Dear Editor,

I read the case report by Ece et al. (1) with great interest for what I would like to extend my thanks. Such clinical entities are faced in almost all specialties of medicine and surgery what encourages to try to generalize them under the common term “implant migration” and to explain the possible similar underlying pathogenesis. Examples are migrated hernia meshes, hemostatic clips, contraceptive implants and so on (2, 3).

The question is: “Why implants migrate?” The most common cause of migration of hernia plugs has been shown to be poor surgical technique (4). Without any doubt, an implant should be fixed in place adequately what will probably prevent at least early migration. Adequately fixed implants (either meshes, clips or other implanted prostheses) can also migrate as a result of sliding via external forces and enter adjacent structures at points of least resistance (2).

Following the implantation of biomaterials, host reactions incorporate a combination of many processes including blood-material interactions, inflammation (acute then chronic), development of granulation tissue, foreign body reaction, and fibrous capsule development (5). Blood-material interaction, which is characterized by protein adsorption to the implant surface and formation of transient provisional matrix, is followed by acute inflammation which lasts about two weeks (5). If implant infection occurs, acute inflammation can persist beyond the third week and subsequent phases of host reaction, such as granulation and fibrous capsule formation, can be defective. This means that the implant will not be adequately fixed in place by fibrous tissue and can migrate easily conquering surrounding tissues at points of least resistance. This pathogenesis concerns surgically-implanted biomaterials that are embedded in tissues, and all sides and/or parts are surrounded by tissues.

A different type of implants is intraluminal implants that are generally implanted by means of endoscopy or interventional radiology, such as bands, clips, stents, which would disconnect from previously contacting tissues or migrate along the lumen rather than migrate to adjacent tissues or organs. This is because the least resistance around the implant is the lumen of the hollow organ.

It is worth emphasizing that another distinct type of implants is “pressure-applying” implants, such as vertical banded gastroplasty meshes, which tend to migrate toward the vector of applied pressure, that is, the gastric lumen (1). The pathogenesis here is again migration toward least resistance, the constricted gastric wall being the site of least resistance.

To conclude, the pathogenesis of migration of different implants is the same. Therefore, they are “different shoes walking the same path”. This clinical entity should probably be generalized under the unique term “implant migration”.

REFERENCES