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***Basic Study***

**Mast cell tryptase and** **carboxypeptidase A expression in body fluid and gastrointestinal tract associated with drug-related fatal anaphylaxis**

Guo XJ *et al*. Tryptase and carboxypeptidase A in anaphylaxis

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**Abstract**

**AIM:** To investigate the expression of mast cell tryptase and carboxypeptidase A in drug-related fatal anaphylaxis.

**METHODS:** The expression of mast cell tryptase and carboxypeptidase A in 15 autopsy cases of drug-related fatal anaphylaxis and 20 normal autopsy cases were detected. First, the expression of mast cell tryptase were determined in stomach, jejunum, lung, heart and larynx by immunofluorescence. Different tissues were removed and fixed in paraformaldehyde solution, then paraffin sections were made for immunofluorescence. Using specific mast cell tryptase and carboxypeptidase A antibodies, the expression of tryptase and carboxypeptidase A in gastroenterology tract and other tissues were observed under fluorescent microscope. Meanwhile, the postmortem serum and pericardial fluid were also collected from drug-related fatal anaphylaxis and normal autopsy cases. The level of mast cell tryptase and carboxypeptidase A in postmortem serum and pericardial fluid were measured using fluor enzyme linked immunosorbent assay (FEIA) and enzyme linked immunosorbent assay (ELISA) assay. The expression of mast cell tryptase and carboxypeptidase A were analyzed in drug-related fatal anaphylaxis cases compared to normal autopsy cases.

**RESULTS:** It was found that carboxypeptidase A was less expressed in gastroenterology tract and other tissues from anaphylaxis-related death cadavers. However, immunofluorescence result showed that tryptase expression was also increased obviously in multiple organs, especially gastrointestinal tract (46.67 ± 11.11 *vs* 4.88 ± 1.56 in stomach, 48.89 ± 11.02 *vs* 5.21 ± 1.34 in jejunum, 33.72 ± 5.76 *vs* 1.30 ± 1.02 in lung, 40.08 ± 7.56 *vs* 1.67 ± 1.03 in larynx, 7.11 ± 5.67 *vs* 1.10 ± 0.77 in heart, *P* < 0.05), from anaphylaxis-related death cadavers compared to normal autopsy cases. Meanwhile, FEIA result showed that the level of tryptase were significantly increased in both sera (43.50 ± 0.48 μg/L *vs* 5.40 ± 0.36 μg/L,*P* < 0.05) and pericardial fluid (28.64 ± 0.32 μg/L *vs* 4.60 ± 0.48 μg/L, *P* < 0.05) from anaphylaxis group in comparison with control group. The concentrations of carboxypeptidase A were also measured by a commercial ELISA kit. It was also increased more than two-fold in anaphylaxis group compared to control, which was as same as tryptase (8.99 ± 3.91 ng/mL *vs* 3.25 ± 2.30 ng/mL in serum, 4.34 ± 2.41 ng/mL *vs* 1.43 ± 0.58 ng/mL in pericardial fluid, *P* < 0.05).

**CONCLUSION:** Combination of mast cell tryptase and carboxypeptidase A detection could improve the forensic identification of drug-related fatal anaphylaxis.

**Key words:** Gastrointestinal tract;Drug-related fatal anaphylaxis; Forensic Pathology; Mast cell carboxypeptidase A; Mast cell tryptase

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**Core tip:** Drug-related fatal anaphylaxis could be occasionally encountered in forensic pathology routine. However, markers in the identification of drug-related fatal anaphylaxis still need further exploration. This study suggested two important markers in drug-related fatal anaphylaxis, which could improve postmortem diagnosis of anaphylaxis in medicolegal expertise.

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**INTRODUCTION**

Drug-induced anaphylaxis, also called allergic shock, is an immunologically mediated event that occurs after drug exposure in sensitized persons, which could constantly lead to death[1-3]. However, postmortem diagnosis of anaphylaxis is still difficult in medicolegal expertise. There was less different in the symptom between fatal anaphylaxis and general sudden death, the pathomorphology in anaphylaxis was also altered less[4,5]. In current autopsy case, the general disease, intoxication and violent death should be first excluded, and the exposure to allergen and the symptom in clinical observation were combined to identify the anaphylaxis[2,6]. Therefore, to explore novel, precise methods for anaphylaxis identification could be important in forensic pathology routine.

