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Biologic Implications of Taper Corrosion in Total Hip Arthroplasty

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The potential for corrosion at the modular head-neck junction in total hip arthroplasty has been well described, however the associated biological implications have recently received increasing clinical attention. Taper corrosion has been reported in patients with metal-on-polyethylene bearing surfaces, but it has also been recognized as an increasing cause of failure in patients with large head metal-on-metal hip replacements. Mechanical factors such as taper geometry, stem design, head size, or neck length may play a role in the etiology of taper corrosion. It can produce a range of clinical symptoms including pain, weakness, and instability that result from adverse local tissue reactions. While systemic effects have been reported, these remain poorly understood at present. A diagnostic algorithm to taper corrosion is provided, and treatment options are reviewed.

KEYWORDS adverse local tissue reaction, corrosion, metal debris, pseudotumor, taper

Modularity at the head–neck junction of the femoral component offers several advantages in total hip arthroplasty (THA), including intraoperative flexibility, decreased implant inventory, and the ability to change the head at the time of future surgery.[1] Yet these advantages do not come without a cost. The potential for corrosion at this modular junction was first described in the early 1980s.[2] Although there were numerous early concerns of fretting and crevice corrosion associated with modularity,[3-7] improvements in taper design and manufacturing have enabled head–neck modularity to become near-universal in modern THA. Accordingly, corrosion at this modular junction has received relatively little attention in recent years.

In the first generation of modular heads, corrosion at this interface was only identified through retrieval analyses[1,3,7-9,10-12] or in rare cases of catastrophic failure.[1,5] Although there have been reports of elevated serum metal levels[13] and particle deposition within local tissues secondary to corrosion at the modular head–neck junction,[14] adverse local tissue reactions (ALTRs) associated with corrosion of modular implants have rarely been described. Our goal is to review the recent literature on the biologic implications of taper corrosion (including current concepts regarding the diagnosis, management, and etiology of this complication), and to report our experience in managing these patients.

Adverse Local Tissue Reactions

In the last decade, adverse local tissue reactions have become better understood as a potential complication of metal-on-metal (MoM) hip resurfacing arthroplasty and total hip arthroplasty.[15-18] Histologic examination of periprosthetic tissues in these cases often demonstrates large areas of necrosis and areas of chronic inflammatory reaction and perivascular lymphocytic aggregates, findings often referred to as aseptic lymphocyte-dominated vasculitis-associated lesions (ALVAL). However, ALTR is not exclusive to these patients and also has been reported in patients with a metal-on-polyethylene bearing (Table 1).

Review of the Literature

Svensson et al was the first to report on an aggressive soft-tissue reaction secondary to taper corrosion in 1988.[19] Presenting symptoms were severe pain and muscle weakness within 3 years of undergoing THA, and upon surgical exploration, a large necrotic soft-tissue mass was encountered and a black deposit was found at the head–neck junction. Analysis of the taper junction from the explanted prosthesis demonstrated evidence of a severe corrosive process. The patient ultimately underwent multiple debridements

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Table 1 Summary of Patients Revised for Corrosion at the Head–Neck Taper. Data is Derived from Published Reports as well as Patients Revised at our Institution Since Publication of our Experience

