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INFLAMMATORY PSEUDOTUMOR OF THE CERVIX: CASE REPORT AND REVIEW OF THE LITERATURE

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SUMMARY – The third recorded case of inflammatory pseudotumor of the cervix is reported. Inflammatory pseudotumor is considered to be a benign, self-limited lesion of obscure etiology. The condition is thought to be related to an unusual tissue response to injury, past infection and autoimmune disorders. Although extremely rare at this site, inflammatory pseudotumor should be considered in the differential diagnosis of a mesenchymal lesion or tumor-like inflammatory and reparative-like lesion. In December 2003, an 18-year-old nullipara presented to our hospital for dysfunctional bleeding. A solitary leiomyoma-like growth measuring 4.2x3.8 cm, originating from the cervix, was incidentally found on gynecologic examination. The tumor was surgically excised. Samples were stained by H&E, Gomori, pancytokeratin, vimentin, smooth muscle actin, myoglobin, desmin, S-100, CD-68, Factor VIII and p53. Microscopic evaluation of H&E samples showed a distinctive mesenchymal lesion composed of spindle cells displaying morphological features of myofibroblasts, fibroblasts and histiocytes mixed with a considerable number of inflammatory cells. Immunohistochemical analysis showed positive reaction to vimentin, smooth muscle actin, myoglobin, CD-68, and some tumor cells were unexpectedly positive for S-100. Gomori and Factor VIII emphasized the reticulin/capillary network. Three years after excision of the inflammatory pseudotumor of the cervix with no histologic evidence of malignancy, the patient is feeling well with no signs of relapse. In this as well as in other similar cases we suggest long-term follow up.

Key words: *Cervix disease – pathology; Cervix disease – immunohistochemistry; Plasma cell granuloma – pathology; Case report*

Introduction

Recently it has been recommended to use the term inflammatory myofibroblastic tumor for lesions called inflammatory pseudotumor (IPT), plasma cell granuloma, omental mesenteric myxoid hamartoma and inflammatory fibrosarcoma. The considerable morphological and clinical overlaps combined with both clinical and genetic evidence of their neoplastic nature led to the unification of these entities[1]. The use of the term inflammatory pseudotumor is now of historical significance, yet being most descriptive.

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IPT has become an almost ubiquitous tumorous condition. It occurs mainly in children and adolescents with a slight female predominance. Tumor size depends on the site, most often in the 5-10 cm range. There is recent evidence of the possible multifocal localization and even congenital recurrence[2]. Curiously enough, overall local recurrence develops in 14% of patients between three months and seven years. IPTs not confined to a single organ have an even higher chance of recurrence, especially if localized in the lungs (46%). Single organ IPT and adequate excision decrease recurrence to 1.5% and 8% in pulmonary and extrapulmonary IPT, respectively[3]. Metastatic disease, recorded in less than 5% of cases, can hardly be predicted on morphological grounds. According to some authors, cases with clearly atypical cytomorphology can be regarded as malignant[1].

The condition is thought to be related to an unusual tissue response to injury, past fungal or Epstein-Barr virus (EBV) infection, and autoimmune disorders. Although asymptomatic in many cases, the most frequent clinical symptoms include fever, night sweats, fatigue, lymphadenopathy, leukocytosis, hypogammaglobulinemia, erythrocyte sedimentation, weight loss, hypochromic anemia in IPT cases localized in the uterus, and symptoms secondary to the presence of the tumor.

During the past five years, more than two hundred cases of IPT have been described. They were mostly localized in the lungs, liver or spleen, not so often in the kidney, bladder, orbit or central nervous system, seldom in the pancreas, parotid gland, ureter, larynx or lymph nodes, and rarely in the testes, stomach or intestines. There are some unique cases dealing with the epidural space of the thoracic spine[4], ethmoid sinus[5], trigeminal nerve[6], diaphragm[7], and there are IPTs of cutaneous[8], oral[9,10] or pituitary[11] localizations.

