Proceedings of
The Second National Congress on Fluids Engineering
August 22-24, 2002, Muju, Korea

Impact of Complex Hemodynamics to the Management of ArterioVenous (AV) Fistula

Byung-Boong Lee

동정맥루의 복합성 혈류학 소견이 그 관리에 미치는 영향

이병붕

Abstract

Human circulatory system between heart and tissue is not directly connected in normal condition but mandatory to go through the capillary system in order to fulfill its physiologic aim to deliver oxygen and nutrients, etc. to the tissue and retrieve used blood together with waste products from the tissue properly. When abnormal connection between arterial and venous system (AV fistula), these two circulatory systems respond differently to the hemodynamic impact of this abnormal connection between high pressure (artery) and low pressure (vein) system. Depending upon the location and/or degree (e.g. size and flow) of fistulous condition, each circulatory system exerts different compensatory hemodynamic response to this newly developed abnormal inter-relationship between two systems in order to minimize its hemodynamic impact to own system of different hemodynamic characteristics. Pump action of the heart can assist the failing arterial system directly to maintain arterial circulation against newly established low peripheral resistance by the AV fistula during the compensation period, while it affects venous system in negative way with increased venous loading. However, the negative impact of increased heart action to the venous system is partly compensated by the lymphatic system which is the third circulatory system to assist venous system independently with different hemodynamics. The lymphatic system with own unique lymphodynamics based on peristaltic circulation from low resistance to high resistance condition, also increases its circulation to assist the compensation of overloaded venous system. Once these compensation mechanisms should fail to fight to newly established hemodynamic condition due to this abnormal AV connection, each system start to show different physiologic decompensation including heart and lymphatic system.

The vicious cycle of decompensation between arterial and vein, two circulatory system affecting each other by mutually negative way steadily progresses to show series of hemodynamic change throughout entire circulation system altogether including heart. Clinical outcome of AV fistula from the compensated status to decompensated status is closely affected by various biological and mechanical factors to make the hemodynmic status more complicated. Proper understanding of these crucial biomechanical factors in particular on hemodyanmic point of view is mandatory for the advanced assessment of biomechanical impact of AV fistula, since this new advanced concept of AV fistula based on biomechanical information will be able to improve clinical control of the complicated AV fistula, either congenital or acquired.

Introduction

Human circulatory system provides necessary oxygen and nutrients to the body tissue via fresh arterial system from the heart and retrieve this used blood with waste products, etc from tissue to the heart mostly by venous system. This transportation function depends mostly to the two major closed conduit systems namely artery and vein besides third conduit system, lymphatics. These three circulatory conduits are delicately connected via capillary system at tissue level to carry the function of unloading of arterial blood to tissue and re-loading of venous blood as well as lymph fluid. Accordingly, three circulatory systems are not directly connected to fulfill these physiologic functions properly and must go through capillary system. However, when this mandatory condition of capillary system should break down for various reason, these three uniquely different hemodynamics of each circulatory system affect to each other negatively. When abnormal connection between arterial and venous system should develop by passing the normal capillary system, this pivotal function of capillary system to maintain delicate "check and balance" between high-pressure (arterial) and low-pressure (venous) system can no longer be available.

Hence, these two circulatory systems will respond differently to this newly developed abnormal connection between arterial and venous system. Depending upon the degree (e.g. size and flow) and location (e.g. aorta and vena cava) of this abnormal connection (e.g. AV fistula), its hemodynamic impact to each system is enormous with the tendency to progress to make the condition worse giving increasing burdening to each other in vicious cycle.

Materials & Methods

59 clinical patients diagnosed as AV shunting malformation (AVM) were thoroughly evaluated for their hemodynamic status with various clinically available diagnostic technology (e.g. Duplex scan, MRI, WBBPS & TLPS and angiography), and 8 patients were confirmed as high-flow AV fistulous type to give serious impacts on arterial system as well as venous system besides heart. In order to arrest cardiovascular impact by this abnormal AV connection (AV

* 성균관대학교의과대학, bblee@smc.samsung.co.kr

fistula), various embolo/sclerotherapy utilizing various (e.g. coil, contour particles, N-butyl cyanoacrylate glue, and absolute ethanol, etc.) were used either as independent therapy or combined therapy with surgical therapy through multidisciplinary team approach.

