Original Article

Appendiceal Abnormalities Associated with Congenital Pouch Colon

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Abstract

Purpose: This retrospective cohort study aims to identify the incidence and type of appendiceal abnormalities in the various subtypes of congenital pouch colon (CPC) associated with anorectal malformation. The possible embryopathogenesis of appendiceal abnormalities in CPC is also discussed. **Methods:** The case records of 74 patients of CPC, managed from 1998 to 2022, were studied, and the subtype of CPC, details regarding the vermiform appendix (presence/absence, number, morphology and location) and other relevant findings were noted. **Results:** The CPC subtypes were Type I (n = 12), Type II (n = 41), Type III (n = 11) and Type IV (n = 10). Overall, 26 patients had a single, normal appendix, 12 patients a short, stubby appendix and 17 appendiceal duplication (Type A2 [n = 9], Type B1 [n = 3] and Type C [n = 5]). Type I CPC cases had appendicular agenesis. A short stubby appendix (26.8%) or Type A2/B1/C appendicular duplication (31.3%) was frequent in Type II CPC and Type C duplication in Type III CPC. Type IV CPC patients had a single, normal appendix. In 10 (24.4%) patients with Type II CPC, the caecum and normal proximal colon appeared dilated. **Conclusions:** Appendiceal abnormalities are frequent in Type I–III CPC and may result from the abnormal development and dilatation, along with shortening, of a varying length of colon. As in other reports of appendiceal duplication associated with major intestinal, genitourinary or vertebral malformations, appendiceal duplication in patients with CPC was of the Type A2/B1/C subtypes.

Keywords: Agenesis appendix, appendix, congenital pouch colon, duplex appendix, Meckel's diverticulum

Introduction

Congenital pouch colon (CPC) is characterised by a pouch-like dilatation of a shortened colon associated with an anorectal malformation (ARM).[1,2] CPC is categorised into four subtypes based on the length of normal colon proximal to the colonic pouch.[1] In the more severe subtypes, either the normal colon is absent with the ileum opening directly into the colonic pouch (Type I) or the ileum opens into a short segment of caecum which then opens into the pouch (Type II). The less severe subtypes have a longer length of normal colon proximal to the colonic pouch (Type III and IV CPC).[1] In males with CPC, the colonic pouch usually ends in a colovesical fistula (CVF)[1-4] opening just proximal to the bladder neck.[5] The terminal fistula in girls usually opens either in the urethra or in the vestibule in a 'high' (more cranial) intervaginal position, just posterior to the urethral meatus,[6] correlating with the 'high' fistula in males. CPC is classified as a 'rare variant' in the Krickenbeck classification of ARM[7] and is much more common in India than in the rest of the world, with a review showing that about 90% of reported cases were from India.[8]

Although variations in the size and position of the vermiform appendix (VA) are common,[9] other major appendiceal abnormalities are uncommon. Collins[10] reported an incidence of 0.0009% for appendiceal agenesis (1 in 104,066 appendectomies). Similarly, appendiceal duplications are uncommon, with a reported incidence of 0.0004%[11] to 0.0009%.[12] However, appendiceal abnormalities have frequently been reported in association with CPC, especially with the more severe I and II subtypes.[1,3-5,8,13]

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The most widely accepted 'Cave–Wallbridge' classification of appendiceal duplication, proposed by Cave in 1936[14] and later updated and modified by Wallbridge,[15] classified appendiceal duplications based on their anatomic location. Subsequently, as other anomalies of the VA were reported, further modifications were proposed to include the anomaly of horseshoe appendix.[16,17] Ngulube *et al.*,[18] later, described a comprehensive classification of appendicular anomalies [Table 1].

The purpose of the present study was to estimate the incidence of appendiceal abnormalities in patients of CPC and to correlate the type and form of any appendiceal abnormality with the subtype of CPC and other relevant anatomic findings. The possible embryopathogenesis of appendiceal abnormalities in the various subtypes of CPC is also discussed.