Drug-induced anaphylaxis is known to be initiated by foreign drugs binding to specific immunoglobulin E on mast cells[7,8]. Then various kinds of mediators are secreted from the mast cells, thereby inducing anaphylaxis[7,9,10]. Tryptase is a serine protease mainly stored in the granules of mast cells, which could be released at the onset of anaphylaxis[11]. Several studies had reported that serum tryptase levels could be a reliable indicator of anaphylaxis because of its long serum half-life compared to other secreted mediators[11-13]. However, the normal value of tryptase remains controversial in different countries. Thus, more precise standard should be investigated. Another chemical mediator, mast cell carboxypeptidase A, also attract the focus in postmortem diagnosis of anaphylaxis. Carboxypeptidase A is also a secreted protease in mast cells, which could be released during the activation of mast cells to mediated the process of acute anaphylaxis[14,15].

Therefore, whether the level of carboxypeptidase A or combining detection of carboxypeptidase A and tryptase could be meaningful in postmortem diagnosis of anaphylaxis. In this study, the expression of tryptase and carboxypeptidase A in multiple organs of cadaver was detected by immunofluorescence. Fluor enzyme linked immunosorbent assay (FEIA) and enzyme linked immunosorbent assay (ELISA) were also used to measure the level of tryptase and carboxypeptidase A in postmortem serum and pericardial fluid, respectively.

**MATERIALS AND METHODS**

***Immunofluorescence of tryptase in different tissues***

During autopsy, the stomach, jejunum, lung, heart and larynx were removed for fixation, paraffin embedding to make sections. The immunofluorescence of tryptase was performed as previously described with minor alterations[16]. Briefly, mouse anti-human mast cell tryptase, mouse anti-human carboxypeptidase A and rabbit anti-mouse IgG-TRITC (Santa Cruz, Chicago, USA) were used to mark tryptase in different organs. The sections were observed under fluorescent microscope (BX61, Olympus, Japan). 10 random visual field were imaged on each sections for analysis of tryptase-positive numbers. All experiments were approved by the Ethics Committee of Shanxi Medical University.

***Quantification of tryptase and carboxypeptidase A levels in serum and pericardial fluid***

Blood was collected from the right cardiac cavity and centrifuged. The serum and pericardial fluid was stored at -80°C until use. Samples from 35 autopsy cases were measured. The causes of death in anaphylaxis group (15 cases, 10 male, 5 female) included 3 of penicillin, 3 of ceftriaxone, 3 of levofloxacin, 5 of lomefloxacin *via* intravenous drip, 1 of ibuprofen *via* oral administration. The anaphylaxis was diagnosed by clinical features, the anaphylaxis symptoms occurs in all cases within 30 min. All postmortem autopsy were performed in 72 h. For control group, 20 cases without allergic reaction, craniocerebral injury, coronary heart disease and recreational drug use were selected. The level of tryptase in serum and pericardial fluid was measured by a commercial FEIA kit (Pharmacia Diagnostics, Uppsala, Sweden). Carboxypeptidase A levels were determined using ELISA kit (Huamei Bio, Wuhan, China) following its protocol.

***Statistical analysis***

Data were expressed as mean ± SE, student t test was used to compare differences between groups, *P* < 0.05 denotes significant.

**RESULTS**

***The expression of tryptase in different organs of anaphylaxis cadaver***

The immunofluorescence was performed to detect the expression of carboxypeptidase A and tryptase in different organs. However, less carboxypeptidase A was expressed in tissues from anaphylaxis cadaver (Data not shown). We next detected the expression of tryptase in different organs. Figure 1 showed that multiple tryptase-positive particle could be observed in mucous layer and less muscular layer in stomach and jejunum from anaphylaxis cadaver. In contrast, tryptase was less expressed in tissues from normal autopsy cases. We also detected the expression of tryptase in some other tissues. The tryptase could be observed in bronchia wall and small vessel wall in lung, small vessel wall in submucosa of larynx, periphery mesenchyme small vessels in heart. However, there was less tryptase-positive particle in tissues from control group (Figure 1, Table 1).