Results

In spite of aggressive approach to control local and systemic impact of high-flow AV fistula to cardiac, arterial and venous system on 8 patients with AV shunting type of congenital vascular malformation, the results were not all successful. One patient died of heart failure after the abandonment of continuous treatment following series of major set-back to control intraosseous AV fistulae resulting in unsuccessful amputation. Initial amputation of hand and fingers was inevitable to control massive bleeding along the gangrene of dorsum of hand and digits due to acute ischemia superimposed with severe venous hypertension, before the adequate control of massive AV fistulae at upper and forearm soft tissues as well as humeral bone. Multisession transarterial embolotherapy to multiple AV fistulae with coil and glue were performed following initial amputation of hand and forearm to provide adequate control of distal ischemia as well as venous hypertension to prevent amputation stump break down. However, second and third amputation along forearm and upper arm were also required as life-saving procedure mainly to control recurrent episodes of acute massive bleeding from the stump, which occurred in-between aggressive control of multiple AV fistulae but they were also failed mostly due to inaccessible control of AV fistulae within the bone as well as intrathoracic extension. Patient abandoned further treatment and took its nature course. Another patient with massive bleeding from extensive AV fistulae along the elbow region underwent emergency life saving embolotherapy with coil and glue to stop bleeding from the AV fistulae eroded through the skin of forearm. Initial control of AV fistulae with embolotherapy was quite successful to restore near normal arterial blood flow to peripheral tissue with substantial improvement of venous hypertension on hand secondary to AV fistulae as well besides normal cardiac function. However, the patient developed total paralysis of hand and fingers due to ischemic damage to the nerve especially following the tourniquet compression to control bleeding before the referral to SMC to stay in non-functional condition with extensive venous malformation combined. Therefore, following successful control of AV fistulae along the shoulder and upper arm to make above-elbow amputation to be carried on safely, distal amputation was performed successfully with well healed stump and subsequently completed effective control of residual vascular malformation, mostly venous along the shoulder and chest wall with appropriate embolosclerotherapy. The other six patients with AV fistulae type of AVM resulting in various degree of hemodynamic derangement were individually managed successfully. Preoperative embolotherapy and subsequent surgical excision on 4 patients, and independent embolosclerotherapy only using various agents on 2 patients were performed to achieve clinically satisfactory condition with full restorement of normal hemodynamics eradicating complex hemodynamic phenomenon of arterial ischemia and simultaneous venous hypertension, etc.

Discussion

Each circulatory system exerts different compensatory hemodynamic response to this newly developed abnormal interrelationship in order to minimize hemodynamic impact to own system of different hemodynamics. Failing arterial system of high pressure due to sudden drop of peripheral resistance due to abnormal direct connection to venous system of low-pressure bypassing normal tissue perfusion can be initially compensated by increased cardiac pump action so that it is able to maintain high pressure condition as before against newly established low peripheral resistance during the compensatory period and successfully abort ischemic condition on peripheral tissue. However, this compensatory action in arterial system by increased heart pump action will result in negative impact directly to the venous system via same abnormal AV connection (AV fistula) with increased loading to the venous system as well as lymphatic system indirectly. The lymphatic system as the third circulation system to assist venous system has own unique lymphodynamics based on autoregulated peristaltic circulation from low resistance to high resistance condition in contrast to venodynamics, so that the compensatory capacity of normal lymphatic system with this self regulatory condition in high pressure (50-60 mmHg) has own limit, and can not provide substantial compensation to this mostly venous overloading. However, the negative impact of the increase heart pump action to the venous system is partly compensated by this lymphatic system to assist venous system independently with different hemodynamics, and the venous system will have to encounter this new condition of increased pressure and volume by arterialized condition via AV fistula. During the compensation period of venous system, the normal function of venous valve system to block the retrograde blood flow (reflux) remains intact so that venous hypertension toward proximal vein (toward tissue) can be prevented but instead distal venous system (toward heart) only shows significant hypertension due to overloading to this venous system via AV fistula. This overloading to systemic venous system is naturally extended to the pulmonary circulation system first and then to the right side of heart to start to induce cardiac overloading to get into self-propelling vicious cycle.

Once these compensation mechanisms through arterial, venous, and lymphatic system as well as heart pump action should fail to fight effectively to this newly established hemodynamic condition via AV fistula, each circulatory system including heart will start to show different physiologic decompensation. Arterial system can no longer maintain adequate arterial circulation distal to AV fistula to the peripheral tissue resulting in ischemic condition, while venous system proximal to AV fistula (toward tissue) also no longer able to maintain normal valvular function to prevent retrograde flow and allow steady reflux to peripheral tissue with further blocking of normal antegrade venous flow from the tissue, resulting in chronic venous insufficiency with severe venous hypertension in tissue level as direct outcome of decompensation. Venous hypertension at this tissue level can further deteriorate into the venous gangrene in extreme occasion due to hampered arterial inflow via normal capillary system by this severe venous hypertension. The vicious cycle of decompensation between arterial and venous two circulatory system affecting to each other in negative way via AV fistula steadily progresses to show series of hemodynamic change throughout entire circulation system altogether including heart failure as the final outcome. Clinical outcome of abnormal AV connection (AV fistula) from the compensated status to the decompensated status is closely affected by various biological and mechanical factors to make the hemodynamic status more complicated.

Conclusion

Proper understanding of these crucial biomechanical factors in particular, on hemodynamic point of view is mandatory for the advanced assessment of biomechanical impact of AV fistula since this new advanced concept of AV fistula based on biomechanical information will be able to improve clinical control of the complicated AV fistula either congenital or acquired.