Materials and Methods

In this retrospective cohort study, the case files of 74 patients of CPC associated with anorectal agenesis, managed at our centre over the 25-year period from 1998 to 2022, were scrutinised. All institutional ethical practices and standards were followed, and patient confidentiality was maintained. The case records of the 74 patients provided a clear description with pictorial representation (clear drawing and/or photograph) of the gross anatomy of the colonic pouch and the findings regarding the VA. The study group included some patients who had been part of earlier reports and studies of CPC published from our centre.[5,13,19-21]

The subtype of CPC,[1] details regarding the VA (presence/absence, number, morphology and location) and other relevant operative findings were noted. Duplication of the VA was classified according to Ngulube *et al.*'s[18] classification [Figure 1]. Any associated malformations identified were recorded. The data were analysed to identify the various anomalies of the VA encountered in the study group and to correlate the subtype of CPC with the findings regarding the VA.

Results

The 74 patients included 36 males and 38 females (male:female = 1: 1.06). Type II CPC was the most common ($n = 41$, 55.4%), followed by Type I ($n = 12$, 16.2%), Type III ($n = 11$, 14.9%) and Type IV CPC ($n = 10$ [all males], 13.5%). All male patients had the colonic pouch terminating in a CVF.

The findings regarding the VA in the patients along with details of any morphologic/numerary abnormalities of the VA are shown in Table 2 and Figures 2-9.

All cases of Type I CPC had appendicular agenesis, whereas only 7/41 (17.1%) patients with Type II CPC [Figure 2] and no patient with Types III/IV had agenesis of the VA. A short stubby appendix (11/41, 26.8%) [Figure 3] or Type A2/B1/C appendicular duplication (13/41, 31.3%) [Figures 4-8] were

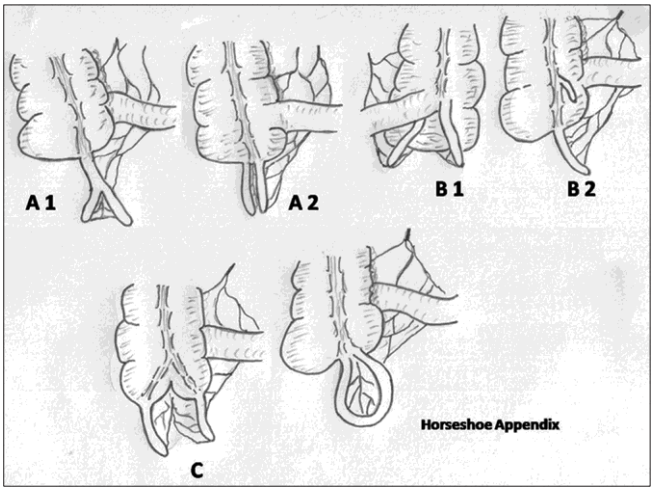


Figure 1: Diagrammatic representation of the types of duplex appendix (Table 1)[18]

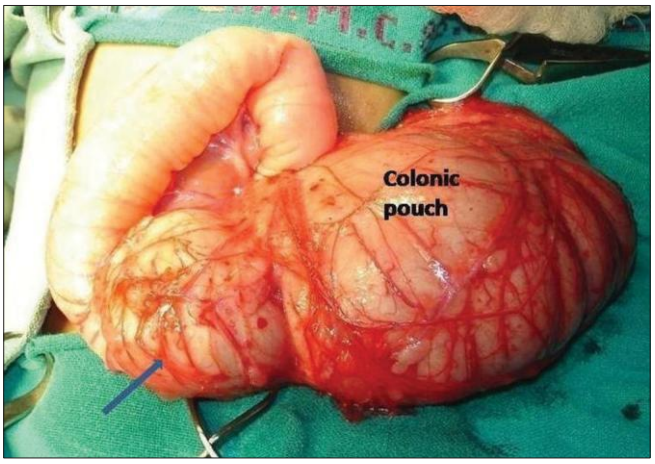


Figure 2: Type II congenital pouch colon in a 4-month-old girl with the absence of the appendix and partial sacral agenesis. Arrow points to the dilated caecum

