

血清 PCT 和 hs-CRP 水平对急性脑梗死患者伴发感染的预测价值

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【摘要】 目的 分析急性脑梗死患者早期降钙素原(PCT)和超敏C-反应蛋白(hs-CRP)的变化趋势,并探讨二者对急性脑梗死伴发感染的预测价值。方法 选择2014年5月至2019年5月肥城矿业中心医院神经内科收治的206例急性脑梗死患者,收集患者临床资料及发病24、48、72 h血清PCT和hs-CRP水平。根据发病5 d内是否发生感染将患者分为感染组($n=69$)和非感染组($n=137$)。并以同期60例健康体检者作为健康对照组。分析各组血清PCT和hs-CRP水平的变化趋势;采用受试者工作特征曲线(ROC)分析血清PCT和hs-CRP水平在鉴别急性脑梗死是否合并感染中的价值。结果 感染组和非感染组患者24、48、72 h血清PCT水平均显著高于健康对照组,且感染组48 h和72 h血清PCT水平进一步高于非感染组($\mu\text{g/L}$: 0.28 ± 0.08 比 0.19 ± 0.03 , 0.31 ± 0.07 比 0.15 ± 0.06 , 均 $P < 0.05$)。与24 h时相比,感染组患者48 h和72 h时血清PCT水平明显升高,而非感染组则明显降低。感染组患者24、48、72 h血清hs-CRP水平均明显高于非感染组和健康对照组(mg/L : 5.86 ± 1.73 比 5.45 ± 1.08 , 5.25 ± 1.33 , 8.01 ± 2.41 比 5.67 ± 2.13 , 5.25 ± 1.33 , 14.25 ± 2.19 比 12.30 ± 1.87 , 5.25 ± 1.33 , 均 $P < 0.05$),而非感染组仅72 h时血清hs-CRP水平显著高于健康对照组。与24 h时相比,感染组和非感染组患者48 h、72 h血清hs-CRP水平均明显升高。ROC曲线分析显示,24 h时血清PCT和hs-CRP水平对急性脑梗死患者伴发感染不具有预测价值[ROC曲线下面积(AUC)为0.440、0.576,均 $P > 0.05$]。48 h时血清PCT诊断急性脑梗死伴感染的AUC为0.850 [95%可信区间(95%CI)=0.784~0.916],当截断值为0.25 $\mu\text{g/L}$ 时敏感度为66.7%,特异度为97.8%;血清hs-CRP诊断的AUC为0.759(95%CI=0.689~0.830),当截断值为6.80 mg/L 时敏感度为66.7%,特异度为76.6%;二者联合时AUC为0.911(95%CI=0.859~0.964),敏感度为90.5%,特异度为86.9%。72 h时血清PCT诊断急性脑梗死伴感染的AUC为0.952(95%CI=0.916~0.989),当截断值为0.23 $\mu\text{g/L}$ 时敏感度为89.9%,特异度为93.4%;血清hs-CRP诊断的AUC为0.753(95%CI=0.678~0.828),当截断值为14.01 mg/L 时敏感度为60.9%,特异度为83.2%;二者联合时AUC为0.954(95%CI=0.918~0.991),敏感度为97.1%,特异度为89.8%。表明48 h和72 h血清PCT的诊断价值高于hs-CRP,而PCT联合hs-CRP的预测价值高于单一指标。结论 急性脑梗死本身会影响血清PCT的产生;发病后72 h血清PCT水平高于0.23 $\mu\text{g/L}$ 并参考血清hs-CRP水平,对判断急性脑梗死患者发生感染具有较高的预测价值。

【关键词】 急性脑梗死; 感染; 降钙素原; 超敏C-反应蛋白; 预测

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Predictive value of serum procalcitonin and hypersensitive C-reactive protein levels in patients with acute cerebral infarction complicated with infection

