Journal of Health Sciences

CASE REPORT Open Access

Stromal vascular fraction and autologous activated platelet-rich plasma cellular therapy in the treatment of ankylosing spondylitis: A case series

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ABSTRACT

Introduction: Ankylosing spondylitis (AS) is a chronic inflammatory autoimmune disorder manifesting as chronic back pain and progressive spinal rigidity, which heavily affects patients' quality of life. Regenerative cellular therapy has the potential as a treatment modality for AS. In this case report, we reported two cases of male patients with AS treated with combined stromal vascular fraction (SVF) and autologous activated platelet-rich plasma (aaPRP).

Case Report: Two cases of 55-year-old and 58-year-old male patients presented to Hayandra Clinic with chronic back pain and progressive spinal stiffness. Both patients presented with "high" and "very high" disease activity according to the AS disease activity score – C-reactive protein (ASDAS-CRP) were treated with four treatments of the SVF and aaPRP combination, followed by four aaPRP treatments. The patients showed a reduction of disease activity and CRP to inactive disease as measured through the ASDAS-CRP score.

Conclusion: Cellular therapy with SVF and aaPRP has the potential to reduce disease activity through modulation of inflammation and regeneration of damaged tissues in active disease of AS patients.

Keywords: Ankylosing spondylitis; autoimmune disorder; platelet-rich plasma; stromal vascular fraction

INTRODUCTION

Ankylosing spondylitis (AS) is a chronic autoimmune axial spondyloarthritis affecting the spine, sacroiliac joints, and adjacent soft tissues. HLA-B27–mediated inflammation leads to fibrosis, calcification, and eventual fusion of the spine, with early symptoms, including back pain, stiffness, and reduced flexibility. Onset typically occurs in the second to third decade of life, severely impacting quality of life, and can progress to the characteristic rigid "bamboo spine" (1-3).

Present management involves long-term biologics and nonsteroidal anti-inflammatory drugs (NSAIDs) to control inflammation and symptoms, but these regimens do not reverse structural damage. Prolonged NSAID use raises safety concerns, while biologics carry risks, such as infection and tuberculosis reactivation, particularly in endemic regions, such as Indonesia. Many patients require multiple biologic agents over years, with only a third ever discontinuing therapy (2-6). Given these limitations, halting inflammation and regenerating damaged tissue through cellular therapy offers promise. We report two cases of AS treated with combined stromal vascular fraction (SVF) and autologous activated platelet-rich plasma (aaPRP).

SVF was obtained from ~500 mL of abdominal fat collected by manual lipoaspiration using a 2.4 mm cannula with tumescent anesthesia. The lipoaspirates were processed in fractions: 60% of the lipoaspirate was allocated for primary SVF (SVF-1) isolation, while the remaining 40% was used for repeat SVF isolation (SVF-2). In addition, one-quarter (1/4) of the SVF-1 product was further subdivided to generate secondary fractions, with 3/5 allocated to SVF-3 and 2/5 to SVF-4. The lipoaspirates were then digested with an in-house xenofree enzymatic process and incubated at 37°C, 300 rpm for 1 h. Enzymes were inactivated with low-glucose (1 g/L) DMEM containing 4 mM L-glutamine, followed by centrifugation at 600 x G for 5 min. The SVF pellet was resuspended in saline, and cell count/viability was assessed by trypan blue staining (7).

aaPRP was obtained through withdrawal of 24 mL venous

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Submitted: 23 September 2025/Accepted: 20 July 2026

DOI: https://doi.org/10.17532/jhsci.2026.2934

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blood from the patient, which underwent separation in sodium citrate blood collection tubes and was further centrifuged at 188 x G for 10 min. The plasma layer underwent a second centrifugation at 1690 x G for 10 min, leaving 2.5 mL plasma with platelet pellets resuspended as inactivated PRP. BEST Calcium activator (0.15 mL, Hayandra Inspira Medika, Indonesia) was added to form clots, which were removed before adding 10 mL normal saline. The PRP was then light-activated (AdiLight-1, Adistem Ltd., Hong Kong) for 20 min. For administration, the SVF pellet was resuspended in saline to obtain a 15 mL suspension, mixed with 3 mL aaPRP, and infused intravenously in 100 mL saline over 30 min. For paralumbar delivery, 3 mL of the SVF–aaPRP mixture was injected into the lumbar spine (7,8).

