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Yang H[1], Zhai W[1], Jiang Y[2], Kuo H[2]

1. *Department of Medical Research, Buddhist Tzu Chi General, Hualien, Taiwan,* ***2.*** *Department of Urology, Buddhist Tzu Chi General Hospital and Tzu Chi University, Hualien, Taiwan*

SIMILARITY AND DISSIMILARITIES OF KETAMINE CYSTITIS AND INTERSTITIAL CYSTITIS - A PROTEOMICS ANALYSIS AMONG KETAMINE CYSTITIS, INTERSTITIAL CYSTITIS AND NORMAL BLADDERS

Hypothesis / aims of study

To identify significantly differentially expressed proteins between (i) patients with interstitial cystitis/painful bladder syndrome (IC/PBS) and asymptomatic control (AC) subjects, (ii) patients with ketamine cystitis (KC) and AC subjects, and (iii) patients with IC/PBS and KC with the use of proteomic techniques.

Study design, materials and methods

Three patients each with KC and IC/PBS undergoing partial cystectomy and augmentation enterocystoplasty were enrolled consecutively. In the same time, 3 patients with bladder cancer or prostate cancer undergoing radical surgery who never had episode of urinary tract infection or irritative bladder symptoms were also included and serve as controls. The bladder wall specimens obtained during partial cystectomy and augmentation enterocystoplasty (IC/PBS and KC) or radical operations (radical cystectomy or radical prostatectomy, AC) was harvested and sent for pathological examination and urological laboratory for investigations. A proteomic approach was used to study the proteins associated with KC and IC/PBS and bioinformatics was used to construct the protein-protein network.

Results

A total of 31 proteins was significantly different between IC/PBS and KC bladders and these proteins were connected by protein-protein interaction network (Fig. 1). Nine of these proteins were up-regulated in KC (Table 1). Interestingly, 6 proteins including filamin, serpin peptidase inhibitor, CCT8 protein, 3-phosphoglycerate dehydrogenase, phosphoglycerate kinase and vinculin were observed only in IC/PBS (Table 2).

Interpretation of results

This study demonstrated that the etiology of IC/PBS and KC might be mediated by multiple signalling pathways.

Concluding message

The identified proteins contributing to the spectrum of IC/PBS and KC bladders may be used to elucidate the etiology of IC/PBS and KC and as candidate biomarkers for diagnostic test.

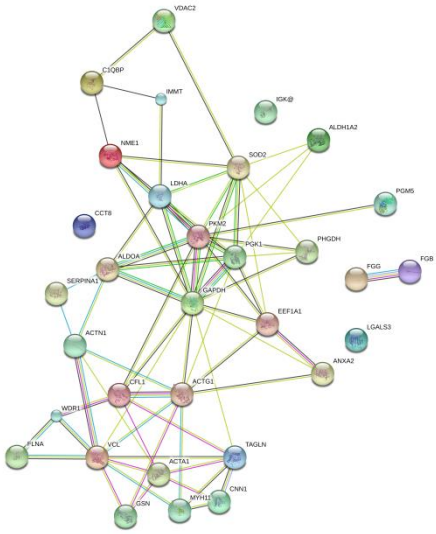

Fig. 1. The protein-protein interaction network of the identified differentially expressed proteins.

Table 1. Up-regulated proteins in KC bladders.

Protein Name	Gene Name	Score	Fold Diff. (IC/KC)
nucleoside diphosphate kinase A isoform a	NME1	51	-2.15973
voltage-dependent anion channel	VDAC2	292	-2.18246
manganese superoxide dismutase	SOD2	119	-2.29745
cofilin-1	CFL1	88	-2.40547
galactose-specific lectin	LGALS3	58	-2.48627
immunoglobulin kappa light chain VLJ region	IGK@	94	-2.82638

Annexin A2	ANXA2	424	-3.42068
complement component 1 Q subcomponent-binding protein, mitochondrial precursor	C1QBP	46	-3.72944
L-lactate dehydrogenase A chain isoform 1	LDHA	78	-4.086

Table 2. Proteins appeared in IC/PBS but not KC bladders.

Protein Name	**Gene Name**	**Score**	**Fold Diff. (IC/KC)**
Vinculin	VCL	753	+
filamin A, alpha (actin binding protein 280)	FLNA	422	+
Serpin peptidase inhibitor, clade A	SERPINA1	477	+
T-complex protein 1 subunit theta/ CCT8 protein	CCT8	254	+
3-phosphoglycerate dehydrogenase	PHGDH	80	+
Chain A, Crystal Structure Of Human Phosphoglycerate Kinase Bound To D-Adp	PGK1	359	+

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