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【Abstract】 Objective To analyze the changes of early procalcitonin (PCT) and hypersensitive C-reactive protein (hs-CRP) in patients with acute cerebral infarction, and to explore the predictive value of both for acute cerebral infarction with infection. **Methods** 206 acute cerebral infarction patients admitted to the department of neurology of Feicheng Mining Center Hospital from May 2014 to May 2019 were enrolled. Clinical data of patients and serum PCT and hs-CRP levels at 24, 48 and 72 hours after onset were collected. Patients were divided into infected group ($n = 69$) and non-infected group ($n = 137$) according to whether infection occurred within 5 days after onset. And 60 healthy people in the same period were selected as the healthy control group. The trends of serum PCT and hs-CRP levels in each group were analyzed. The receiver operating characteristic (ROC) curve was used to analyze the values of serum PCT and hs-CRP levels in identifying acute cerebral infarction with infection. **Results** The serum level of PCT at 24, 48 and 72 hours in the infected group and the non-infected group were significantly higher than those in the healthy control group, and the serum level of PCT at 48 hours and 72 hours in the infected group were significantly higher than those in the non-infected group ($\mu\text{g/L}$: 0.28 ± 0.08 vs. 0.19 ± 0.03 , 0.31 ± 0.07 vs. 0.15 ± 0.06 , both $P < 0.05$). Compared with 24 hours, the serum PCT level in the infected group at 48 hours and 72 hours were significantly increased, but decreased in the non-infected group. The serum hs-CRP level in the infected group at 24, 48 and 72 hours were significantly higher than

those in the non-infected group and the healthy control group (mg/L: 5.86 ± 1.73 vs. 5.45 ± 1.08 , 5.25 ± 1.33 ; 8.01 ± 2.41 vs. 5.67 ± 2.13 , 5.25 ± 1.33 ; 14.25 ± 2.19 vs. 12.30 ± 1.87 , 5.25 ± 1.33 ; all $P < 0.05$). And the serum hs-CRP level in the non-infected group at 72 hours was significantly higher than that in the healthy control group. Compared with 24 hours, the serum hs-CRP level in the infected group and non-infected group at 48 hours and 72 hours were significantly increased. It was shown by ROC curve analysis that serum PCT and hs-CRP levels at 24 hours had no predictive value for infection in patients with acute cerebral infarction [area under ROC curve (AUC) was 0.440, 0.576 respectively, both $P > 0.05$]. At 48 hours, the AUC of serum PCT in diagnosis of acute cerebral infarction with infection was 0.850 [95% confidence interval (95%CI) = 0.784–0.916], the sensitivity and specificity were 66.7% and 97.8% when the cut-off of PCT was 0.25 $\mu\text{g/L}$; the AUC of serum hs-CRP was 0.759 (95%CI = 0.689–0.830), the sensitivity and specificity were 66.7% and 76.6% when the cut-off of hs-CRP was 6.80 mg/L; the AUC of PCT combined with hs-CRP was 0.911 (95%CI = 0.859–0.964), the sensitivity was 90.5%, the specificity was 86.9%. At 72 hours, the AUC of serum PCT in diagnosis of acute cerebral infarction with infection was 0.952 (95%CI = 0.916–0.989), the sensitivity and specificity were 89.9% and 93.4% when the cut-off of PCT was 0.23 $\mu\text{g/L}$; the AUC of serum hs-CRP was 0.753 (95%CI = 0.678–0.828), the sensitivity and specificity were 60.9% and 83.2% when the cut-off of hs-CRP was 14.01 mg/L; the AUC of PCT combined with hs-CRP was 0.954 (95%CI = 0.918–0.991), the sensitivity was 97.1%, and the specificity was 89.8%. The results showed that the diagnostic value of serum PCT at 48 hours and 72 hours were higher than those of hs-CRP, and the predictive value of PCT combined with hs-CRP was higher than those of single index. **Conclusion** Acute cerebral infarction itself has an effect on serum PCT level; serum PCT level above 0.23 $\mu\text{g/L}$ at 72 hours after onset and reference to serum hs-CRP level have a high predictive value for the diagnosis of infection in patients with acute cerebral infarction.

