Is the “V” Sign and The “Butterfly Wings” Finding on MRI Specific For The Percheron Artery Infract? Four Cases Presented With MRI and Clinical Findings

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Abstract

Paramedian thalamic arteries are separated from the P1 segment of each posterior cerebral artery. Multiple variations are known to be present. One of these rare variations is called the “Percheron artery” and arises from the P1 segment of the posterior cerebral artery as a single root. This artery supplies blood to bilateral paramedian thalamus and rostral mid-brain. The occlusion of the Percheron artery (PA) occurs rarely, resulting in a large number of various neurologic findings and symptoms. This leads the physician to consider etiologic factors other than vascular causes. The patients are observed to have sudden and dramatic clinical signs and symptoms. Thus, the manifestations need to be immediately recognized to start the appropriate treatment for the patient. During the acute period, magnetic resonance with diffusion weighted imaging is essential for diagnosis. In this report, we present 4 patients with bilateral rostral mid-brain and paramedian thalamic infarct secondary to PA occlusion, in whom we detected “butterfly wings” pattern in the mesencephalon together with a “V sign” along thepial surface of the mesencephalon by diffusion weighted imaging during the acute period. As far as we know, these imaging findings, which we believe are specific for this artery, have not been reported in the Turkish literature before. We believe these findings are significant for the accurate diagnosis, timely sensitive therapy of the PA occlusion-associated infarcts and in avoiding unnecessary procedures during the acute period.

Keywords: Percheron artery, thalamus, midbrain, infraction, MRI

Özet

sunmak istedik. Bilgilerimize göre bu artere özgü olduğunu düşündüğümüz bu görüntüleme bulgularından Türkçe literatürde daha önce bildirilmemiştir. Bu bulguların akut dönemde PA okluzyonuna bağlı infarktlarının doğru tanısı, zamana uygundur sensitive tedavi ve gerekli prosedürlerden kaçmak için önemli olduğunu düşünmektediz.

**Anahtar Kelimeler:** Percheron arter, talamus, orta beyin, infarkt, MRG

**INTRODUCTION**

The blood flow of the thalamus is quite complex and involves a large number of vascular variants. The Percheron artery (PA) is one of these variants and arises from the proximal segment of one of the two posterior cerebral segments as a single root\(^2\). It provides blood supply to the bilateral paramedian thalamus, the midbrain and rarely to the anterior thalamus\(^{1,2,15}\). It is present at a rate of 4%-12% in the society and occlusion-associated infarctions are rare\(^2\). It is reported to develop in 0.2-0.3% of all the ischemic infarctions and in 4-35% of all thalamic infarctions\(^{2,9,13}\). The most common etiologic causes include small vessel disease and cardiac embolism\(^{2,15}\).

Its occlusion is a characteristic ischemic pattern that commonly results in paramedian thalamus involvement with or without bilateral relatively symmetrical midbrain involvement\(^{9,11}\). Typical clinical findings are changes in the mental status, vertical gaze palsy and memory impairment\(^{2,3,4,9,13,15}\). However together with the complex anatomy and functions of the thalamus and the extension of the PA to the brain stem expands this spectrum of clinical findings\(^1\). Thus, the clinical findings can be commonly mistaken for a wide spectrum of diseases such as metabolic and toxic encephalopathies, infectious-inflammatory diseases or infiltrative malignancies\(^{1,3,9,11}\). This may lead the physician to consider an etiology other than stroke. During the acute period, a normal result obtained from brain computed tomography (CT) is the other factor complicating the diagnosis. The common use of magnetic resonance imaging (MRI) may make recognition of this specific clinical manifestation easier in the early period. It is recommended that the essential diagnosis be considered as PA occlusion if bilateral paramedian thalamic infarction is detected\(^{11}\).

Accurate diagnosis of PA infarctions is important for administering timely appropriate sensitive treatment and avoiding unnecessary procedures. New studies provided data on the specific pattern and the overall clinical spectrum of the lesion on MRI, however they did not reveal a certain correlation between the clinical findings and MRI results\(^2\).

PA infarctions may be recognized via conventional imaging techniques. However we wanted to present 4 patients with bilateral paramedian thalamic and rostral midbrain infarction, in whom we detected a “V sign” and “butterfly wings” finding on the diffusion-weighted images (DWI) and fluid low attenuation inversion recovery (FLAIR) weighted axial sections, a finding that had not been reported in the Turkish literature to our knowledge.

