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**A Potential Concomitant Sellar Embryonic- Remnant associated Collision Tumor**

*Wang, et al*

Supplementary appendix-1

**Sections page**

**1) Systematic reviews and meta-analyses (PRISMA) guidelines and checklist 1**

**2) Inclusion criteria -** **The diagnosis standard of pituitary adenoma; Explanation of cases without modern imaging in the 1920s and 1970s Additional explanation 4**

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**Supplementary Table 1: Checklist of study selection in the study**

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| --- | --- | --- | --- |
| **Section/topic** | **#** | **Checklist item** | **Reported on page #** |
| **TITLE** | | |  |
| Title | 1 | Identify the report as a systematic review, meta-analysis, or both. | Page1 |
| **ABSTRACT** | | |  |
| Structured summary | 2 | Provide a structured summary including, as applicable: background; objectives; data sources; study eligibility criteria, participants, and interventions; study appraisal and synthesis methods; results; limitations; conclusions and implications of key findings; systematic review registration number. | Page2 |
| **INTRODUCTION** | | |  |
| Rationale | 3 | Describe the rationale for the review in the context of what is already known. | Page4 |
| Objectives | 4 | Provide an explicit statement of questions being addressed with reference to participants, interventions, comparisons, outcomes, and study design (PICOS). | Page6 |
| **METHODS** | | |  |
| Protocol and registration | 5 | Indicate if a review protocol exists, if and where it can be accessed (e.g., Web address), and, if available, provide registration information including registration number. | Page6 |
| Eligibility criteria | 6 | Specify study characteristics (e.g., PICOS, length of follow-up) and report characteristics (e.g., years considered, language, publication status) used as criteria for eligibility, giving rationale. | Page6 |
| Information sources | 7 | Describe all information sources (e.g., databases with dates of coverage, contact with study authors to identify additional studies) in the search and date last searched. | Page6 |
| Search | 8 | Present full electronic search strategy for at least one database, including any limits used, such that it could be repeated. | Page6 |
| Study selection | 9 | State the process for selecting studies (i.e., screening, eligibility, included in systematic review, and, if applicable, included in the meta-analysis). | Page7 |
| Data collection process | 10 | Describe method of data extraction from reports (e.g., piloted forms, independently, in duplicate) and any processes for obtaining and confirming data from investigators. | Page7 |
| Data items | 11 | List and define all variables for which data were sought (e.g., PICOS, funding sources) and any assumptions and simplifications made. | Page7 |
| Risk of bias in individual studies | 12 | Describe methods used for assessing risk of bias of individual studies (including specification of whether this was done at the study or outcome level), and how this information is to be used in any data synthesis. | Page8 |
| Summary measures | 13 | State the principal summary measures (e.g., risk ratio, difference in means). | Page8 |
| Synthesis of results | 14 | Describe the methods of handling data and combining results of studies, if done, including measures of consistency (e.g., I2) for each meta-analysis. | Page8 |

|  |  |  |  |
| --- | --- | --- | --- |
| **Section/topic** | **#** | **Checklist item** | **Reported on page #** |
| Risk of bias across studies | 15 | Specify any assessment of risk of bias that may affect the cumulative evidence (e.g., publication bias, selective reporting within studies). | Page8 |
| Additional analyses | 16 | Describe methods of additional analyses (e.g., sensitivity or subgroup analyses, meta-regression), if done, indicating which were pre-specified. | Page8 |
| **RESULTS** | | |  |
| Study selection | 17 | Give numbers of studies screened, assessed for eligibility, and included in the review, with reasons for exclusions at each stage, ideally with a flow diagram. | Page8 |
| Study characteristics | 18 | For each study, present characteristics for which data were extracted (e.g., study size, PICOS, follow-up period) and provide the citations. | Page9 |
| Risk of bias within studies | 19 | Present data on risk of bias of each study and, if available, any outcome level assessment (see item 12). | Page9 |
| Results of individual studies | 20 | For all outcomes considered (benefits or harms), present, for each study: (a) simple summary data for each intervention group (b) effect estimates and confidence intervals, ideally with a forest plot. | Page10 |
| Synthesis of results | 21 | Present results of each meta-analysis done, including confidence intervals and measures of consistency. | Page11 |
| Risk of bias across studies | 22 | Present results of any assessment of risk of bias across studies (see Item 15). | Page12 |
| Additional analysis | 23 | Give results of additional analyses, if done (e.g., sensitivity or subgroup analyses, meta-regression [see Item 16]). | Page12 |
| **DISCUSSION** | | |  |
| Summary of evidence | 24 | Summarize the main findings including the strength of evidence for each main outcome; consider their relevance to key groups (e.g., healthcare providers, users, and policy makers). | Page13 |
| Limitations | 25 | Discuss limitations at study and outcome level (e.g., risk of bias), and at review-level (e.g., incomplete retrieval of identified research, reporting bias). | Page16 |
| Conclusions | 26 | Provide a general interpretation of the results in the context of other evidence, and implications for future research. | Page17 |
| **FUNDING** | | |  |
| Funding | 27 | Describe sources of funding for the systematic review and other support (e.g., supply of data); role of funders for the systematic review. | Page18 |

**The diagnosis standard of pituitary adenoma**

The diagnosis criteria for pituitary adenoma is based on the classification of classification of tumors of the pituitary gland by WHO in 2017 (1).