Ethical statement

Written informed consent was obtained from both patients for publication of this case report and any accompanying images. All procedures were conducted in accordance with the ethical principles of the Declaration of Helsinki. Patient anonymity and confidentiality were maintained throughout the study and manuscript preparation.

CASE REPORT

Case illustration 1

A 55-year-old male patient presented with worsening low back pain (visual analogue scale [VAS] 5) and spine mobility limitation for the past 5 years. He was diagnosed with AS based on physical examination and HLA-B27 test and was given daily oral sulfasalazine 4 × 500 mg. On presentation, his C-reactive protein (CRP) level was very high (94.5 mg/dL). Upon scoring with the AS Disease Activity Score – CRP (ASDAS-CRP), the patient showed a very high disease activity. The patient was then continued with oral sulfasalazine and underwent a total of four intravenous SVF and aaPRP cell therapy with a 2-week interval in between therapies, and continued with four IV aaPRP treatments only (Table 1). Administration of the treatment regimen was given through ultrasound-guided paralumbar injection. Two months after four SVF and aaPRP treatments, the patient's CRP level dropped to 2.7 mg/dL with no back pain and without NSAID consumption, which indicated an inactive disease state. The patient subsequently underwent four additional individual IV aaPRP therapies with a 2-week interval before undergoing monthly aaPRP treatment. On 1 year follow-up, the patient did not experience any flare or symptoms and has been off his sulfasalazine medication for 2 months.

Case illustration 2

A 58-year-old male patient presented with worsening neck and back stiffness and low back pain (VAS 2–3) for the past 3 years. He was diagnosed with AS based on radiographic imaging and was treated with NSAIDs for pain relief. Upon presentation, he had stooped posture with thoracic kyphosis, cervical flexion, and right hip flexion contracture (Figure 1). On physical examination, there was range of motion limitation on his neck, back, and shoulders. His high-sensitivity-CRP level was 0.18 mg/dL. He was assessed with high disease activity on ASDAS-CRP score of 3.0 (Table 1). This score reflects the combined contribution of substantial morning stiffness, a high patient global assessment driven by significant functional limitation and deformity, as well as a contribution from CRP through logarithmic transformation, resulting in a total score within the high disease activity range.

The patient was treated with the same treatment regimen and interval with the previous case through ultrasound-guided paralumbar injection. The number of cells and viability of SVF cells are shown in Table 1. Upon finishing the SVF and aaPRP combination treatment regimen, based on physical examination and CRP level, he had inactive disease with ASDAS-CRP score (Table 1). The patient was then continued with monthly aaPRP treatment. On 2 months follow-up, he reported no flare of disease.

DISCUSSION

AS is a chronic autoimmune disease classified under axial spondyloarthritis (axial SpA), with onset typically in the second to third decade. Early symptoms, such as back pain are often overlooked or misdiagnosed, delaying treatment until spinal rigidity has progressed (9,10). Both of our patients fulfilled the Assessment of Spondyloarthritis International Society (ASAS) criteria for AS (2,11). Both patients presented with chronic back pain, spinal stiffness, and elevated inflammatory markers; one was HLA-B27 positive, while the other demonstrated radiographic sacroiliitis.