**METHODS**

**CASE 1:** A 72-year-old male patient was brought to the emergency department with the complaints of acute loss of consciousness and collapse. The patient was found to be normal in physical examination (PE) and neurologic examination (NE) revealed no pathology other than mild confusion. The CT requested by the emergency department was considered normal; and there was no abnormality on ECG and PA chest radiograph. It was learnt that he had developed diplopia in the morning of the same day and presented to the neurology clinic, and MRI and magnetic resonance
angiography (MRA) had been requested after the examination. He was admitted to the neurology clinic under the preliminary diagnosis of transient global ischemia and transient brain stem ischemic stroke. Antiaggregant was initiated. At this time, an unclear “V” shaped hyperintense image was detected along the pial surface of the mesencephalon in the interpeduncular fossa (Image 1A). This was considered to be a possible brain stem infarction. His medical history revealed that he had received treatment for hypertension (HT) for a period but had discontinued treatment due to good follow-up results. In addition, he had been smoking one pack daily for nearly 30 years.

The patient was reported to experience somnolence 4 hours after his admission and could not be woken up. The neurologic examination revealed that he had stupor status, no motor deficit and had positive bilateral Babinski’s sign. On DWI, we observed that the V-shaped image detected on the first MRI in the midbrain had become distinct and there was also an asymmetrical hyperintense image in the bilateral paramedian thalamus (Image 1B,C). The patient was also observed to have hypointense lesions in the same location on apparent diffusion coefficient (ADC) images (Image 1D). Particularly, the involvement of the bilateral paramedian thalamus suggested the presence of PA occlusion-associated infarction and thus the patient was admitted to the neurology intensive care unit. The laboratory investigations were normal. The color doppler ultrasonography (CDUS) of the bilateral carotid-vertebral arteries revealed no pathology other than mild intimal thickening of the vessel walls. Cardiology examination revealed no pathology. Transthoracic echocardiography (TTE) revealed normal findings. His state of consciousness was impaired on the next day, he developed coma and irregular respiration. He was assessed by the chest diseases department and no primary lung pathology was considered. The patient who developed fever and deteriorated overall health status died two days after admission.

**CASE II:** A 79-year-old woman was brought to the emergency department since she had been lying still and mutely, not answering any questions, eating or drinking for the last one day. From her amaness, we learnt that she had suddenly experienced dizziness, blacked out and aggravated, then got better and remained so for a while; and subsequently, she developed the above mentioned clinical manifestation. Her medical story included regular use of medication for HT and untreated cardiac valvular disease.

Her PE seemed normal except high blood pressure (180/100 mmHg). On NE, she was observed to have eyes closed, opened her eyes upon audio stimuli, she had no spontaneous movements but was able to move her all 4 extremities with external stimuli. When she opened her eyes, she seemed to pay attention to those around her however she stared blankly and closed her eyes after a short while. The eyes were mildly deviated downwards in the primary position. Her clinical manifestation was considered to be akinetic mutism. Her ECG, PA chest radiograph and laboratory investigations were normal. The CT requested at the emergency department revealed no pathology. The simultaneously performed DWAs revealed a “V” shaped hyperintense image along the pial surface of the bilateral rostral mesencephalon. This hyperintense image detected on the bilateral mesencephalon resembled “butterfly wings” as a whole (Image 2A). Together with these findings, symmetric diffusion MRI revealed hyperintense lesions in the bilateral paramedian thalamus and hypointense lesions on ADC and she was diagnosed with infarction (Image 2B,C). The patient was admitted to the intensive care unit with the diagnosis of PA occlusion.

Antiaggregant was initiated. The patient was assessed for cardiac etiology and she was found to have a normal TTE and no
cardiac pathology was considered. Her clinical course was stable and there was no change for two days. However, starting from the third day, she had deterioration in her consciousness and increased somnolence. There were no other additional neurologic pathological findings. On the fourth day, she developed state of coma, fever and impaired respiration. She was evaluated for fever and respiratory failure. No causal pathology could be detected and she died on the same day.