【Key words】 Acute cerebral infarction; Infection; Procalcitonin; Hypersensitive C-reactive protein; Prediction

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近年来急性脑梗死发病率逐年上升,且呈年轻化趋势,随着临床诊疗技术的进步,多数患者在采取有效治疗后预后良好^[1],然而仍有 16%~64% 的患者在脑卒中后发生感染,且是影响患者预后不良的危险因素^[2]。由于在发生脑梗死时可能会导致中枢性发热^[3],因而增加了判断是否发生感染的难度。降钙素原(PCT)作为血清降钙素的前体物质,在健康者血清中含量甚微甚至检测不到,而在发生细菌性感染患者血清中显著升高,且与炎症程度有关,并随治疗而变化^[4-6]。超敏C-反应蛋白(hs-CRP)作为非特异性急性相反应蛋白,是预测细菌性感染的辅助性指标,但缺乏特异性^[7-8]。本研究旨在分析急性脑梗死患者早期血清中PCT和hs-CRP水平的变化,并探讨二者对急性脑梗死患者伴发感染的预测价值。

1 资料与方法

1.1 研究对象:选择2014年5月至2019年5月在本院神经内科治疗的急性脑梗死患者;同期,从体检中心选取健康体检者60例作为健康对照组。

1.1.1 纳入标准:①符合第四届全国脑血管病学术会议修订的脑梗死诊断标准;②发病至入院时间在24h内;③首次发病,且发病年龄在18岁以上。

1.1.2 排除标准:①出血性脑卒中、短暂性脑缺血发作者;②大手术后、严重创伤、休克、心肺复苏、急性呼吸窘迫综合征、烧伤、创伤、神经系统肿瘤、

胰腺炎等伴有可能引起PCT升高的因素者;③严重肝肾功能障碍者;④近7d内有使用促炎因子释放药者;⑤伴有高血压、糖尿病、心血管疾病以及各种急慢性感染者。

1.2 伦理学:本研究符合医学伦理学标准,并通过医院伦理委员会批准(批准号:ZZYX201404083322),所有治疗及检测均获得过患者或家属知情同意。

1.3 观察指标:收集患者性别、年龄、身高、体重、体重指数(BMI)、烟酒嗜好、合并内科慢性病、血压,入院时美国国家卫生研究院卒中量表(NIHSS)评分、TOAST分型、空腹血糖(FBG)、血脂〔总胆固醇(TC)、甘油三酯(TG)、低密度脂蛋白胆固醇(LDL-C)、高密度脂蛋白胆固醇(HDL-C)〕等生化指标,发病24、48、72h时血清PCT(双抗体夹心法)和hs-CRP〔酶联免疫吸附试验(ELISA)〕水平,以及感染相关指标。

1.4 分组:根据发病5d内是否发生感染,将患者分为感染组和非感染组。

1.5 统计学方法:利用SPSS 21.0软件分析数据。计量资料行正态性检验,符合正态分布的资料以均数 \pm 标准差($\bar{x} \pm s$)表示,两组间比较采用 t 检验,多组间比较采用单因素方差分析和LSD- t 检验;计数资料采用 χ^2 检验。用受试者工作特征曲线(ROC)分析血清PCT和hs-CRP水平鉴别急性脑梗死是否合并感染的价值。 $P < 0.05$ 为差异有统计学意义。

2 结果

2.1 基线资料(表1):共纳入206例急性脑梗死患者,其中感染组69例,男性40例、女性29例,年龄(56.6±8.4)岁;非感染组137例,男性75例、女性62例,年龄(58.4±8.9)岁;健康对照组60例,男性37例、女性23例,年龄(57.9±8.7)岁。3组性别、年龄差异均无统计学意义(均 $P>0.05$),具有可比性。69例感染患者体温均 $>37^{\circ}\text{C}$,其中上呼吸道感染28例,肺部感染19例,泌尿系统感染16例,急性肠炎5例,皮肤感染1例。在急性脑梗死患者中,感染组与非感染组BMI、高血压、糖尿病、吸烟史、饮酒史、血压、NIHSS评分、TOAST分型、FBG、TC、TG、LDL-C和HDL-C比较差异均无统计学意义(均 $P>0.05$)。