Study	Age (years)	Sex	Femoral Component	Taper	Head	Interface	Years to Revision
Cooper	66	F	Zimmer VerSys Beaded FullCoat	12/14	32 mm, + 3.5	Co-alloy/Co-alloy	3.4
Cooper	65	F	Zimmer VerSys Beaded FullCoat	12/14	28 mm, + 10.5	Co-alloy/Co-alloy	6.5
Cooper	69	F	Zimmer VerSys Beaded FullCoat	12/14	36 mm, + 7	Co-alloy/Co-alloy	4.3
Cooper	58	F	Zimmer VerSys Beaded FullCoat	12/14	28 mm, + 10.5	Co-alloy/Co-alloy	5.1
Cooper	41	F	Zimmer VerSys Beaded FullCoat LHC	12/14	32 mm, + 5	Co-alloy/Co-alloy	2.1
Unpublished	69	M	Zimmer VerSys Beaded FullCoat LHC	12/14	40 mm, + 7	Co-alloy/Co-alloy	2.4
Unpublished	59	F	Zimmer VerSys Beaded FullCoat LM	12/14	32 mm, + 0	Co-alloy/Co-alloy	3.5
Cooper	61	M	Zimmer VerSys Beaded FullCoat Revision	12/14	32 mm, + 10.5	Co-alloy/Co-alloy	0.8
Unpublished	65	F	Zimmer Epoch FullCoat	12/14	36 mm, + 0	Co-alloy/Co-alloy	3.3
Cooper	46	M	Zimmer VerSys Fiber Metal Taper	12/14	32 mm, −3.5	Ti-alloy/Co-alloy	5.1
Cooper	62	F	Zimmer M/L Taper, Kinectiv Modular Neck	12/14	32 mm, + 0	Ti-alloy/Co-alloy	0.7
Svensson	60	F	Benoist Girard Lord Prosthesis	(n/a)	(n/a)	Co-alloy/Co-alloy	3.5
Cooper	55	F	DePuy Bantam Full Porocoat	11/13	28 mm, + 5	Co-alloy/Co-alloy	8.9
Walsh	79	M	DePuy Prodigy Full Porocoat	12/14	36 mm, (n/a)	Co-alloy/Co-alloy	2
Cooper	70	F	Stryker Accolade	V40	36 mm, + 0	Ti-alloy/Co-alloy	2.2
Meftah	83	F	Stryker Accolade	V40	28 mm, (n/a)	Ti-alloy/Co-alloy	3
Lindgren	70	M	Stryker Accolade	V40	28 mm, + 4	Ti-alloy/Co-alloy	2.8
Mao	71	F	Stryker Accolade	V40	32 mm, (n/a)	Ti-alloy/Co-alloy	7
18 Patients	63.8	72% F 28% M					3.7

and a resection arthroplasty, and was left with a completely denervated leg with a draining wound and both arterial and venous insufficiency; she declined the option of a hemipelvectomy.

To the best of our knowledge, there were no additional case reports of ALTR from head–neck corrosion in patients with a metal-on-polyethylene bearing until 2010, yet at the time this review was prepared, there have been four since that time (Table 1). Meftah et al[20] reported surgical findings of abnormal soft-tissue reaction, a large collection of turbid fluid, and visible products of corrosion at the modular head–neck junction 3 years following THA; histology of the tissue was consistent with ALVAL. Lindgren et al[21] reported a large adverse tissue reaction that led to pain and recurrent instability at 2 years after primary THA; operative findings at revision were consistent with necrotic tissue and corrosion at the head–neck junction; histology confirmed lymphocytic infiltration and large areas of necrosis. A subsequent Canadian report from Walsh et al documented an inflammatory pseudotumor causing pain, weakness, and an enlarging buttock mass less than 2 years after the index surgery[22]; histologic findings demonstrated necrotic material and a palisading chronic inflammatory reaction of lymphocytes and histiocytes, focally forming perivascular lymphocytic aggregates. Most recently, a case report by Mao et al[23] described a presentation of intractable trochanteric bursitis with a large palpable trochanteric mass 7 years after THA; after multiple failed attempts to manage the bursitis, the hip was surgically explored and significant corrosion was found at the modular head–neck junction. The authors describe a 20-cm long turbid fluid-filled trochanteric cyst found to be in direct communication with the hip joint, with histologic features consistent with ALVAL.

Our Experience

Since 2009, we have revised 13 patients for a diagnosis of corrosion at the modular head–neck junction. We recently published our results on 10 of these 13 patients,[24] but present our most recent data here. Unfortunately as we become more familiar with the clinical manifestations of this entity, we continue to accumulate more cases and presently have several more patients awaiting revision surgery.