***Determination of tryptase and carboxypeptidase A in postmortem serum and*** ***pericardial fluid***

We examined tryptase in sera and pericardial fluid from 15 autopsy cases who died of anaphylaxis and 20 control cases. The level of tryptase were significantly increased in both sera and pericardial fluid from anaphylaxis group in comparison with control group (Table 2). The concentrations of carboxypeptidase A were also measured. It was also increased more than two-fold in anaphylaxis group compared to control, which was as same as tryptase (Table 3). Together results suggested that both tryptase and carboxypeptidase A were increased in drug-related fatal anaphylaxis.

**DISCUSSION**

Drug-induced fatal anaphylaxis is frequently occurred in medicolegal expertise. Some current indicators of anaphylaxis, including IgE and histamine, were lack of specify or stability[17-19]. Compared to other secreted mediators, tryptase and carboxypeptidase A have a long half-life in vivo, which speculated the superiority of the two proteases in postmortem diagnosis of anaphylaxis[20,21]. In the present study, we measured the levels of mast cell tryptase and carboxypeptidase A in postmortem serum and pericardial fluid. Schwartz, *et al*[22] had reported that the concentration of tryptase increased rapidly after allergic shock occurs, and it could be detected until 4 d autopsy. Moreover, the severity of allergic reaction could be highly related to the level of tryptase[13,23,24]. Although there were different standard of serum tryptase in normal adults among countries, this value which was higher than 10 μg/L could be considered abnormal[25,26]. We found that the level of tryptase in serum from anaphylaxis group was 8-fold higher than control. Meanwhile, this value in pericardial fluid was also about 6-fold. This results were consistent with previous studies, suggested that tryptase could be a specific, precise marker in the postmortem diagnosis of anaphylaxis. However, it was also reported that serum tryptase level increased in coronary heart disease, mastocytosis patients and some drug abuser[27-30]. Therefore, these cases should be excluded before diagnosis of anaphylaxis.

Another chemical mediator secreted from mast cells, carboxypeptidase A (also known as carboxypeptide A3, CPA3), was increased in allergic reaction, which was positively correlated to chymases[31,32]. Meanwhile, as tryptase, the carboxypeptide was also highly expressed in epithelial of asthma patients[33,34]. We confirmed that the level of carboxypeptidase A increased significantly in both postmortem serum and pericardial fluid from anaphylaxis cadaver. Although there was less investigation of carboxypeptide levels in the postmortem serum from anaphylaxis cases, it could speculated that the alteration of carboxypeptide was also meaningful. In combination with tryptase measurement, the two indicators could improve the postmortem diagnosis of anaphylaxis. Furthermore, to determine these mediators from the pericardial fluid in the closed serous cavity would also help avoiding the possible contamination after death.

During medicolegal expertise, the detection often developed long term after death. It increased the difficulty to obtain the serum or pericardial fluid samples. Therefore, to determine the expression of chemical markers in different organs from cadaver was also important. It was found that although carboxypeptidase A was less expressed in tissues from both normal and anaphylaxis cadaver, the expression of tryptase in stomach, jejunum, lung, heart, and larynx from drug-induced anaphylaxis group increased obviously compared to control group.

In conclusion, the expression of mast cell tryptase and carboxypeptidase A in body fluid and postmortem organs, especially gastrointestinal tract, could be meaningful in the identification of drug-related fatal anaphylaxis. Taking together with immunofluorescent identification, measurement of serum mast cell-specific tryptase and carboxypeptidase A levels might be a novel precise method which could improve postmortem diagnosis of anaphylaxis in medicolegal expertise. And the detection of tryptase level in postmortem organs could also be meaningful in the case that hard to collect serum/pericardial fluid and advanced state of decay during medicolegal expertise.

**COMMENTS**

***Background***

Drug-related fatal anaphylaxis could be occasionally encountered in forensic pathology routine. However, markers in the identification of drug-related fatal anaphylaxis still need further exploration.

***Research frontiers***

To explore novel markers and methods for the identification of drug-related fatal anaphylaxis could be important in forensic identification.

***Innovations and breakthroughs***

This article provide new evidences for using mast cell tryptase and carboxypeptidase A as biomarkers to identify drug-related fatal anaphylaxis. And it suggested that the expression of mast cell tryptase and carboxypeptidase A in gastroenterology tract and other tissues might be important marker in the case that hard to collect serum/pericardial fluid and advanced state of decay during medicolegal expertise.