表1 感染组与非感染组急性脑梗死患者基线资料比较

指标	感染组 (n=69)	非感染组 (n=137)	t/ χ^2 值	P值
年龄(岁, $\bar{x}\pm s$)	56.6±8.4	58.4±8.9	1.395	0.164
男性[例(%)]	40(58.0)	75(54.7)	0.194	0.660
BMI(kg/m ² , $\bar{x}\pm s$)	23.26±2.30	23.19±3.12	0.174	0.862
高血压[例(%)]	50(72.5)	103(75.2)	0.177	0.674
糖尿病[例(%)]	20(29.0)	48(35.0)	0.760	0.383
吸烟史[例(%)]	19(27.5)	33(24.1)	0.289	0.591
饮酒史[例(%)]	11(15.9)	18(13.1)	0.298	0.585
收缩压(mmHg, $\bar{x}\pm s$)	153.98±13.05	151.41±14.23	1.261	0.209
舒张压(mmHg, $\bar{x}\pm s$)	81.27±5.81	80.36±7.20	0.910	0.364
NIHSS评分(分, $\bar{x}\pm s$)	7.46±2.81	6.78±2.31	1.859	0.064
TOAST分型[例(%)]			1.426	0.700
大动脉粥样硬化	18(26.1)	40(29.2)		
心源性梗死	19(27.5)	42(30.7)		
小动脉闭塞	10(14.5)	22(16.0)		
其他病因	22(31.9)	33(24.1)		
FBG(mmol/L, $\bar{x}\pm s$)	4.88±0.41	4.99±0.46	1.641	0.102
TC(mmol/L, $\bar{x}\pm s$)	5.04±0.75	5.08±0.83	0.357	0.721
TG(mmol/L, $\bar{x}\pm s$)	1.72±0.30	1.75±0.26	0.616	0.539
LDL-C(mmol/L, $\bar{x}\pm s$)	3.82±0.92	3.61±0.84	1.661	0.098
HDL-C(mmol/L, $\bar{x}\pm s$)	1.97±0.39	1.88±0.38	1.646	0.101

注: BMI为体重指数, NIHSS为美国国家卫生研究院卒中量表, FBG为空腹血糖, TC为总胆固醇, TG为甘油三酯, LDL-C为低密度脂蛋白胆固醇, HDL-C为高密度脂蛋白胆固醇; 1 mmHg=0.133 kPa

2.2 急性脑梗死患者与健康者血清PCT、hs-CRP水平比较(表2):感染组和非感染组发病24、48、72 h血清PCT水平均显著高于健康对照组,且感染组48 h和72 h血清PCT水平明显高于非感染组(均 $P<0.05$);与本组24 h时相比,感染组48 h和72 h血清PCT水平逐渐升高,而非感染组逐渐降低(均 $P<0.05$)。感染组24、48、72 h时血清hs-CRP水平显著高于非感染组和健康对照组,而非感染组仅72 h时血清hs-CRP水平高于健康对照组(均 $P<0.05$);与本组24 h时相比,感染组和非感染组48 h、72 h血清hs-CRP水平均明显升高(均 $P<0.05$)。

表2 是否伴感染两组急性脑梗死患者与健康对照组各时间点血清PCT和hs-CRP水平变化比较($\bar{x}\pm s$)

组别	例数 (例)	PCT($\mu\text{g/L}$)		
		24 h	48 h	72 h
感染组	69	0.24±0.04 ^a	0.28±0.08 ^{abc}	0.31±0.07 ^{abcd}
非感染组	137	0.26±0.08 ^a	0.19±0.03 ^{ac}	0.15±0.06 ^{acd}
健康对照组	60	0.07±0.02	0.07±0.02	0.07±0.02
F值		202.493	324.581	445.037
P值		<0.001	<0.001	<0.001

组别	例数 (例)	hs-CRP(mg/L)		
		24 h	48 h	72 h
感染组	69	5.86±1.73 ^{ab}	8.01±2.41 ^{abc}	14.25±2.19 ^{abcd}
非感染组	137	5.45±1.08	5.67±2.13 ^c	12.30±1.87 ^{acd}
健康对照组	60	5.25±1.33	5.25±1.33	5.25±1.33
F值		8.200	37.466	425.816
P值		<0.001	<0.001	<0.001

