Platelet-Rich Plasma Application Does Not Influence Heterotopic Bone Formation Following Total Hip Arthroplasty

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Statement of Purpose: Heterotopic ossification (HO) between the proximal femur and the pelvis can occur following total hip arthroplasty (THA) at an overall incidence of about 43% (1). Extensive HO can result in ankylosis with limitation of motion and painful impingement. During THA, reaming spreads marrow, along with osteogenically competent cells, into well-vascularized muscle. Signaling molecules, including TGF-β, are thought to operate upon these progenitor cells and lead to HO. Autologous platelet-rich plasma (PRP), intraoperatively prepared and applied to the surgical site, may positively influence wound healing and reduce complications. (2) However, activated platelets release TGF-β and other signaling proteins and PRP's effect on HO is unknown. We performed a retrospective clinical study to test the hypothesis that the incidence of HO in a THA-PRP cohort would be greater than that in a THA-Control cohort. If true, this would suggest that PRP be used cautiously in THA procedures.

Methods: This retrospective study examined 179 THA's performed in 167 patients by a single surgeon (Mark A. Klaassen, M.D., Elkhart, IN) with a primary diagnosis of osteoarthritis. Using a posterior lateral approach, uncemented femoral and acetabular components were implanted, with either metal-on-metal or metal-on-polyethylene articulation. Nonsteroidal anti-inflammatory drugs (NSAIDs) were stopped two weeks before surgery and were not routinely prescribed postoperatively. In the THA-PRP cohort, ~6 ml of autologous, thrombin-activated PRP was prepared using the Gravitational Platelet System (Biomet Biologics, Warsaw, IN) and sprayed on the exposed bone, synovium, capsule, and muscle during closure. In the THA-Control cohort, PRP was not applied. Table 1 summarizes the patient demographics. Radiographs (anterior-posterior and lateral) were taken immediately post-op, then at 6 weeks, 3 months, and 1 year. HO was graded per the Brooker Classification (3) and compared using the Chi Square test with significance taken as $p<0.05$

Table 1. Summary of patient demographics.

	THA-PRP	THA-Control
Dates of Surgery	1/2004-2/2006	3/1999-12/2003
No. Patients	76	91
No. Hips	85	94
Male/Female	34/42	48/43
Age (years)	71.6 range:48-91	68.4 range:22-92

Results: Figure 1 shows a radiograph exhibiting HO. Data is summarized in Table 2. HO incidence, THA-PRP (12.9%) and THA-Control (21.3%), and severity were not significantly different ($p=0.478$) so the study hypothesis was false. Typically, HO is radiographically visible by 4-6 weeks after surgery and the incidence and extent does not increase beyond six months, so the one year follow-up was sufficient. The etiology of HO formation following THA is believed to involve four steps, 1) an inciting event such as trauma, 2) a signal from the injury site, 3) a supply of mesenchymal cells which, given an appropriate signal, can differentiate into osteoblasts and chondroblasts, and 4) an appropriate environment conducive to the continued production of heterotopic bone. Consequently, there could be multiple points along the HO cascade with which platelets could potentially interact. It is difficult to place the apparent null effect of PRP on HO in a theoretical context since little detail is known about the actual mechanism of HO. One study limitation was that blood transfusion use, pain, and functional return were not included as outcome measures.

Table 2. Incidence of HO by Brooker grade

	None	Grade I	Grade II	Grade III	Grade IV
Control	74	11	7	2	0
PRP	74	7	4	0	0

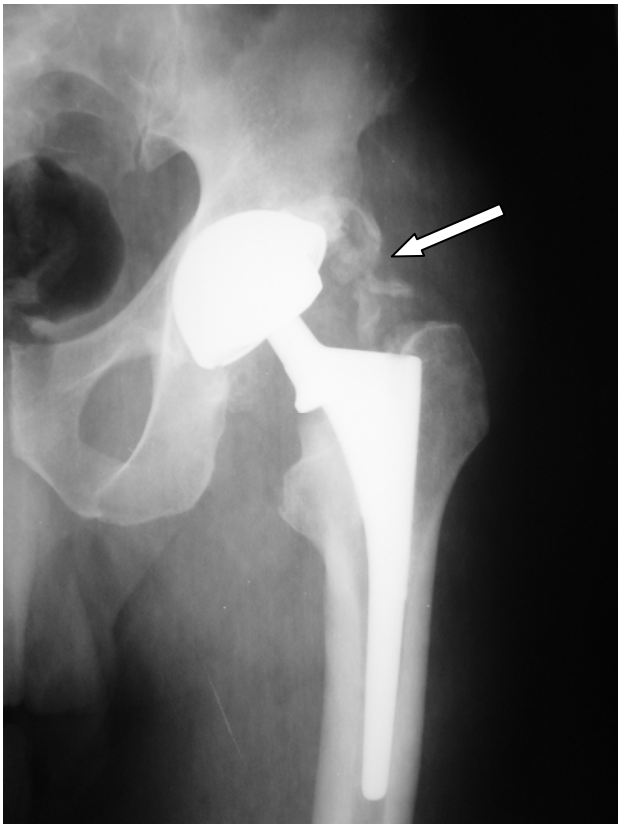

Figure 1. HO bone (see arrow) in a THA-Control hip.

Conclusions: This retrospective clinical study did not show any significant influence of PRP on the incidence or severity of HO following THA. This suggests that PRP can be applied during THA without potentiating this complication. Future work that includes other outcome measures would help clarify the role of PRP in THA.

References:

1. Neal B, ANZ J Surg 2002;72:808-821
2. Pietrzak WS, J Craniofac Surg 2005;16:1043-1053.
3. Brooker AF, J Bone Joint Surg 1973;55-A:1629-1632