***Applications***

Combination of mast cell tryptase and carboxypeptidase A detection could improve theforensic identification of drug-related fatal anaphylaxis.

***Peer-review***

The reviewers suggested some important modification in discussion section. We added some recent references and discussed the associated part to improve the discussion.

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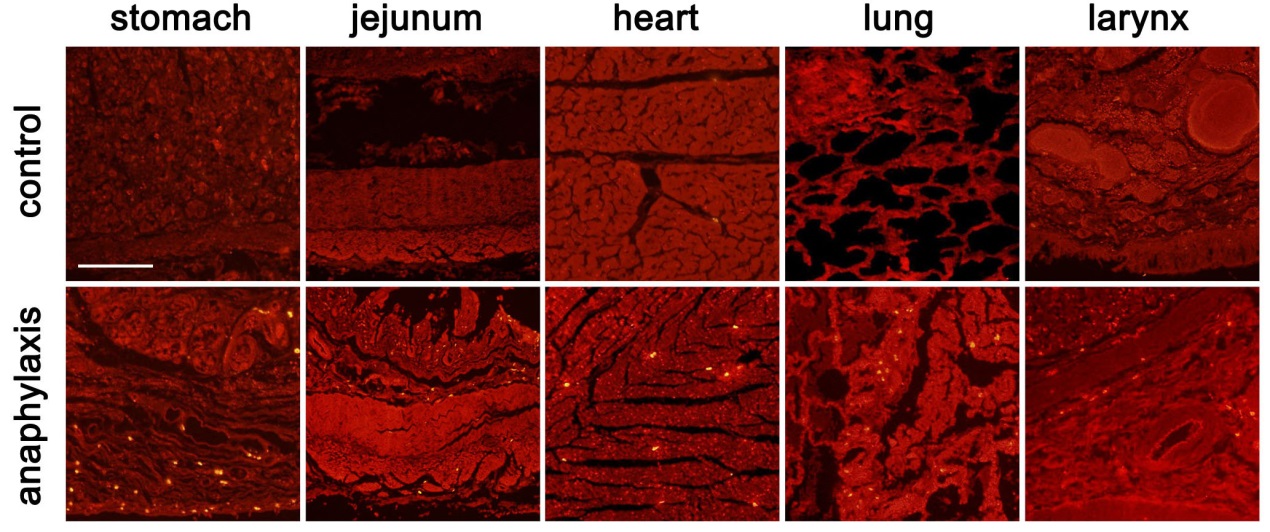
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**Figure 1 Immunofluorescence staining of tryptase in different organs. Scale bar = 200 μm and referred to all panels.**

**Table 1 Number of tryptase-positive particles in anaphylaxis and control group**

|  |  |  |
| --- | --- | --- |
|  | **Control (× 100)** | **Anaphylaxis (× 100)** |
| Stomach | 4.88 ± 1.56 | 46.67 ± 11.111 |
| Jejunum | 5.21 ± 1.34 | 48.89 ± 11.021 |
| Lung | 1.30 ± 1.02 | 33.72 ± 5.761 |
| Larynx | 1.67 ± 1.03 | 40.08 ± 7.561 |
| Heart | 1.10 ± 0.77 | 7.11 ± 5.671 |

1Denotes significant difference *vs* control, *P <* 0.05, *n* = 10.

**Table 2 The expression of tryptase in serum and pericardial fluid**

|  |  |  |  |
| --- | --- | --- | --- |
|  | ***n*** | **Serum (μg/L)** | **Pericardial fluid (μg/L)** |
| Control | 20 | 5.40 ± 0.36 | 4.60 ± 0.48 |
| Anaphylaxis | 15 | 43.50 ± 0.481 | 28.64 ± 0.321 |

1Denotes significant difference *vs* control, *P <* 0.05.

**Table 3 The expression of carboxypeptidase A in serum and pericardial fluid**

|  |  |  |  |
| --- | --- | --- | --- |
|  | ***n*** | **Serum (ng/mL)** | **Pericardial fluid (ng/mL)** |
| Control | 20 | 3.25 ± 2.30 | 1.43 ± 0.58 |
| Anaphylaxis | 15 | 8.99 ± 3.911 | 4.34 ± 2.411 |

1Denotes significant difference *vs* control, *P <* 0.05.