

Comment on the Article „Classifications of Non-Specific Hematogenous Vertebral Osteomyelitis. Critical Review and Suggestions for Clinical Use“

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Topic of the paper seems rather actual while non-specific hematogenous vertebral osteomyelitis (NSHVO) being though a rare pathology as compared to other bones osteomyelitis but stipulates performance of a series of urgent, technically challenging treatment steps. The critical task of current vertebrology is to define clinical indications for such manipulations, their sequence depending on various surgical tactics.

The authors of commented article have rather extensive own experience in diagnostics and treatment of such patients undertook an attempt to conduct a comparative evaluation and introduce some supplements to the available classifications for spine osteomyelitis. Three classifications are analyzed: V.Y. Fischenko (1983) [1], Homagk et al (2016) [2] and Pola et al (2017) [3].

Classification of V.Y. Fischenko (1983) [1] is really qualifying as the basic one while considers cases of NSHVO in many aspects,

• *Comment on the Article*

Bazarov A.Yu. [Classifications of Non-Specific Hematogenous Vertebral Osteomyelitis. Critical Review and Suggestions for Clinical Use]. *Travmatologiya i ortopediya Rossii* [Traumatology and Orthopedics of Russia]. 2019;25(1):146-155. (In Russ.).


DOI: 10.21823/2311-2905-2019-25-1-146-155.

though, as authors correctly pointed, is not suggesting any tactical solutions. Probably the creator of the classification did not aim for this at that time.

The latest NSHVO classifications stipulate algorithms of tactical steps which, on the one hand, simplifies their acceptance, on the other hand, we inevitably lose some important diagnostic and treatment factors, such as consideration and objective evaluation of size and structure of vertebra lesion and secondary inflammatory centers, evaluation of tissue lesion dynamics prior to and during treatment, risk estimation for complications specific for NSHVO [2, 3, 4].

Still we suggest that the attempt to analyze diagnostic assessment approach in those classifications is efficient, though we are far from accepting any current classification as a unified and optimal. We are not aiming to judge on the value of Homagk et al [2] and Pola et al [3] classifications but would like to express our opinion.

There is an important theoretical question: how to present and evaluate such bone lesion signs like „bony destruction“, „destruction spondilodyscitis“ used in classifications of Homagk et al and Pola et al? Those terms reflect bone tissue status at the focus of vertebral lesion. In our opinion these terms are initially insufficiently defined, moreover

 **Cite as:** Grigorovsky V.V. [Comment on the Article „Classifications of Non-Specific Hematogenous Vertebral Osteomyelitis. Critical Review and Suggestions for Clinical Use“]. *Travmatologiya i ortopediya Rossii* [Traumatology and Orthopedics of Russia]. 2019;25(1):156-158. (In Russ.). DOI: 10.21823/2311-2905-2019-25-1-156-158.

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for their application towards infectious inflammation. On the one hand, focus of progressing inflammation with osteonecrosis, possible abscess formation and sequestration is accompanied by destruction. On the other hand, in case of successful sanitation of the focus, meaning elimination of the infectious inflammation process by conservative methods, the pathological remodeling in form of osteonecrosis and reossification will progress. Both processes may result in biomechanical instability and consequently to neurological deficit. Naturally the first option is much worse. Classifications considered in the article do not take into consideration those important pathomorphological features of bone lesion. On the contrary, we consider very important the objective findings in respect of size and status of tissues of the primary osteomyelitis focus in the vertebra for selection of NSHVO treatment tactics. In case visualization methods demonstrate a rather large focus in the vertebra with a trend to form a cavitation, moreover — sequester, it becomes a solid argument for surgical procedure aimed to open the pathology focus, remove necrotic tissues and to drain the area.

Subtype A.1 in the classification of Pola et al seems vague and controversial — „simple discitis without involvement of vertebral body“. In case of an error in classification we should not copy it in the improved version of classification suggested by authors of the commented article. Inflammation in the vertebral body most often starts are spondylitis in bony tissue adjacent to the disc or as an inflammatory focus in the deep body spongiosa, but not as an isolated discitis. We can discuss the scope and location of initial involvement of bony tissue and secondarily — on disc tissue, but size of inflammation focus is also important. Only in case of a hypothetical situation with dystrophic destruction process ingrowth of small vessels is possible through the endplate into the disc to result in pathological vascularization and ossification, then those vessels and sur-

rounding disc tissue can be the area for infection development.

Authors of the commented article do not give an adequate evaluation to this controversy which may contribute to incorrect perception.

In our opinion the notion „septic form of spine osteomyelitis“ used by authors is lame while the term „form“ is applicable first of all to the characteristics of the infection inflammation focus. Focus form is the pathomorphological notion. Being a pathologist who studied tissues of thousands of osteomyelitis focuses from various nosologies I can state that there are only three forms of osteomyelitis focus: 1) destructive form or abscess formation; 2) fibrosing or sclerotic form; 3) fibrosing form with micro abscesses [5]. Pathology focus initially, prior to treatment, classified as one form of osteomyelitis, during treatment or sometimes due to natural tissue sanitation can acquire another form. For example, we examined osteomyelitis focuses classified as fibrosing with micro abscesses upon first procedure, and as fibrosing in result of second procedure. The form of osteomyelitis focus most precisely reflects the features of this disease. Such notions as „sepsis“, „septic condition“ characterize the systemic complication of the local inflammation, that's why we think that descriptions of „spine osteomyelitis complicated by sepsis“ or „spine osteomyelitis with septic process“ are more appropriate.

Subdivision of separate A.5 and B.4 groups of spondylodiscitis accompanied by the systemic inflammatory response (SIR) is hardly efficient while SIR is observed in combination with other no less significant complications of NSHVO: bone destruction, epidural and paravertebral abscesses, biomechanical instability and neurological deficit. Classification of Pola et al states such complications are leading. Exactly these complications and not the level of CRP or even SIR define the evaluation of the clinical status of the patient and treatment tactics. Introduction of separate A.5 and B.4 subtypes leaves questions why

authors omitted other important complications in those groups: paravertebral abscesses, segmental kyphosis, biomechanical instability. Does confirmation of SIR makes all other complications insignificant? In our opinion, if the set of clinical, visualization, clinical and laboratory data suggests sepsis as complication to the NSHVO, it is sufficient to be reflected in the description of this or that subtype after its key specifics, which is done in the classification of Pola et al.

In our opinion it's hardly possible to establish an complete algorithm for clinical tactics in NSHVO. Much in the treatment is depending upon correct estimation of size, location, specifics and dynamics of the primary lesion, meaning evolution of its form, risk for development of secondary focuses in paravertebral tissues, systemic complications and other pathologies, for example, segment instability, neurological deficit, etc.

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