We report on the third recorded case of IPT of the cervix[12,13]. Only seven previously described cases deal with IPT of the uterus[12-14].

Case Report

In December 2003, an 18-year-old nullipara presented to Dr. Ivo Pedišić General Hospital for dysfunctional bleeding. The patient reported no genitourinary, gastrointestinal or respiratory complaints. Her menstruation had been regular up to two weeks before the examination. During the preceding three years she was taking oral contraceptives. She reported no trauma or induced artificial abortion in her history.

A solitary leiomyoma-like growth measuring 4.2x3.8 cm, originating from the cervix was incidentally detected on gynecologic examination. The dimensions and site of the lesion were confirmed by ultrasound.

Surgical extirpation of the tumor was performed while preserving the rest of the uterus. According to the gynecologist performing the operation, the tumor could not be evacuated as easily as expected. Tumor mass seemed to have in part infiltrated the posterior wall of the cervix. The majority of the lower cervix was extirpated accordingly.

The operatively obtained specimen was paraffin embedded and 5-μm sections were stained by standard H&E and special histologic Gomori stain. In order to achieve definitive diagnosis the material was subsequently analyzed by the special histologic Gomori technique and an immunohistochemical palette consisting of pancytokeratin (Dako; 1:50), vimentin (Dako; 1:200), smooth muscle actin (SMA) (Dako, 1:125), myoglobin (Dako;1:300), desmin (Dako; 1:50), S-100 (Dako;1:300), CD 68 (Dako;1:75), Factor VIII (Dako; 1:25) and p53 (Dako; 1:25). Positive expression was analyzed descriptively and semiquantitatively.

Macroscopic examination proved to be difficult because the pathologist had to examine the tumor in pieces measuring from 0.7 to 4.5 cm in diameter. The cut surface of the tumor was solid, whorled and white-yellowish, with hemorrhage and necrosis areas. The border between the tumor and healthy cervix was indistinct.

Microscopic evaluation of distinctive mesenchymal lesion composed of spindle cells displaying morphological features of myofibroblasts, fibroblasts and histiocytes mixed with a considerable number of inflammatory cells revealed a biphasic pattern of tumor cells. The predominant population of mildly atypical but mitotically inactive spindle cells was arranged in interlaced fascicles sometimes growing in a focal whorling pattern within the polymorphous inflammatory infiltrate, mainly consisting of plasma cells and foamy histiocytes, while lymphocytes and neutrophils could be seen in smaller or larger clusters depending on different HPFs (Figs. 1 and 2). Some nuclei seemed to be plump and vesicular. The mass lacked the capsule and the border with the scant tissue of the uninvolved portion of the cervix was ragged.

Immunohistochemical staining showed positive reaction to vimentin, SMA, myoglobin, CD-68 and S-100.

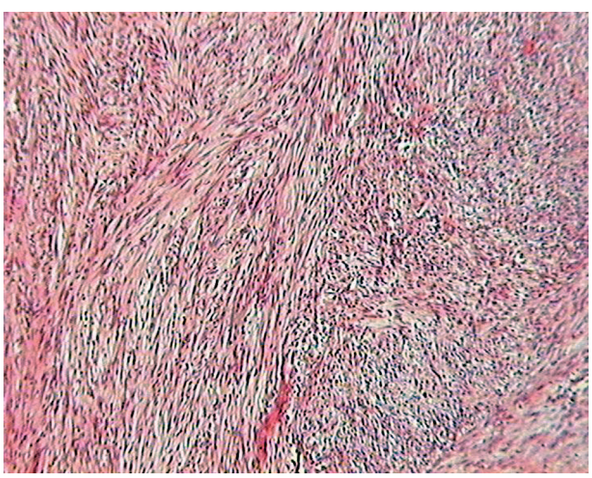

Fig. 1. Inflammatory pseudotumor of the cervix (H&E, X100).

