<table>
<thead>
<tr>
<th>項目</th>
<th>内容</th>
</tr>
</thead>
<tbody>
<tr>
<td>タイトル</td>
<td>腫白血球增多と急激な腰部痛に対する尿路結石の治療法</td>
</tr>
<tr>
<td>論文発表者</td>
<td>豊田雅史；奈園義文；宮本健二；広幡雅之；丸山邦男；原口篤</td>
</tr>
<tr>
<td>引用</td>
<td>泌尿器科紀要 (1988), 34(8): 1357-1361</td>
</tr>
<tr>
<td>発行日</td>
<td>1988-08</td>
</tr>
<tr>
<td>項目</td>
<td>内容</td>
</tr>
<tr>
<td>URL</td>
<td><a href="http://hdl.handle.net/2433/119680">http://hdl.handle.net/2433/119680</a></td>
</tr>
<tr>
<td>データタイプ</td>
<td>部門の詳細報告書</td>
</tr>
<tr>
<td>電子ファイルタイプ</td>
<td>電子ファイル</td>
</tr>
</tbody>
</table>
LEUCOCYTOSIS AND SEVERE PAIN DUE TO URETERAL CALCULI

Yasushi Toyoda, Yoshifumi Naitoh, Kenji Miyamoto, Yasuyuki Hiromoto and Kunio Maruyama

From the Department of Urology, Tokyo Metropolitan Hiroo General Hospital (Chief: Dr. K. Maruyama)

Chuh Haraguchi

From the Department of Urology, Showa University School of Medicine (Director: Prof. K. Imamura)

We reviewed the outcome of 300 cases without urinary tract infections to determine the relationship between leucocytosis and severe pain due to ureteral calculi. Leucocytosis above 9,000 cells per cmm was seen in 87.7% of the cases. Many patients (61%) had a leucocyte count between 9,000 and 12,000, but none had that exceeding 22,000. This leucocytosis can be attributed to a transient distributional alteration by means of leucopheresis to circulation sites from a storage pool such as the blood vessels in the spleen, liver, lung, etc. Since leucocytosis often accompanies ureteral calculi, caution should be exercised in distinguishing it from appendicitis.

Key words: Ureteral calculi, Leucocytosis

INTRODUCTION

Pain caused by ureteral calculi is often accompanied by leucocytosis. Therefore, it becomes occasionally difficult to differentiate it from atypical acute appendicitis. In 1937, Carp reported that leucocytosis was commonly found in the patients with ureteral calculi. In 93 of his 100 cases, the leucocyte count was over 7,000 and in 62 cases it was over 10,000. However, these cases had not been conceivably selected in a strict sense because they included the cases with pyuria (89%). We could not find any reports on the relationship between leucocytosis and severe pain caused by ureteral calculi. Therefore, we performed a clinical study on the relationship.

MATERIALS AND METHODS

The subjects were 300 cases visiting our department suffering from severe pain caused by ureteral calculi. No cases of urinary tract infections were included in this study. We decided that values above 9,000 represented leucocytosis. The leucocyte count was corrected based on the own normal level of erythrocytes because the number of leucocytes was actually often overcounted in the concentrated blood through dehydration depending upon water-intake insufficiency at the time of severe pain.

RESULTS

Fig. 1 shows the number of cases divided from the count of leucocytes at the time of severe pain. Leucocytosis was observed in 263 cases (87.7%). In many cases (61%) the leucocyte count was between 9,000 and 12,000, but in no cases did it exceed 22,000.

Leucocytosis was due to an increase in the number of neutrocytes while the concentration of lymphocytes was relatively decreased in most cases, and monocytes were slightly increased in a few cases.

Although the relationship between the extent of leucocytosis and the severity of pain did not always show a consistent pattern, in the cases with severe leucocytosis more time was required for the leucocyte count to normalize even after the pain disappeared.
Fig. 1. 300 patients with severe pain due to ureteral calculi classified according to leucocyte count. Those with a count over 9,000 accounted for 87.7%.