**CASE III:** An 84-year-old male was referred from another center because of no vacancy. Based on the information obtained from her relatives, he had experienced dizziness, blackingout, blurred vision followed by impaired consciousness and brought to the hospital. His medical story included regular treatment for cardiac valvular disease and HT. NE revealed that he was inclined to sleep, had limited cooperation and orientation and no motor deficit. Results from the physical examination revealed no abnormal findings except cardiac arrhythmia. ECG revealed atrial fibrillation (AF), and the PA chest radiograph was normal. Laboratory results showed no pathology. The CT performed at the other center revealed normal findings except high blood pressure (160/100 mmHg). NE revealed that she was mildly confused, her eyes were deviated downwards in the primary position and she had bilaterally limited upward gaze, right hemiparesis (3-/5), and positive Babinski’s sign on the right. CT performed during the acute period (3 hours after the event) showed no abnormality. DWI showed an unclear “V” shaped hyperintense image along the pial surface in bilateral mesencephalon. The image resembled “butterfly wings” as a whole (Image4A). Additionally, mildly hyperintense images were detected in the bilateral paramedian thalamus (Image4B). Partially symmetrical image detected on the simultaneous FLAIR weighted axial sections in the bilateral rostral mesencephalon, which more resembled butterfly wings and the hyperintense images detected in the bilateral symmetrical paramedian thalamus were considered to be infarction (Image 4C,D) and secondary to PA occlusion. She was admitted to the intensive care unit and antiaggregant was initiated. Cardiac investigation revealed aortic and mitral valvular stenosis, cardiac failure and AF, and anticoagulant medication was recommended with appropriate treatment. After informing his relatives about the anticoagulant treatment, he was put on treatment. He developed fever and impaired consciousness two days later. The etiology of fever was assessed by the infectious diseases department and no pathology was detected. Symptomatic and empirical antibiotherapy was initiated. His fever was refractory to treatment; he lost consciousness and his respiration got impaired. He died one day later.

**CASE IV:** An 81-year-old female was brought to the emergency department due to acute weakness on her right side. She had no disorders other than diabetes and was on regular oral antidiabetic treatment. The physical examination revealed normal findings except high blood pressure (160/100 mmHg). The CT performed during the acute period (3 hours after the event) showed no abnormality. DWI showed a hypointense image along the pial surface in bilateral mesencephalon. The image resembled “butterfly wings” as a whole (Image4A). Additionally, mildly hyperintense images were detected in the bilateral paramedian thalamus (Image4B). Partially symmetrical image detected on the simultaneous FLAIR weighted axial sections in the bilateral rostral mesencephalon, which more resembled butterfly wings and the hyperintense images detected in the bilateral symmetrical paramedian thalamus were considered to be infarction (Image 4C,D) and secondary to PA occlusion. She was admitted to the intensive care unit and antiaggregant was initiated. Her laboratory investigations were normal except increased fasting blood glucose level. No abnormality was observed at the ECG and PA chest radiograph. TTE was normal. The CDUS of the bilateral carotid-vertebral artery revealed 30% stenosis in the right carotid artery. A day later, the
limitation in the upward gaze improved nearly completely and there was a regression in the motor deficit (4/5). The etiologic factor was considered to be diabetes-associated small vessel disease. The patient died on the 5th day of admission due to myocardial infarction.

**Image 1A:** “V” shaped mildly hyperintense image detected along the pial surface of the mesencephalon in the interpeduncular fossa on DWI sections. B: V” shaped hyperintense image detected along the pial surface of the mesencephalon in the interpeduncular fossa on DWI sections. C: Hyperintense lesion detected in the bilateral asymmetrical paramedian thalamic region on DWI sections. D: hypointense lesion detected in the bilateral asymmetric paramedian thalamic region on ADC.

**Image 2A:** V” shaped hyperintense image detected along the pial surface of the mesencephalon on DWI sections of case II. On the same sections, the image in the bilateral rostral mesencephalon is observed to resemble “butterfly wings”. B: relatively symmetrical bilateral paramedian thalamic hyperintense images on DWI sections. C: relatively symmetrical bilateral paramedian thalamic hypointense images on ADC sections.
Image 3: V” shaped hyperintense image detected along the pial surface of the mesencephalon on DWI sections of case III. B: relatively symmetrical bilateral paramedian thalamic hyperintense images on DWI sections. C: “V” shaped hypointense image detected along the pial surface of the mesencephalon on ADC sections. D: symmetrical bilateral paramedian thalamic hypointense images on ADC sections.