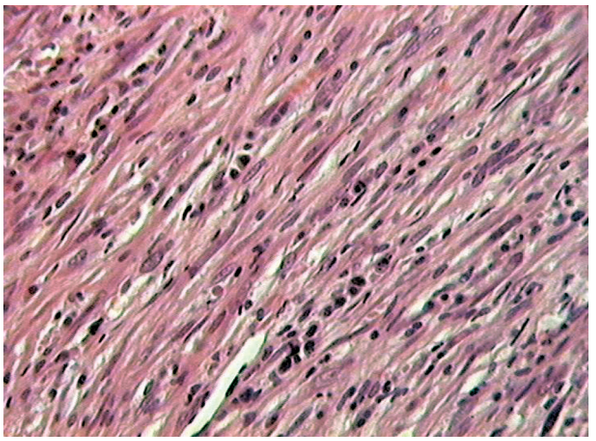

Fig. 2. Inflammatory pseudotumor of the cervix (H&E, X200).

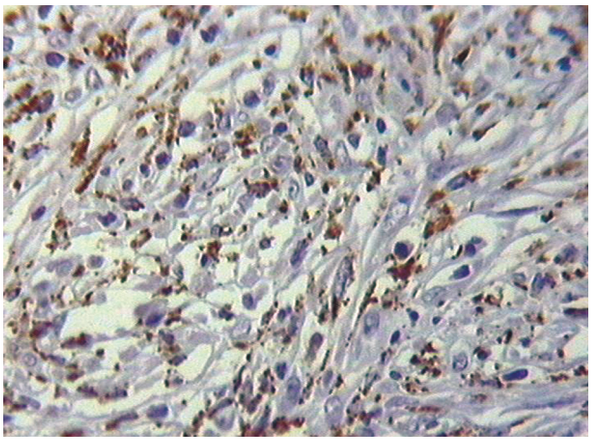

Fig. 4. Inflammatory pseudotumor of the cervix (CD 68, X400).

The cytoplasm of spindle cells showed diffuse and moderately to strongly intensive (++/+++) golden-brown positive reaction to vimentin, SMA (Fig. 3) and myoglobin. Some rare tumor cells that looked like spindle cells showed dusty-golden, moderately intensive (++) reaction of cytoplasm to S-100. The inflammatory infiltrate was positive for CD-68 but recognized only histiocytes with light brown, granular, diffuse and strongly intensive (+++) cytoplasmic reaction (Fig. 4). Pancytokeratin, desmin and p53 showed no expression in tumor samples. Factor VIII and Gomori emphasized the capillary IPT network, but tumor cells were negative.

Discussion

The term IPT was first introduced in 1939 by Brunn who described two lung tumors[18]. Its modern name which implied and linked inflammatory etiology and tumorous mass was introduced in 1954 by Umiker and Ivorsen[19]. The lungs are the usual location but recently there are an increasing number of reports of extrapulmonary sites with liver as the most common site. Nevertheless, IPT of any origin is considered to be a benign, self-limited lesion of obscure etiology which involves evidence supporting both infective and neoplastic processes.

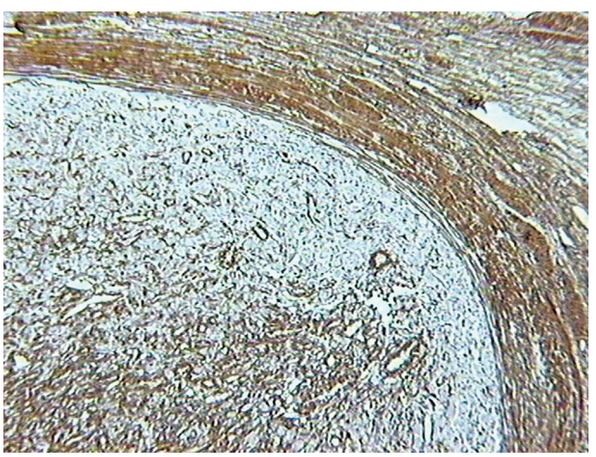

Fig. 3. Inflammatory pseudotumor of the cervix (SMA, X200).

