alzheimer disease are the result of aging change. Only curing the cataract to improve visual quality seems not to prevent further progression of cognitive dysfunction under this theory.

Cognitive dysfunction and cataract are degenerative diseases sharing common risk factors, including age, smoking, lower socioeconomic class, diabetes, and obesity. The necessity for cataract surgery was an indicator for degenerative change. If

the severity of cataract is enough to receive surgery, then it is likely that the other organs could also be at a level of “active” degenerating status. In addition, incident cataract was found to be a “window” for increasing risk of cognitive dysfunction [27, 29]. If these older people need cataract surgery, we should be alerted to the other organs of the patients and refer them to other subspecialists for health examination, such as to a neurologist for a Mini-Mental State Examination (MMSE) survey or brain imaging study to prevent or delay cognitive dysfunction.

The strength of the present study is derived largely from the study design: the 10-year follow-up, the large number of study subjects, the presence of age- and sex-matched controls, the wide range of population age, as well as the ability to recruit newly diagnosed cataract-patients in the study cohort and the ability to identify multiple endpoints in the full follow-up period. Such a study design enabled the investigation of the risk of dementia in patients who received surgery or not and from the initial diagnosis of cataract. It also allowed a comparison of the risk differences under age-stratification. Given that inclusion of these subjects in the control or study group might interfere with the accuracy of a follow-up study, we excluded all cataract or dementia-related diseases diagnosed during the baseline period (1997-2000) and during the follow-up period (2001-2010) to ensure that the diagnosis of dementia or cognitive dysfunction was impressed with a minimum of a one year induction period after diagnosis of cataract. Moreover, using the nationwide administrative database in Taiwan, which allows researchers to trace almost all outpatient visits, allowed us to track up to 94.7% of subjects for 10 years. By the end of the 10th year, less than 5.5% of subjects were lost or unavailable for follow-up data collection.

However, several limitations of our study need to be addressed. First, the diagnosis of cataract, dementia, cognitive impairment, Alzheimer disease and other comorbid medical conditions relying on diagnostic criteria according to the ICD-9-CM definitions may have affected study accuracy. To address this concern, for the cataract cohort, we only recruited subjects who had at least 2 visits where a consensual diagnostic code for

cataract was used. These criteria would minimize the probability of recruiting non-cataract patients. Similarly, subjects were considered to have dementia only when they had at least 2 outpatient visits where the clinical diagnosis of dementia was documented in the medical records. Therefore, the possibility of misclassification was minimized in the study. In addition, we combined drug code with diagnosis of dementia as a validation study to increase the accuracy of the diagnosis from the ICD-code.

Second, the data contain no information about the severity of cataract, family history, genetic data (for example apolipoprotein E ϵ 4 allele), visual acuity, physical activity status, cigarette smoking, alcohol consumption, body mass index, MMSE score, and the level of education. Thus, the variables we used for adjustment might be limited.

Third, the heterogeneity between control and study group cannot be eliminated completely. We could only decrease the difference by using age- and sex-matching, and the regression analysis of the comorbidities related to dementia. Further external validity tests and studies of multiple centers with randomized control trials for subgroup analysis should be conducted and adjusted for invalid data in the NHIRD in future research.

The government in Taiwan is searching for a relatively simple and low cost initial set of indicators to determine if their aging population needs further medical assistance. These indicators can be measured using information that is readily available and adaptable to local hospitals, thereby providing a low-cost way for the doctors to begin to examine the specific medical needs of their aging populations. For this reason, we can disclose from our study findings the simple and low-cost indicator—severity of cataract (need to receive surgery).

In summary, our study provides information regarding the association between cognitive dysfunction and cataract which needs surgery to improve visual function. The patients receiving cataract surgery seem not to have a reduced risk of cognitive dysfunction in the >65 year old group, for whom cataract

surgery simply appears to be an indicator to imply the aging status. We should pay more attention to the patients at the age of 65-74 who need cataract surgery, for they may have a higher risk for cognitive dysfunction.

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