**Inclusion criteria-- Explanation of cases without modern imaging in the 1920s and 1970s Additional explanation**

CT, MRI, DSA, CTA, and MRA were certainly not available until the early 70ies. Prior to the use neuroimaging, light microscope and clinical histopathologic examinations have been used for studies in clinical practice. Based to the histopathological performance, combined with the clinical endocrine characteristics and clinical manifestations, we meet the diagnosis criteria of 2017.

**Statistical methods**

We first used R programming language version 3.4 (statistic package studio) to implement Random forest algorithm and get the important Categorical variable score (We identified sixty-seven meta-analyses (30-94) 30-94. Test for association between unitary-factor and the clinical surgery was the outcome. A series of correlation analysis were preformed to observe the age, histopathology trend of the data and verify the presence of significant Risk of merge of two diseases, developmental relationship between age and RCC coexisting sellar lesion risk in the outcomes. The statistical heterogeneity between specific OR by the *I2*statistic test and Quantitative test, specificity yielded a *P* value.

Next, we performed several post-hoc sensitivity analysis. For comparisons between different age groups, we conducted multiple comparison into account by using Parirwise comparison as a post–hoc test. We illustrated differ age subgroup sensitivity and specificity through curves of the receiver operating characteristic (AUC). The Cox proportional hazard model was used for hazard ratios (HR) with 95% confidence intervals (CIs) were used to quantify the strength of the association between risk coexistent and age. That evaluated whether age was a significant direct effect, between variable, age and RCC coexisting sellar lesion in the present cases, risk of merger of two disease. Levene’s Test was used to test the homogeneity of variance prior to ANOVA analysis, and measured age and RCC coexistent sellar lesion inter-observer variability.

Finally, we conducted two multivariate logistic regression analysis to identify the predictors of two disease coexistent, various subtype pathological (HR and sensitivity, specificity) and the predictively accuracy. A Cox proportional-hazard model for multivariable analysis was applied for variables (subtype pituitary adenoma, other sellar region lesions) that proved to be significant in the univariate. Hazard ratios (HR) with 95% confidence interval (95% CI) were determined using univariate or multivariable analysis. We used the Cox hazard regression model to calculate the risk degree of pathology typing. Receiver operating characteristic (ROC) analysis was used to identify the most disease likely to merge (various types of PA, Hypo, aneurysm, Chor & CP, and other sellar lesion) by plotting evaluation by sellar region lesion coexistence RCC status, and predictive accuracy was determined by measuring area under the ROC curve-AUC, specificity and sensitivity. AUC of 0.7 was considered to be sufficiently discriminative; AUC of 0.5 is equivalent to a coin toss. The most suitable univariate was selected by comparing the area under the curves (AUC) from the ROC curves for Cut point and by performing logistic regression analysis.

The Kaplan-Meier (KM) method overall survival (OS) and relapse free survival (RFS) analysis was performed for six subgroups of patients. Classified on the basis of coexisting with various sellar lesion. The significant of the survival curve was assessed using the log-rank (Mantel- Haenszel) test.

**Divide into groups additional notes**

In the retrieved literature, we can see that the GH adenoma (acromegaly,Hematoxylin and eosin-H&E, immunohistochemistry-IHC) is the most common type. What causes this phenomenon? Base on this point view, we subdivided the pituitary adenoma group to separate the Somatotroph (acromegaly) adenoma.

**Clinically nonfunctional /silent adenoma -Some problems that need further explanation**

We classify according to the 2017 WHO classification of tumors of the pituitary gland, which is evolved from that based on the expression of adeno-hypophyseal hormones and immunohistochemical hormone expression and ultrastructural characteristics method. Hormone-negative adenoma are not equivalent to nonfunctioning adenoma, as IHC can be expressed in several adenoma types yet be clinically silent. The majority of nonfunctional adenoma literature are found to be gonadotroph adenomas and null cell tumors nearly half of nonfunctioning tumors. A significant subset of hormone-negative adenomas included silent Corticotroph adenoma. Silent ACTH adenoma, clinically silent GH PRL, and TSH adenoma are rare. Patients with nonfunctioning adenomas and acromegaly have also been shown to have a significantly higher inci­dence of malignancy than the general population (5). only around 2% of non-functioning pituitary neuroendocrine tumors lack signs of pituitary cell lineage differentiation, being thus classified as null-cell adenomas (1,2,3,4).

**Why combining chordoma and cranipopharyngioma?**

Despite they are not even remotely associated with each other (diagnostically). However, In terms of origin, embryogenic the notochordal originates from occipital bone, the notochord induces the outpouching of the roof of the stomodeal ectoderm that results in formation of Rathke’s pouch. Base on this point view, we put two disease in one subgroup

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**Identified sixty-seven literature meta-analyses**

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