There are many reports which associate IPT with *M. avium intracellulare*, *C. jejuni*, *B. sphaericus*, *C. burnetti*, EBV and *E. coli*. Most recently, Navai *et al*. have described IPT of the testes as part of an acute retroviral syndrome[20]. Association of IPT and previous abdominal surgery, trauma, ventriculoperitoneal shunt, radiotherapy, steroid usage, and some genetic factors has also been reported[21].

According to some authors, EBV is detectable in a large number of spindle cells in 66.7% of hepatic and 50% of splenic IPTs[22,23]. Spindle cells in cases of IPT are often interpreted as but not proven to be myofibroblasts. The finding of EBV in actin-positive spindle cells might suggest a similarity to EBV-positive smooth muscle tumors[22,23]. As most positive EBV tumors are localized in the liver and spleen, Chan considers them different from the IPT of organs and proposes the term IPT of follicular dendritic cell type[24]. However, Chan *et al.* firmly distinguish follicular dendritic cell sarcoma and IPT; the follicular cell type sarcoma has CD21 and CD35 immunoreactive cells and a low percentage of CD68 immunoreactive cells or even lack of inflammatory infiltrate along with morphological nuclear atypia[25]. Radhi *et al*. present an interesting case of a 7-year-old boy

with a retroperitoneal tumor mass mimicking IPT on needle biopsy. Surgical resection and histopathology revealed actinomycotic granules with associated suppurative inflammation. Gomori methenamine silver, although not pathognomonic of actinomycosis, was strongly supportive of the diagnosis[26].

As early as 1986, Dehner tried to finalize the IPT story concluding that it was a "self-limited polymorphic proliferative lesion whose etiology is unknown"[27]. However, there are some authors whose experience is contradictory with the pure inflammatory nature of IPT. This mostly relates to its potential for local recurrence, development of multifocal, noncontiguous tumors, infiltrative local growth, vascular invasion, clonal characteristics and malignant transformation[14]. Interestingly, these examples did not include any uterus IPT.

In addition, cytogenetic analyses revealed rearrangements of the anaplastic lymphoma kinase (ALK) gene and hence an overexpression of ALK protein in inflammatory myofibroblastic tumors, but not in sarcomatoid neoplasms[14,24,27]. According to these authors, ALK expression confirms the following: first, the distinctive clinicopathologic entity of IPT; second, it defines the etiology by revealing gene fusion at chromosome 2p23 resulting in ALK protein expression; third, it helps in differentiating from sarcomas which are negative for ALK; and fourth, positive expression is associated with favorable prognosis. The investigators believe that at least a subset of IPT is of neoplastic rather than of inflammatory origin[28-31].

