Successful Catheter Intervention for Acute Coronary Syndrome in a Patient with Antiphospholipid Syndrome

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SUMMARY

Antiphospholipid syndrome (APS) has the clinical manifestations of systemic vascular thrombotic disorders. Although coronary events are infrequent4,7,8) they have been described. Early coronary interventions and vein graft bypass frequently failed because of thrombosis.5,9

Here we present a case of successful coronary intervention and management of acute coronary syndrome under a strictly controlled coagulation state in an APS patient. (Jpn Heart J 2001; 42: 627-631)

Key words: Antiphospholipid syndrome, Coronary intervention, Thrombotic disorder

ANTIPHOSPHOLIPID syndrome (APS) is identified by the presence of antiphospholipid antibodies and lupus anticoagulant. This syndrome has the clinical manifestations of systemic thrombotic disorders, including recurrent deep vein thromboses, pulmonary thromboembolisms and brain strokes. Although coronary events are infrequent4,7,8) they have been described in patients with APS. Early coronary interventions and vein graft bypasses have frequently failed because of thrombosis.5,9) Here, we present a case of successful coronary intervention for thrombotic lesion of an APS patient, and successful management under strictly controlled coagulation.

CASE REPORT

A 50-year-old Japanese female had a history of Raynaud's phenomenon in both of her fingers, and a butterfly rash over her cheeks and bridge of the nose in 1992. Following a diagnostic workup, her illness was diagnosed as systemic lupus erythematosus (SLE) and she was treated with corticosteroids. In 1993, under a tapering regimen of corticosteroids, she manifested a persistent untreated-
able fever, as well as muscular pain in both extremities. Antiphospholipid antibodies were elevated at that time. She was diagnosed with APS secondary to overlap syndrome, which includes SLE, dermatomyositis and Sjogren syndrome. In 1993, she had depressive insanity that was treated with antidepressants. In 1999, she had multiple cerebral infarctions that were treated with anticoagulant.

More recently, in May 2000, she presented with two weeks of chest oppression. She was immediately admitted with a strong retro-sternal chest pain and while in a cold sweat. On admission, her height was 156 cm and body weight was 56 kg. Blood pressure was 173/103 mmHg and heart rate was 74 beats per minutes. The electrocardiogram showed 7 mm ST elevation in the precordial and II, III, and aVF leads. The echocardiogram demonstrated a deterioration of the anterior wall segment. These findings provided us with a definite diagnosis of acute coronary syndrome. Emergent coronary angiography (CAG) revealed total obstruction with thrombi in the left anterior descending artery and collateral filling through a diagonal branch artery (Figure 1A). Next, we performed coronary intervention. Within an hour, coronary recanalisation was successful, though residual stenosis remained with longitudinal dissection (Figure 1B). Although we considered implanting a coronary stent, we decided against this intervention due to the risk of thrombotic complications from antiphospholipid antibodies.

![Figure 1](image.png)

**Figure 1.** A: Emergent coronary angiography revealed total obstruction with thrombi in the left anterior descending artery (arrow) and collateral filling through a diagonal branch artery. B: After coronary intervention, CAG revealed residual stenosis remained with longitudinal dissection. (arrow head).
The results of the laboratory studies included the following: white blood cell count, $8.6 \times 10^3/\mu l$; red blood cell count, $496 \times 10^3/\mu l$; platelet count, $20.4 \times 10^4 /\mu l$; maximum creatine kinase, 45 IU/l; glutamic oxaloacetic transaminase, 32 IU/l; lactic dehydrogenase, 224 IU/l; C-reactive protein, 1.8 mg/dl; and erythrocyte sedimentation rate, 89 mm/hour. The activated partial thromboplastin time was 36.1 seconds (normal<25 seconds). Positive results were obtained for the LE test, lupus anticoagulant, IgG and IgM antiphospholipid antibodies, as well as anti SS-A and SS-B antibodies. Anti-cardiolipin-$\beta_2$ glycoprotein I antibodies were found to be 108 U/ml (normal <3.5 U/ml), specked antinuclear antibodies were 2560 times, anti-double stranded DNA antibodies were 12.9 IU/ml (normal<12 IU/ml) and CH50 was 24.6 U/ml (normal=30 to 40 U/ml). Negative results were obtained for the anti-Sm, anti-Jo1 and anti-RNP antibodies.

After the procedure she was treated with corticosteroids (predonisolone 20 mg, once a day), cilostazol (200 mg, twice a day), and ticlopidine (200 mg, twice a day). Although warfarin had been administered before this event, the coagulation was poorly controlled (PT-INR; prothrombin time internationalized ratio of 1.3). We attempted to maintain warfarin at an internationalized ratio of 2.5 to 3 under the administration of heparin (10,000 unit/day) for the next 36 days. The follow-up CAG 2 weeks later revealed that focal dissection still remained. The impaired myocardial wall motion recovered according to subsequent echocardiogram.

![Figure 2](image.png)

Figure 2. After 6 months, CAG showed the repaired stenosis and dissection.
grams. Following discharge, neither occlusive signs nor bleeding complications were documented. The regular follow-up CAG after 6 months showed a repaired dissection without significant residual stenosis (Figure 2).

**DISCUSSION**

APS has systemic vascular thrombotic manifestations including recurrent deep vein thromboses, pulmonary thromboembolisms, and brain strokes. These thromboses often occur and recur in multiple organs. Coronary events have been described to occur in approximately 5% of patients with APS, especially patients under age 45. Typically, coronary events tend to occur secondary to other thrombotic events. The underlying mechanisms for cardiac thrombosis are speculated to be as follows. First, antiphospholipid antibodies, especially IgG against cardiolipin, may suppress the activity of the platelet membrane or some other components including thrombomodulin related to prothrombin, protein C and protein S. This change in activity results in the inactivation of fibrinolysis. Second, APS is frequently present with SLE, which is involved in non-inflammatory myocardial microvasculopathy. The result is to trigger multiple occlusive thrombi in the coronary circulation. Third, the antibodies interfere with the formation of the prothrombin activator-a complex of factors Xa, V calcium and platelet factor III- that leads to prolonged activated partial thromboplastin times. Last, the antibodies suppress the endothelial cells which produce the vaso-dilating prostacyclin. These effects change the normally controlled coagulation process into a hyper-coagulable state.

Clinically, coronary intervention and vein graft bypass failures appear to be highly influenced by thrombotic events. However, the regular use of steroids, anticoagulants, and antiplatelets can prevent many thrombotic complications. Anticoagulation therapy, which is commenced immediately after the coronary intervention, may contribute to long-term coronary patency. In this case, anticoagulation therapy was appropriately commenced with corticosteroids, and had been maintained during admission of 30 days until the coagulation process was well controlled.

Recently some reports have described coronary stent implantation for APS patients. However, in this case, we tried not to implant a coronary stent, and could manage the APS patient under strictly controlled coagulation. Although residual stenosis remained with dissection, neither occlusive signs nor bleeding complications were observed.

In conclusion, under strictly controlled coagulation we could successfully perform coronary interventions without stent implantation and manage acute coronary syndrome in a patient with APS.
REFERENCES