Image 4 A: V” shaped hyperintense image detected along the pial surface of the mesencephalon on DWI sections of case IV. On the same sections, the totality of the image in the bilateral rostral mesencephalon is observed to resemble “butterfly wings”. B: relatively symmetrical bilateral paramedian thalamic mildly hyperintense images on DWI sections. C: “V” shaped hyperintense image detected along the pial surface of mesencephalon on the simultaneous FLAIR weighted axial sections. On the same sections, the totality of the image in the bilateral rostral mesencephalon is observed to resemble “butterfly wings. D: Bilateral relatively symmetrical paramedian thalamic hyperintense image on the FLAIR-weighted axial sections.

Figure 1: Four variants of the arterial supply to thalamus and mesencephalon are seen. Type IIb indicates the Percheron artery (T, thalamus; M, midbrain; PCA: posterior cerebral artery; BA, basilar artery; PeA, Percheron artery) (from reference no 1).
DISCUSSION
The thalamus contains strategic nuclei and integrates many significant cortical functions. Thus, mesodiencephalic infarctions may lead to complex syndromes with patients experiencing a wide range of symptoms from motor deficit to behavioral and sensorial changes. The location, size and clinical course of the ischemic injury are determined by the arterial supply to the mesencephalo-diencephalic junction. The blood supply to the thalamus and mesencephalon is provided both by the anterior and the posterior circulation. The anterior circulation usually supplies blood to the thalamus and the anterior and posterior parts of the mesencephalon via thalamo-perforating arteries arising from the posterior communicating artery while posterior circulation supplies blood to the medial parts of the thalamus and the midbrain via branches arising from the P1 segment of the posterior cerebral artery (PCA) with lateral and superior parts being supplied by the branches arising from the P2 segment\(^9\). In other words, the blood supply to the thalamus is provided by the polar(thalamo-tuberal), paramedian (thalamo-perforating) and posterior choroidal arteries, respectively\(^{3,7,9}\). The distribution and branching of the thalamic arteries and the sites they provide blood supply vary among different individuals\(^3\). The vascular anatomy of the posterior circulation was studied by Percheron, a French neurologist, in the 70s\(^{1,3}\). Percheron described four variations of the paramedian perforating arteries that supply the thalamus (Figure 1). One of these variations is that paramedian arteries arise from the P1 segment of the PCA as a single root, which is called PA\(^{4,9}\). The occlusion of this rare anatomic variation also occurs rarely. The occlusion of PA leads to infarctions that simultaneously and relatively symmetrically involve the bilateral paramedian and rostral midbrain\(^{9,11}\). Lazzora et al. found four different patterns for infarctions secondary to PA occlusion; bilateral paramedian thalamic infarction with bilateral rostral midbrain infarction (43%), bilateral paramedian-thalamic infarction without midbrain infarction (38%), midbrain, anterior thalamus and bilateral paramedian thalamic infarction (14%) and anterior thalamus and bilateral thalamic infarction without midbrain infarction (5%)\(^9\). The most commonly seen pattern is bilateral paramedian thalamic infarction with bilateral rostral midbrain infarction. In a more recent study conducted by Arauz, the most common pattern was bilateral paramedian infarction with bilateral rostral midbrain infarction (53%)\(^2\). At our clinic, we detected five cases of bilateral paramedian thalamic infarction during the last four years during which MRI was used routinely in the acute period. Four of these patients also had rostral midbrain involvement. In one case, bilateral paramedian thalamic infarction was found. Since she had only isolated bilateral ptosis, her specific clinical manifestation was reported in a separate paper.