In terms of purely morphological grounds, the non-neoplastic nature of our case was obvious. Myofibroblastic spindle cells, with mildly polymorphic nuclei and lack of pathological mitotic activity were demonstrated by positive diffuse and intensive staining of cytoplasm with vimentin, SMA and myoglobin. The interfering mononuclear cells included lymphocytes, plasma cells and many histiocytes, which were positive for CD 68. A fine capillary network, hardly seen in H&E samples, emerged after staining with Factor VIII, while Gomori made reticulin fibers visible. Unexplained positivity for S-100 appeared in some spindle cells. Although studies show consistent negativity for S-100, some particularly should be mentioned. Kroumpouzos *et al*. report S-100 positivity in a case of cutaneous Rosai-Dorfman disease, which presented histopathologically as an inflammatory pseudotumor[32]. Another case of lymph node myofibroblastoma, a lesion with many features in common with IPT, showed positivity of some spindle cells for S-100, which could not be explained by the authors. Ambrosiani *et al*. suggest that both lesions are a peculiar type or a different stage of abnormal lymph node reactivity[33]. There are also some controversies about cytokeratin expression. Most reports show consistent negativity, but there are some authors who found positive cytokeratin expression in tumor cells appearing as myofibroblastic[27,34,35]. Swanson *et al.* describe several cases of overlapping immunohistochemistry of malignant lesions[36]. Tumor spindle cells were negative to desmin. Interestingly, Trombetta *et al*. report positive staining of myofibroblasts to vimentin, cytokeratin and desmin in IPTs[34]. In our case IPT showed an absolute lack of cytokeratin and desmin expression. There was no positivity for p53. Reports on p53 expression in IPT are controversial and a uniform conclusion cannot be drawn from available investigations. Still, Hussong *et al*. confirmed that IPT tumors positive for p53 showed recurrence or malignant transformation[37]. An interesting study by Ledet *et al*. compared p53 expression for IPT and sarcoma. All sarcomas were positive for p53 and all IPTs were consistently negative, even the one that had recurred for several times and another one subsequently dedifferentiated to sarcoma[38]. Although p53 expression was positive in one of 15 IPT cases, while p53 gene missense mutation was revealed in two cases by polymerase chain reaction (PCR), Yamamoto *et al*. do not support the theory that p53 plays a major role in the pathogenesis of IPT[39].

Therapy consists of surgical resection, which is the most common and so far the best treatment, when it is possible. There is no uniform and unequivocal recommendation in the literature whether and when to use additional therapy. All data concerning adjuvant therapy like corticosteroids, radiotherapy and chemotherapy suggest early, frequent and prolonged vigilance, and in some cases it was successful[3,40]. However, additional therapy for IPTs that show a high risk of recurrence indicated by ill-defined margins or intra-abdominal, mesenteric, omental, and retroperitoneal localizations is strongly supported[41].

According to some authors, pronounced inflammatory reaction is the main characteristic of IPT. Therefore, the logical option is anti-inflammatory treatment suggested by Hakozaki *et al*.[42]. According to Su *et al*., a trial of NSAID treatment may both confirm the diagnosis of IPT and treat the tumor successfully.

Although extremely rare in the uterus, and especially in the cervix, IPT should be taken in consideration if differential diagnosis of a mesenchymal lesion of the

cervix is questionable, and if another non-neoplastic condition, especially a tumor-like inflammatory and reparative-like lesion, is a matter of differential diagnosis.

It may be said that the course of IPT is characterized by low but not insignificant possibility of malignant behavior including local invasiveness, recurrence or malignant transformation. Therefore, we suggest a palette of immunohistochemical agents with p53 or ALK in addition to standard immunohistochemical procedures. Negative p53 expression or positive ALK expression should be considered as a favorable IPT diagnosis.

According to the studies mentioned above, a lot about IPT needs to be clarified. First and foremost, the statement that IPT is of neoplastic origin according to cytogenetic and PCR results should be undeniably confirmed in larger series. Flow cytometry, FISH, karyotype analysis and detection of fusion oncogenes should be welcomed, and if possible, become a regular addition to the histopathologic work-up because it could help explain local recurrence or even malignant transformation leading to postoperative complications.

Controversial immunohistochemical findings challenge the pathologist to further investigate IPT and genesis of myofibroblasts. There are still open questions implied by karyotype abnormalities connected with malignant behavior. Were those IPTs malignant since the onset and not recognized as such at the moment of detection or did they just dedifferentiate?

Three years after surgical IPT excision with no histologic evidence of malignancy, and without any adjuvant therapy, our patient is feeling well, with no signs of relapse. In this as well as in other similar cases we suggest long-term follow up.