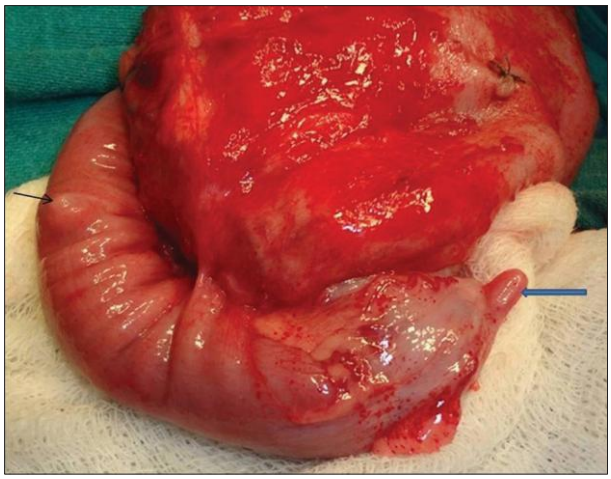


Figure 3: Type II congenital pouch colon in a 7-month-old girl with a short, stubby appendix (wide arrow) and a short Meckel's diverticulum 9 cm proximal to the caecum (fine arrow)

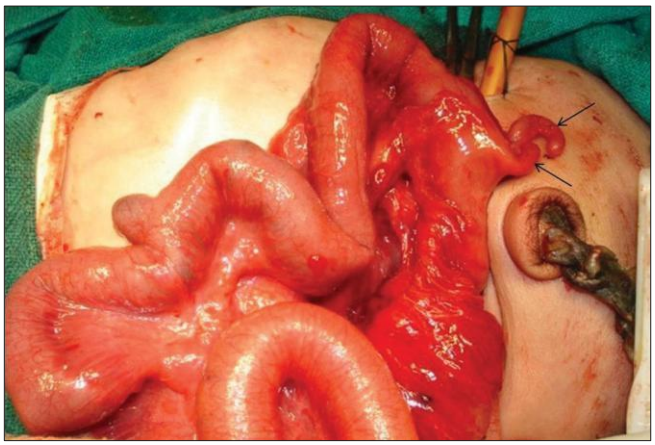


Figure 4: Newborn female with Type II congenital pouch colon, Meckel's diverticulum and Type A2 duplex appendix (arrows)



Figure 5: Newborn male with Type II congenital pouch colon, wide-based Meckel's diverticulum 3–4 cm proximal to ileocaecal valve (arrow), Type A2 duplex appendix (fine arrows) and unilateral intra-abdominal testis

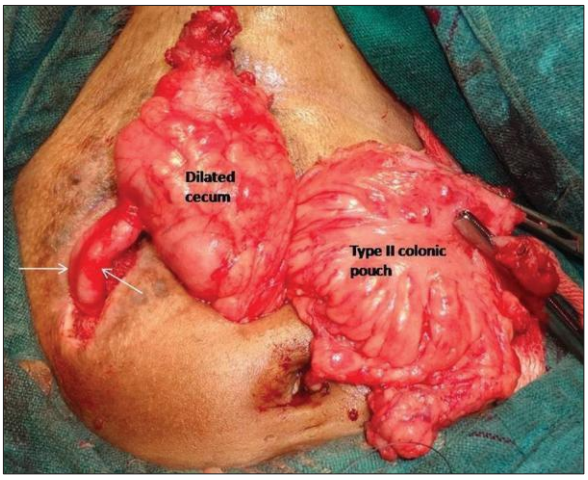


Figure 6: Type II congenital pouch colon in a 2.5-year-old girl with dilated caecum and Type A2 duplex appendix (arrows)

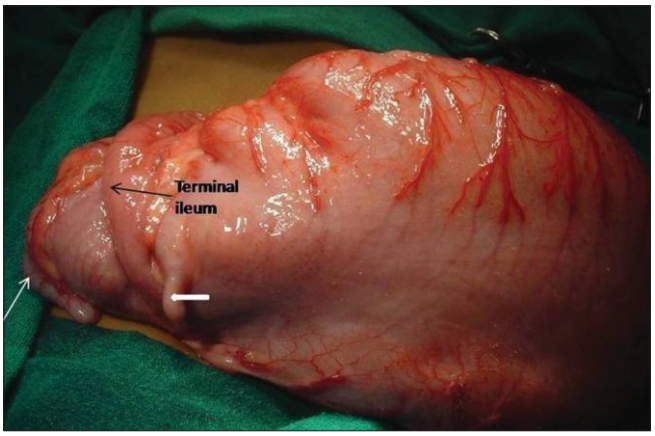


Figure 7: Type II congenital pouch colon in a 1.5-year-old girl with Type B1 appendiceal duplication. Note the dilated caecum and the short appendix on one side (wide arrow) and normal-sized appendix on the other side (fine arrow)