Disease activity was assessed with the ASDAS-CRP, which showed very high activity in case 1 and high activity in case 2. Our case 1 patient had visited an internist 1 year before presentation to our clinic and had been treated with NSAIDs and sulfasalazine. He presented to our clinic with a very high disease activity, indicating an inadequate

TABLE 1. Summary of disease progression after treatment with SVF and aaPRP

Case	Age, Sex	Treatments	SVF cell report (cell number, viability)	Disease activity before treatment	Disease activity after treatment	Follow-up	Adverse event
1	55, Male	4 SVF+aaPRP and 4 aaPRP, continued with monthly aaPRP	1st: 1.38×10^9, 99.45% 2nd: 0.8×10^9, 98.7% 3rd: 0.28×10^9, 99.7% 4th: 0.18×10^9, 87.5%	ASDAS score: 4.1 points CRP: 94.5 mg/dL	ASDAS score: 0.9 points CRP: 2.7 mg/dL	14 months	None
2	58, Male	4 SVF+aaPRP and 4 aaPRP, continued with monthly aaPRP	1st: 1.51×10^9, 99.5% 2nd: 0.50×10^9, 97.56% 3rd: 0.30×10^9, 94.44% 4th: 0.20×10^9, 98.15%	ASDAS score: 3.0 points CRP: 0.18 mg/dL	ASDAS score: 1.1 point CRP: 0.15 mg/dL	7 months	None

SVF: Stromal vascular fraction, aaPRP: Autologous activated platelet-rich plasma, ASDAS: Ankylosing spondylitis disease activity score, CRP: C-reactive protein



FIGURE 1. Patient's presentation showing the classical bamboo spine posture with stooped posture, thoracic kyphosis, cervical flexion, and right hip flexion contracture.

response of his therapeutic regimen despite good compliance. According to the ASAS – European League Against Rheumatism recommendation, therapeutic regimen evaluation should be done after 3 months of therapy; our patient who came to us was still in a very high disease activity even though he had taken the medication for 1 year. This indicated an ineffective therapeutic regimen to control the inflammation. On the other hand, our case 2 patient had visited an internist 2 years before presentation and was only given NSAIDs. As he experienced temporary pain relief upon consumption of NSAID, he did not visit his internist for the 1-month evaluation of NSAID and continued the medication on his own. However, as the pain relief decreased over time, the patient finally came to our clinic. Upon evaluation, he was on a high disease activity with an ASDAS-CRP score of 3.0. This clearly showed that his NSAID-only regimen was inadequate. These two cases had been managed with NSAIDs, with case 1 also receiving sulfasalazine, yet their disease activity remained inadequately controlled (11).

We treated both patients with SVF combined with aaPRP. SVF provides mesenchymal stem cells (MSCs) with regenerative and anti-inflammatory effects, while aaPRP enhances MSC proliferation and augments anti-inflammatory cytokine release (12-16). Case 1 achieved inactive disease after four IV infusions of SVF plus aaPRP, discontinued NSAIDs, and later stopped sulfasalazine after 1 year of monthly aaPRP, remaining symptom-free without medication. Case 2 also reached inactive disease with reduced stiffness and remained flare-free on monthly aaPRP. These cases demonstrate the potential of SVF combined with aaPRP to reduce disease activity in AS and maintain remission without adverse events.

Our two cases highlighted the potential of SVF combination with aaPRP to reduce the disease activity of AS patients with very high or high disease activity to inactive disease without any reported adverse events. These are shown through the reduction of inflammatory pain and stiffness in our patients, which represented the anti-inflammatory and regenerative effects exerted. Moreover, through these cases, aaPRP can be used to maintain the inactive disease state of our patients. However, it is important to note that these two cases remain to be preliminary findings with a very small sample size (n = 2), lack of a control group, and an observational design. We hope that our findings can be a foundation for further studies. Future studies with larger sample sizes and control groups are needed to confirm our findings.

CONCLUSION

Cellular therapy using SVF and aaPRP may have potential to reduce disease activity in AS through modulation of inflammation and support of tissue repair. However, these findings should be interpreted with caution, and further well-designed controlled studies are needed to confirm efficacy, safety, and long-term outcomes.

ACKNOWLEDGMENTS

The authors would like to thank the staffs of HayandraLab and staffs of Hayandra Clinic for their support in making this case report possible.

DECLARATION OF INTERESTS

Authors declare no conflict of interests.

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