References

1. FLETCHER CDM. Soft tissue. In: FLETCHER CDM, editor. Diagnostic histopathology of tumors. 2nd ed. Toronto: Churchill Livingstone; 2000, p. 1500.
2. THOMPSON RJ, BARRETT AM, DILDEY P. Congenital multifocal inflammatory pseudotumor: a case report. J Pediatr Surg 2003;38:E17-9.
3. JANIK JS, JANIK JP, LOVELL MA, HENDRICKSON RJ, BENSARD DD, GREFFE BS. Recurrent inflammatory pseudotumors in children. J Pediatr Surg 2003;38:1491-5.
4. SEOL HJ, KIM SS, KIM JE, LEE SH, WON JY. Inflammatory pseudotumor in the epidural space of the thoracic spine: a case report and literature review of MR imaging findings. AJNR Am J Neuroradiol 2005;26:2667-70.
5. SAKURAI T, KAMADA H, YASUOKA Y, FURUYA N. Inflammatory pseudotumor arising in the ethmoid sinus: a case report. Nippon Jibiinkoka Gakkai Kaiho 2005;108:806-9.
6. JUNG TY, JUNG S, LEE MC, MOON KS, KIM IY, KANG SS *et al*. Hemorrhagic intracranial inflammatory pseudotumor originating from the trigeminal nerve: a case report. J Neurooncol 2005;76:139-42.
7. HÖER J, STEINAU G, FÜZESI L, GUNAWAN B, SCHUMPELICK V. Inflammatory pseudotumor of the diaphragm. Pediatr Surg Int 1999;15:387-90.
8. YUNG A, MERCHANT W, GOULDEN V. A case of isolated cutaneous pseudo-inflammatory tumour. Clin Exp Dermatol 2005;30:369-72.
9. KARTHIKEYAN BV, PRADEEP AR. Plasma cell granuloma of gingiva. Indian J Dent Res 2004;15:114-6.
10. YU E, MONTANERA W. Periodontoid pseudotumor: CT and MRI imaging. Neuroradiology 2005;47:328-33.
11. AL-SHRAIM M, SYRO LV, KOVACS K, ESTRADA H, URIBE H, AL-GAHTANY M. Inflammatory pseudotumor of the pituitary: case report. Surg Neurol 2004;62:264-7.
12. GÜCER F, ALTANER S, MÜLAYIM N, YAPICIER O. Invasive inflammatory pseudotumor of uterine cervix: a case report. Gynecol Oncol 2005;98:325-8.
13. ABENOZA P, SHEK YH, PERRONE T. Inflammatory pseudotumor of the cervix. Int J Gynecol Pathol 1994;1:80-6.
14. AZUNO Y, YAGA K, SUEHIRO Y, ARIYAMA S, OGA A. Inflammatory myoblastic tumor of the uterus and interleukin 6. Am J Obstet Gynecol 2003;189:890-1.
15. BEN-AROYA Z, BENHARROCH D, HALLAK M, KACHKO L, KATZ M. Inflammatory pseudotumor (plasma cell granuloma) of the pelvic cavity. J Reprod Med 2002;47:767-9.
16. BLAKE CB, TAYLOR GP, CLEMENT PB. Inflammatory pseudotumor of the uterus. Int J Gynecol Pathol 1987;6:275-86.
17. KARGI H, OZER E, GOKDEN N. Inflammatory pseudotumor of the uterus: a case report. Tumori 1995;81:454-6.
18. BRUNN H. Two interesting benign lung tumors of contradictory histopathology: remarks on the necessity for maintaining chest tumor registry. J Thorac Surg 1939;9:119-31.
19. UMIKER WO, IVERSON L. Postinflammatory "tumors" of the lung. J Thorac Surg 1954;28:55-62.
20. NAVAI N, YAP RL, GUPTA R, FRASER TG, GONZALEZ CM. Inflammatory pseudotumor of the testis: a novel presentation of acute retroviral syndrome. Int J Urol 2005;12:424-6.
21. KARNAK I, SENOCAK ME, CIFTCI AO, CAĐLAR M, BINGÖL-KOLOĐLU M, TANYEL FC *et al*. Inflammatory myofibroblastic tumor in children: diagnosis and treatment. J Pediatr Surg 2001;36:908-12.
22. ARBER DA, KAMEL OW, van de RIJN M, DAVIS RE, MEDEIROS LJ, JAFFE ES *et al*. Frequent presence of the Epstein-Barr virus in inflammatory pseudotumor. Hum Pathol 1995;26:1093-8.