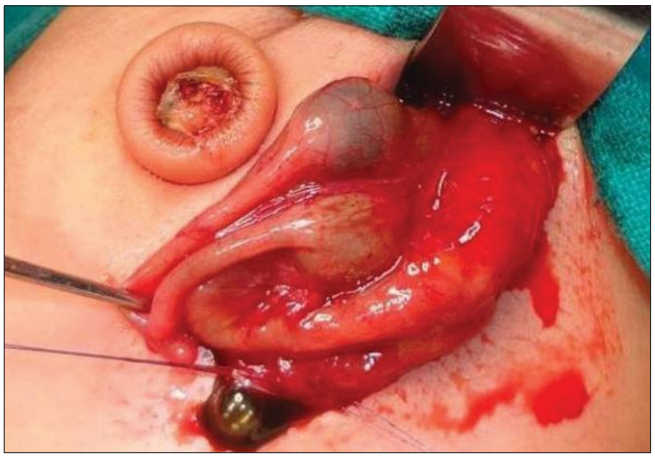


Figure 8: Newborn male with Type II congenital pouch colon, Meckel's diverticulum, Type C duplex appendix and unilateral intra-abdominal testis

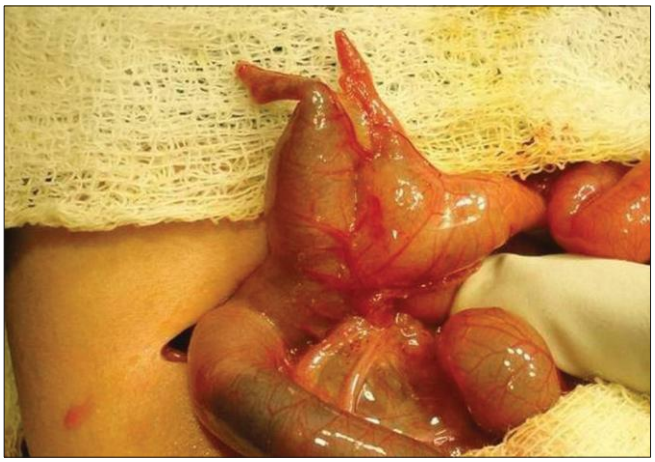


Figure 9: Newborn male with Type III congenital pouch colon and Type C appendiceal duplication

frequent findings in Type II CPC. All four patients of Type III CPC with appendiceal duplication (36.36%) had Type C duplication [Figure 9]. The appendix was normal in all patients with Type IV CPC. In 10/41 (24.4%) patients with Type II CPC, in all of whom the caecum alone or with a very short

Table 1: Classification of anomalies of the vermiform appendix[18]

Appendiceal anomaly
Agenesis: Absence of appendix
Multifarious appendices
A: Partial duplication or complete duplication on the normal site of a normal caecum.
A1 Partial duplication with both appendices sharing a common base like 'Y shaped' on a single caecum (bifid appendix)
A2 Complete duplication with both appendices side by side on normal site of single caecum
B: Complete duplication of the appendix on a single caecum
B1 Avian type or 'bird-like appendix': Two appendices symmetrically placed on either side of the ileocaecal valve
B2 Taeni coli cecum type: One appendix arising from the usual site of the caecum and the other arising from the caecum along the taenia
B3 Taenia coli hepatic flexure type: One appendix arising from the usual site of the caecum and the other arising from the hepatic flexure of the colon along the taenia
B4 Taenia coli splenic flexure type: One appendix arising from the usual site of the caecum and the other arising from the splenic flexure of the colon along the taenia
C: Duplication of the caecum, each has its own appendix
D: Triplex appendix: Complete triplication of appendix on the caecum
Shape anomalies
Horseshoe appendix
Frontal disposition of mesoappendix
Sagittal disposition of mesoappendix