Although PA infarctions are rare, their clinical course is well-established\(^9\). The most commonly reported clinical features include altered consciousness ranging from mild somnolence to coma, vertical gaze paralysis and memory disturbances\(^{7,15}\). However since the supply region of the artery may range from the anterior thalamus to the midbrain, it may also manifest with other various clinical signs\(^{1,2}\). Some of these signs include hemiparesis, hemisensory loss, anterograde and retrograde amnesia, perseveration, confabulation, third cranial nerve disorders, movement disorders, and epileptic seizures\(^{3,7,9,14,16}\). Impaired consciousness, mood changes and memory impairment are associated with bilateral
involvement of the thalamus\textsuperscript{(15)}. Here, the involvement of the reticular activator system, and also the impaired connections between the anterior, orbitofrontal and medial prefrontal cortex may explain the changes in mental status\textsuperscript{(13)}. In all of our patients, the consciousness had been affected during the acute period. Short-term loss of consciousness was described before the event in Case I and II. While Case I re-experienced loss of consciousness 4 hours after the event, the Case II developed akinetic mutism. In two patients, a clinical course resembling transient global ischemia was observed. This is considered to be associated with the transient ischemia of thalamus. There are rare case reports supporting this in the literature\textsuperscript{(3,7)}. The common clinical symptom in all four cases is the impairment of consciousness to varying extents and this reflects the bilateral thalamic involvement in PA infarctions\textsuperscript{(2)}. The clinical course of Case II was considered to be akinetic mutism. This is thought to result from the injury to the median and interlaminar nucleus and the impaired connection of these structures with prefrontal, orbital and anterior cingulate cortex\textsuperscript{(3)}. There are a few reports describing cases of PA occlusion manifesting with akinetic mutism in literature\textsuperscript{(6,11)}. The common finding suggesting involvement of the midbrain or the presence of a thalamo-subthalamic lesion is upward gaze paralysis\textsuperscript{(3,9)}. However, upward gaze paralysis may also occur in cases where the midbrain is not affected. This finding results from the impairment of the cortical inputs transversely passing the thalamus towards the rostral interstitial medial nucleus\textsuperscript{(9,13,15)}. We observed vertical gaze paralysis only in two cases (II and IV). The complaint of diplopia occurring before the onset of the event in Case I suggests an effect on the brain stem; however, no supportive findings were obtained at the examination. In Case III, we failed to obtain the detailed data on the story of blurred vision due to the patient’s loss of consciousness. These complaints suggested transient involvement of the third cranial nerve nucleus. There are case reports in the literature, which state that certain patients may have only oculomotor nerve paralysis or hemiparesis\textsuperscript{(7,8,13)}. Motor deficit was observed only in case IV and partially improved. This finding is associated with the involvement of the corticospinal pathways at the level of midbrain. However, motor deficit is a finding that is reported in patients with PA infarction with or without rostral brain involvement\textsuperscript{(2)}. As mentioned above, it is not always possible to observe all three findings in the conventional clinical triad in PA infarction cases. In our cases, the common finding secondary to PA infarction was altered consciousness. The second most common finding can be described as vertical gaze paralysis and impaired vision suggesting brain stem ischemia. The most significant finding indicating midbrain involvement is the vertical gaze paralysis\textsuperscript{(3,15)}. However, this symptom is not observed in all cases and may occur also without midbrain involvement\textsuperscript{(9)}. Mostly, patients present with an acute impairment of consciousness and this extends the spectrum of the differential diagnosis. Thus, since it is difficult to establish a definitive diagnosis through the clinical course, imaging methods such as CT, MRI, and angiography need to be utilized. Since CT is usually completely normal during the acute period, the diagnosis can easily be overlooked\textsuperscript{(1)}\textsuperscript{1}. The most important supplementary diagnostic method is MRI. What suggests the presence of a PA infarction during this period is the hyperintense lesions in the bilateral symmetric or asymmetric paramedian thalamus with or without bilateral rostral midbrain involvement detected on DWI, followed by T2 and FLAIR-weighted sections\textsuperscript{(9)}. PA provides blood supply to the periaqueductal gray substance, i.e. mesencephalon-diencephalic region in a
bilateral manner (13) (Image 2). As can be seen in Image-2, the artery supplies this region both towards the thalamus and along the pial surface of the mesencephalon, resembling a “V” shape. Thus, it is possible that occlusion-associated infarctions may have a similar appearance. Lazzuro et al. named this appearance detected on diffusion and FLAIR images as the “V” sign and reported a sensitivity rate of 64% for this type of infarctions (9). In all of our cases, we detected a marked “V” sign along the pial surface of the midbrain on DWI during the acute period (Image 1A, B, Image 2A, Image 3A, and Image 4A). Particularly in Case I, during the acute period when the event manifested with clinical findings such as transient global ischemia or transient ischemic attack, the same image was detected on axial section with diffusion MRI (Image 1A). This may indicate that the “V” sign may be specific for PA occlusion. All of the hyperintense lesions observed in the bilateral mesencephalon rostral tegmentum in Case II and IV resembled “butterfly wings” (Image 2A and 4A). The concomitant MRI performed in Case IV also showed the same image on the FLAIR-weighted sections (Image 4C). Hochman et al. claimed for the first time in 1985 that the image in the mesencephalon detected on CT of a patient with infarction secondary to PA occlusion resembled a “butterfly” (6). Subsequently, Sparacia et al. named the MRI images of a case they presented for PA occlusion-associated infarction as “butterfly wings” (12). We detected this finding clearly in only two cases. In the other two cases, we failed to observe this finding and thought that this may result from the fact that all sections obtained were not at the same level. Thus, both the “V” sign and the “butterfly wings” finding may be specific for bilateral thalamic infarction with involvement of rostral mesencephalon secondary to PA occlusion. However, the small number of cases limits this conclusion. In addition, the “top of the basilar artery syndrome” may be considered in the differential diagnosis of bilateral thalamic and midbrain infarctions. However, in this clinical course, there is a tendency for involvement of the superior cerebellar artery (SCA) and PCAsupply regions (9,11). The authors report that PA occlusion should be considered as the primary diagnosis in case of bilateral paramedian thalamic infarction (2,11). None of our cases had an image consistent with infarction of the SCA and PCA supply regions. This proves the accuracy of the diagnosis. Conventional angiography is not recommended in diagnosis. These arteries are too small to be imaged by angiography and could be rarely imaged using conventional angiography (1,3). In addition, failure of detection doesn't rule out their presence because they are occluded (3,11). Therefore, conventional angiography, an invasive method, is not routinely used for suspected cases of PA occlusion (1) and it is considered that it may not be necessary (3).