23. ARBER DA, WEISS LM, CHANG KL. Detection of Epstein-Barr virus in inflammatory pseudotumor. Semin Diagn Pathol 1998;15:155-60.

24. CHAN JKC. Inflammatory pseudotumor: a family of lesions of diverse nature and etiologies. Adv Anat Pathol 1996;3:156-71.

25. CHAN JK, FLETCHER CD, NAYLER SJ, COOPER K. Follicular dendritic cell sarcoma. Clinicopathologic analysis of 17 cases suggesting a malignant potential higher than currently recognized. Cancer 1997;79:294-313.

26. RADHI J, HADJIS N, ANDERSON L, BURBRIDGE B, ALI K. Retroperitoneal actinomycosis masquerading as inflammatory pseudotumor. J Pediatr Surg 1997;4:618-20.

27. FREEMAN A, GEDDES N, MUNSON P, JOSEPH J, RAMANI P, SANDISON A *et al*. Anaplastic lymphoma kinase (ALK) staining and molecular analysis of inflammatory myofibroblastic tumours of the bladder: a preliminary clinicopathological study of nine cases and review of the literature. Mod Pathol 2004;17:765-71.

28. COOK JR, DEHNER LP, COLLINS MH, MA Z, MORRIS SW, COFFIN CM *et al*. Anaplastic lymphoma kinase (ALK) expression in the inflammatory myofibroblastic tumor: a comparative immunohistochemical study. Am J Surg Pathol 2001;25:1364-71.

29. COFFIN CM, PATEL A, PERKINS S, ELENITOBA-JOHNSON KS, PERLMAN E, GRIFFIN CA. ALK 1 and p80 expression and chromosomal rearrangements involving 2p23 in inflammatory myofibroblastic tumor. Mod Pathol 2001;14:569-76.

30. TREISSMAN SP, GILLIS DA, LEE CLY. Omental-mesenteric inflammatory pseudotumor. Cytogenetic demonstration of genetic changes and monoclonality in one tumor. Cancer 1994;73:1433-7.

31. SNYDER CS, DELL´AQUILLA M, HAGHIGHI P, BAERGEN RN, SUH YK, YI ES. Clonal changes in inflammatory pseudotumor of the lung. Cancer 1995;76:1545-9.

32. KROUMPOUZOS G, DEMIERRE MF. Cutaneous Rosai-Dorfman disease: histological presentation as inflammatory pseudotumor. A literature review. Acta Derm Venereol 2002; 82:292-6.

33. AMBROSIANI L, BELLONE S, CECCHETTI G, CERETTI E, MESSA E, TAVANI E. Lymph node myofibroblastoma: report of a submandibular case with peculiar morphologic and immunohistochemical characteristics. Pathologica 1994;86:541-5.

34. TROMBETTA G, ALO PL, BERLOCO P, CARICATO M, SCALPONE R, De QUARTO A, Di TONDO U. A case of multifocal inflammatory pseudotumor of the liver. Pathologica 1997;89:69-76.

35. SONOBE H, OKADA Y, SUDO S, IWATA J, OHTSUKI Y. Inflammatory pseudotumor of the urinary bladder with aberrant expression of cytokeratin. Report of a case with cytologic, immunocytochemical and cytogenetic findings. Acta Cytol 1999; 43:257-62.

36. SWANSON PE, MANIVEL JC, WICK MR. Immunoreactivity for Leu-7 in neurofibrosarcoma and other spindle cell sarcomas of soft tissue. Am J Pathol 1987;126:546-60.