Table 2: The vermiform appendix in patients with congenital pouch colon (*n*=74)

Type of CPC	Normal, single appendix, *n* (%)	Short, stubby appendix, *n* (%)	Absent appendix, *n* (%)	Appendiceal duplication		
				A2, *n* (%)	B1, *n* (%)	C, *n* (%)
Type I CPC (*n*=12)			12 (100)			
Type II CPC (*n*=41)	10 (24.4)	11 (26.8)	7 (17.1)	9 (22) Including 4 with short, stubby appendices	3 (7.3) Including 1 with one appendix shorter than normal	1 (2.4)
Type III CPC (*n*=11)	6 (54.54)	1 (9.09)				4 (36.36)
Type IV CPC (*n*=10)	10 (100)					
Total cases (*n*=74)	26 (35.1)	12 (16.2)	19 (25.7)	9 (12.2)	3 (4.0)	5 (6.75)

CPC: Congenital pouch colon

length of normal colon was present, the caecum and normal colon appeared dilated [Figures 2, 6 and 7].

Associated abnormalities

Associated abnormalities included minor sacral abnormalities (*n* = 5, 6.76%), hypospadias (*n* = 4, 11.1% of males), unilateral undescended testis (*n* = 2, 5.6% of males), unilateral/bilateral hydroureteronephrosis (*n* = 9, 12.15%) and unilateral hydronephrosis (*n* = 2, 2.7%). In 11 patients, the urinary bladder appeared large and flaccid. Five patients (6.8%), all with Type II CPC, had a Meckel's diverticulum [Figures 3,5 and 8] which, in one patient, was very small [Figure 3]. In two patients, the Meckel's diverticulum was just 4–5 cm proximal to the ileocaecal junction [Figure 5]. One patient with Type II CPC had a double Meckel's diverticulum.

Discussion

During normal embryogenesis, a diverticulum representing the anlage of the caecal apparatus appears as a conical pouch on the antimesenteric border of the caudal limb of the midgut loop, just beyond its apex,[22] at the 5th–6th week of foetal life.[22-24] A fully developed VA is seen in the 10-week-old embryo.[25]

The ceco-appendiceal outgrowth is capable of considerable morphological variations, and according to Robinson,[26] its morphological instability accounts for variations in length, the failure to differentiate and the phenomenon of double or triple appendix.

Appendiceal agenesis is usually an incidental finding during laparotomy[27] although it has been reported in a case with multiple major anomalies including congenital absence of the mesentery[28] and in a patient of congenital short bowel with malrotation.[29] In another report, a child with left-sided gastroschisis had the absence of the caecum and VA, shortened large bowel with no distinction of small and large bowel junction and intestinal malrotation.[30]

Nageswaran *et al*.[31] reviewed the literature on appendiceal duplications and identified 141 cases. Categorising by the Cave–Wallbridge classification,[15] there were 22 Type A, 8 Type B1, 46 Type B2 and 10 Type C cases.[31] In another recent review, Ayoub *et al*.[32] described a case of Type B1 appendiceal duplication and reported the frequency of Type A duplication to be 18%, Type B: 59% (Type B1: 6%, Type B2: 37% and Type B unclassified: 15%), Type C: 8%, horseshoe: 5% and

triple appendix: 2%, whereas 5% were unclassified. A common finding was that, as compared to the other varieties, Type B1 and C appendiceal duplications were relatively uncommon.[31,32] Type B1 and C duplications are also more likely to be associated with other intestinal, genito-urinary (GU), vertebral or other malformations.[31,33-35] Yanar *et al*.[36] felt that the possible relationship between anorectal-GU malformations and appendiceal duplications could be explained by the close anatomic association of the distal hindgut and the urorectal septum (URS) in the embryologic origin. Type B2 duplication, the most common variety and the one most likely to be misdiagnosed or mismanaged, is usually unrelated to other congenital anomalies.[31]