The most common etiologic causes of PA occlusion include small vessel disease and cardiac embolism (2,15). We considered that small vessel disease was involved in the etiology in three of the cases (I, II and IV). While only HT anamnesis was obtained in Case I and II, no pathology was detected with cardiac assessment. Case IV had no findings supportive of cardiac etiology except diabetes and the high arterial blood pressure measured at the time of initial event. Since case III had AF, the primary cause was considered to be cardiac embolism. In addition, patient's HT that could be associated with small vessel disease should also be considered. The most common cause we detected among our cases was small vessel disease and this was consistent with the literature data.

Gentilini reported that the prognosis was “neither good nor severe” in case of paramedian infarctions (5). However cases of patients with complete or near complete cure have also been reported (7,10). All of the
four patients in the cases we reported died. While one died due to MI (Case IV), the other three patients were not detected to have pathology other than infarction. This was inconsistent with the literature. However, Arzu et al. reported that the group of patients with midbrain involvement had a tendency for poor outcome and a significant difference from the group without involvement\(^2\). Having published the largest series in the literature, Lazzoro et al. reported that only 26% of all cases were discharged. In this series, considering that the most common type was midbrain involvement, we could suggest that the infarctions of this site have a poor prognosis\(^6\). We can partially explain the poor prognosis since all our patients had rostral midbrain involvement. However we considered that another and potentially important factor was the advanced age (ages of the patients: 72, 79, 84, 81).

Kostanian and Cramer used thrombolytic treatment in a patient with PA occlusion as confirmed by angiography and reported a good outcome\(^8\). Thrombolytic treatment is recommended by authors in case of PA occlusion\(^3,4,13\). Thus, it is important that the diagnosis of infarctions secondary to PA occlusion is clearly established within the time interval recommended for thrombolytic treatment.

In conclusion, this entity may be easily overlooked via CT during the acute period. This should be considered in case of patients with acute and unexplained loss of consciousness only. The diagnosis should be clarified using diffusion MRI without delay. Since angiography is an invasive method and there are difficulties in routine practice, this method is not recommended to be used in the diagnosis of PA occlusion. We believe that, detection of the MRI findings described above during the acute period can confirm the diagnosis and enable immediate treatment of this clinical picture which has a relatively poor prognosis. While PA variation and occlusion-associated infarcts are rare, our small sample size is a major limitation for a clear conclusion.

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