37. HUSSONG JW, BROWN M, PERKINS SL, DEHNER LP, COFFIN CM. Comparison of DNA ploidy, histologic, and immunohistochemical findings with clinical outcome in inflammatory myofibroblastic tumors. Mod Pathol 1999;12:279-86.

38. LEDET SC, BROWN RW, CAGLE PT. p53 immunostaining in the differentiation of inflammatory pseudotumor from sarcoma involving the lung. Mod Pathol 1995;8:282-6.

39. YAMAMOTO H, ODA Y, SAITO T, SAKAMOTO A, MIYAJIMA K, TAMIYA S *et al*. p53 Mutation and MDM 2 amplification in inflammatory myofibroblastic tumours. Histopathology 2003;42:431-9.

40. SHAH MD, McCLAIN KL. Intracranial plasma cell granuloma: case report and treatment of recurrence with methotrexate and 6-mercaptopurine. J Pediatr Hematol Oncol 2005;27:599-603.

41. COFFIN CM, WATTERSON J, PRIEST JR, DEHNER LP. Extrapulmonary myofibroblastic tumor (inflammatory pseudotumor). A clinicopathologic and immunohistochemical study of 84 cases. Am J Surg Pathol 1995;19:859-72.

42. HAKOZAKI Y, KATOU M, NAKAGAWA K, SHIRAHAMA T, MATSUMOTO T. Improvement of inflammatory pseudotumor of the liver after nonsteroidal anti-inflammatory agent therapy. Am J Gastroenterol 1993;88:1121-2.

Sažetak

UPALNI PSEUDOTUMOR GRLIĆA MATERNICE: PRIKAZ SLUČAJA I PREGLED LITERATURE

I. Novosel, D. Babić, J. Ilić i *S. Seiwerth*

Prikazani slučaj je treći dosad opisani upalni pseudotumor grlića maternice. Upalni pseudotumor smatra se dobroćudnom, samoograničavajućom tvorbom nejasne etiologije. Povezuje se s neuobičajenim odgovorom tkiva na ozljedu, prethodnu infekciju ili s autoimunim poremećajem. Iako iznimno rijedak na ovome mjestu, upalni pseudotumor bi se diferencijalno dijagnostički trebao uzeti u obzir ako se radi o mezenhimalnoj tvorbi nalik tumoru ili reparativnoj promjeni. U prosincu 2003. u našu bolnicu je zaprimljena 18-godišnja nulipara zbog disfunkcionalnog krvarenja. Slučajni nalaz pri ginekološkom pregledu bila je solitarna tumorska masa nalik leiomiomu, koja je mjerila 4,2 puta 3,8 cm, podrijetlom iz grlića maternice. Tumor je kirurški odstranjen. U analizi tumorskih uzoraka primijenjeni su: H&E, Gomori, pancitokeratin, vimentin, SMA, mioglobin, desmin, S-100, CD-68, Faktor VIII. i p53. Mikroskopska analiza H&E uzoraka pokazala je karakterističnu mezenhimalnu promjenu građenu od vretenastih stanica koje pokazuju morfološke značajke miofibroblasta, fibroblasta i histiocita pomiješanih sa znatnim brojem upalnih stanica. Imunohistokemijska analiza pokazala je pozitivnu reakciju na vimentin, SMA, mioglobin, CD-68, dok su pojedinačne tumorske stanice bile neočekivano pozitivne na S-100. Gomori i Faktor VIII. naglasili su retikulinsko-kapilarnu mrežu. Tri godine nakon odstranjenja upalnog pseudotumora bez histoloških naznaka malignosti bolesnica se osjeća dobro i nema recidiva bolesti. U ovom kao i u drugim sličnim slučajevima preporučili bismo dugotrajno praćenje bolesnica.

Ključne riječi: *Bolest grlića maternice – patologija; Bolest grlića maternice - imunohistokemija; Granulom plazmatskih stanica – patologija; Prikaz slučaja*