The association of appendiceal abnormalities with CPC has been reported earlier.[1-5,8,13,19,37] In a review of 566 cases of CPC, the VA was absent in 61 cases (10.8%), whereas a double appendix was present in 34 patients (6%).[8] Narasimharao *et al*.[1] reported 56 cases of 'pouch colon syndrome' in whom the VA was often absent (n = 6, 10.7%) or duplicated (n = 8, 14.3%). The true incidence of appendiceal anomalies may be underestimated as records are often incomplete, and our study, in contrast to earlier reports, showed appendiceal abnormalities in 48/74 (64.9%) cases. In addition, in several large series,[1,2,4,19] the type of appendiceal abnormality has not been described clearly or correlated with the subtype of CPC. However, the absence of the VA has been reported with Type I CPC,[38-41] and in several reports, Type II CPC was associated with Type A2 appendiceal duplication[42] or Type B1/C duplication.[34,43-46] Arestis *et al*.[47] reported a boy with Type II/III CPC (normal colon 12 cm at 5 years of age), short stubby appendix and malrotation. Sangkhathat *et al*.[48] described a case of Type III CPC with Type C appendiceal duplication. In a recent report, Mathur *et al*.[49] reported 4 girls with Type II CPC and cloaca who had appendiceal duplication (Type A2 = 3, including a case in which one of the appendices was short and stubby, and Type C = 1).

CPC is believed to result from a very 'early' error in the differentiation and growth of the mesenchyme of the URS, the adjacent developing hindgut and post-arterial limb of the midgut with the failure of rostro-caudal growth of the URS leading to the arrest of cloacal septation at a very early stage.[5] The early error in mesodermal differentiation in the adjacent post-arterial limb of the midgut and the hindgut could lead to its 'dysmorphogenesis' or 'defective development' before it lengthens[13,19,50] resulting in CPC and explaining the high incidence of associated abnormalities of the post-arterial limb of the midgut and hindgut. In Type I CPC, the shortening and abnormal development involve the entire colon. This abnormal segment later dilates to form the colonic pouch, and it is very likely that the anlage of the caecum and VA gets incorporated into the dilated segment, leading to appendicular agenesis. In the more common Type II CPC, in our study, appendiceal agenesis was less frequent (7/41, 17.1%), with almost a fourth of patients having a single, normal VA. A short stubby VA was, however, present in 11 patients (26.8%), perhaps due to incorporation of the proximal part of the appendicular anlage into the frequently dilated caecum that was observed with this subtype. If the dysmorphogenesis and dilatation lead to splitting of the appendicular anlage, appendicular duplication may occur, as shown by 31.7% of cases of Type II CPC and 36.4% of cases of Type III CPC having Type A2/B1/C appendicular duplication, the Type C duplication probably resulting from splitting of the entire caecal diverticulum. In 4 (9.8%) cases of Type II CPC, both the Type A2 duplicated appendices were short and stubby, perhaps due to a combination of the processes outlined above.

Our study shows that appendiceal duplication in Type II/III CPC was most frequently Type A2, followed by Type C and B1 duplication. These different subtypes of duplex appendix probably result from differences in the degree of the dilatation of the affected colonic segment and the location of the appendicular anlage. A closer study also reveals that there is little difference between Type B1 and Type C duplications. In Type B1 duplication, the duplicated appendices are often perched on separate small outpouchings of the caecum, quite similar to the picture in Type C duplications. This is also evident from a study of some reports of appendiceal duplication in patients of CPC[44,45] as well as a few cases of Type B1 duplex VA in patients without CPC.[5,32,51] Significantly, CPC does not appear to be associated with bifid (Type A1)[18] appendiceal duplication, Type B2-B4 duplication or other appendicular anomalies. These anomalies, frequently described in isolated case reports, are probably the result of a separate embryological error and are not frequently associated with other major organ malformations.[31]

Conclusions

Appendiceal abnormalities are frequently associated with Type I–III CPC and may result from the consequence of the abnormal development and dilatation, along with shortening, of a varying length of colon in CPC. Other anomalies of the post-arterial limb of the midgut and hindgut and the GU tract may also be present. All cases of Type I CPC had appendicular agenesis, whereas a short stubby appendix or Type A2/B1/C appendicular duplication was frequent in Type II and III CPC. All patients with Type IV CPC had a normal VA. As in other reports of appendiceal duplication associated with major intestinal, GU or vertebral malformations, patients with CPC had Type A2/B1/C appendiceal duplication.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient (s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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