Fig. 2. A 36-year-old male with severe pain lasting for a long period. Leucocytes count increased one hour after the onset, and normalized many hours after spontaneous stone passage; arrow indicates use of analgesics.
For example, as shown in Fig. 2, a case suffering from more severe and longer lasting pain developed leucocytosis approximately one hour after the onset of pain and the leucocyte count exceeded 20,000 within 18 hours. This leucocytosis remained for 24 hours after the pain had completely disappeared by spontaneous stone passage.

On the other hand, in the case shown in Fig. 3, the response was less sensitive to severe pain. Leucocytosis developed slowly and was not so severe. This leucocytosis rapidly returned to the normal level whenever only pain improved.

As shown in Fig. 4 and 5, however, leucocytosis usually remained for about half a day after the recess of pain. In most of cases the leucocyte count gradually returned to the normal level within about one or two days when pain was relieved or disappeared.

**DISCUSSION**

A clinical analysis of 100 consecutive cases by Carp revealed pyuria in 89% of the cases. Therefore, we can assume that urinary tract infections were included in these cases. Moreover, the relationship between pain and leucocytosis has been
considered less reliable because 65% of the episodes were seen between attacks. In addition, we do not agree that values above 7,000 indicate leucocytosis.

Still colic attack is often accompanied with the increase of leucocytes. Leucocytosis above 9,000 was seen in as much as 87.7% of our cases. The percentage of leucocytosis may actually be much higher because the patients with a leucocyte count under 9,000 may include those with a lower than normal count in usual conditions, those whose leucocyte count is determined shortly before leucocytosis developed following a colic attack, and those whose response to severe pain is delayed.

No cases exceeded 22,000 leucocytes in our study. Although Carp reported that the leucocyte count was between 15,000 and 30,000 in 20% of his cases, the grade of leucocytosis and this percentage may considerably higher in those cases complicated with urinary tract infections.

By the exclusion of the cases with urinary tract infections in our study, reliable findings could be obtained on the relationship between the attack of severe pains and leucocytosis. However, the onset time and course of leucocytosis were not consistent, because they are probably associated with the severity, frequency and duration of pain and the individual differences in the response to pain.

Since the leucocytosis following the attack of severe pain caused by ureteral calculi is due to matured neutrocytes and the upper limit is transient and lower than that caused by inflammation, the leucocytosis can be classified as physiologic neutrophilia proposed by Wintrobe2; during strenuous exercise, after epinephrine injection, in association with convulsion or paroximal tachycardia, etc. That is, this leucocytosis can be attributed to a transient distributional alteration by means of leucopheresis2 to circulation sites from a storage pool such as the blood vessels in the spleen, liver, lung, etc.

REFERENCES


(Accepted for publication February 9, 1988)
和文抄録

尿管結石による疼痛と血中白血球増多症

都立広尾病院泌尿器科（医長：丸山邦夫）
蒙田 美，内藤 善文，宮本 憲治
桜本 泰之，丸山 邦夫
昭和大学医学部泌尿器科学教室（主任：今村一男教授）

尿路感染症を合併していない尿管結石症例 300 例を対象として、尿管結石による疼痛と血中白血球増多症について検討した。

9,000/cmm 以上の白血球増多症が、全例の 87.7 ％に認められた。9,000 ～ 12,000/cmm の白血球増多症が、全例の 61 ％と、ほとんどどの症例はこの範囲であった。22,000/cmm 以上の白血球増多症となる症例はなかった。

増加した白血球は好中多核白血球で、storage pool つまり臓器血管内に抑制されている白血球の流出による一時的な分布異常に基づくものであろう。尿路結石による激痛で血中白血球の増多症を起こすことは極めて普通のことであるから、急性炎症と鑑別の際などには留意しておかねばならない。
（泌尿器要 34：1357-1361, 1988）