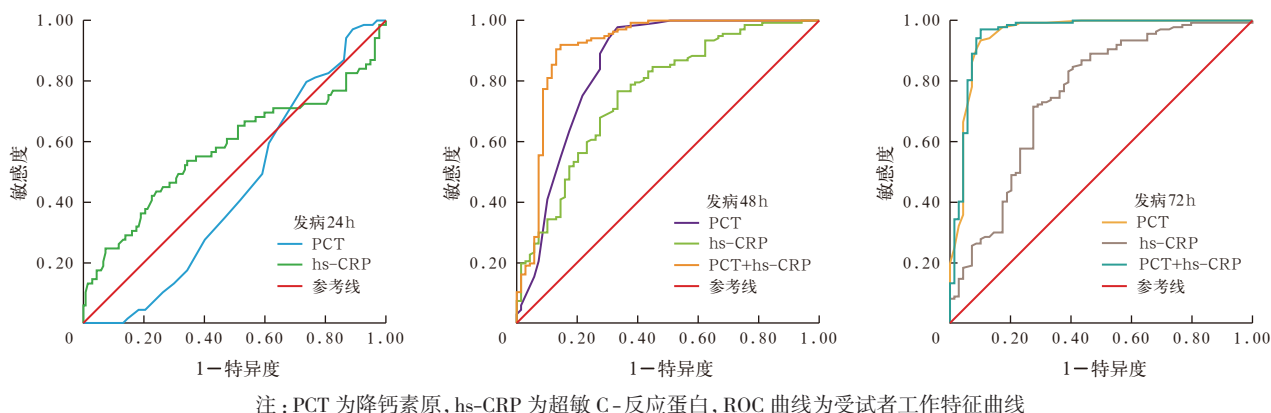
注: PCT为降钙素原, hs-CRP为超敏C-反应蛋白;与健康对照组比较,^a $P<0.05$;与非感染组比较,^b $P<0.05$;与本组24 h比较,^c $P<0.05$;与本组48 h比较,^d $P<0.05$

2.3 血清PCT和hs-CRP水平对急性脑梗死患者伴发感染的预测价值(表3;图1):ROC曲线分析显示,24 h时血清PCT和hs-CRP水平对急性脑梗死患者伴发感染不具有预测价值。48 h和72 h血清PCT、hs-CRP水平对急性脑梗死患者伴发感染均有预测价值(均 $P<0.01$),且二者联合的预测价值高于单一指标。

表3 发病后不同时间点血清PCT、hs-CRP水平对急性脑梗死患者伴发感染的预测价值

指标	AUC	95%CI	P值	截断值	敏感度(%)	特异度(%)	准确性(%)	阳性预测值(%)	阴性预测值(%)	阳性似然比	阴性似然比
24 h PCT	0.440	0.362~0.517	0.157								
24 h hs-CRP	0.576	0.485~0.666	0.076								
48 h PCT	0.850	0.784~0.916	<0.001	0.25	66.7	97.8	82.3	96.8	74.6	30.32	0.34
48 h hs-CRP	0.759	0.689~0.830	<0.001	6.80	66.7	76.6	71.7	74.0	69.7	2.85	0.43
48 h PCT+hs-CRP	0.911	0.859~0.964	<0.001		90.5	86.9	76.8	87.4	90.1	6.91	0.11
72 h PCT	0.952	0.916~0.989	<0.001	0.23	89.9	93.4	91.7	93.2	90.2	13.62	0.11
72 h hs-CRP	0.753	0.678~0.828	<0.001	14.01	60.9	83.2	72.1	78.4	68.0	3.63	0.47
72 h PCT+hs-CRP	0.954	0.918~0.991	<0.001		97.1	89.8	93.4	90.5	96.9	9.52	0.03

注: PCT为降钙素原, hs-CRP为超敏C-反应蛋白, AUC为受试者工作特征曲线下面积, 95%CI为95%可信区间;空白代表无此项



注: PCT 为降钙素原, hs-CRP 为超敏 C-反应蛋白, ROC 曲线为受试者工作特征曲线

图1 发病 24、48、72 h 时的血清 PCT 和 hs-CRP 水平预测急性脑梗死患者伴发感染的 ROC 曲线

3 讨论

目前临床上尚无诊断脑梗死急性期合并感染的特异性指标。有研究表明,急性脑梗死合并感染患者血白细胞计数(WBC)、中性粒细胞计数与非感染患者差异无统计学意义^[9]。PCT作为一种无激素活性的糖蛋白,多在细菌感染时产生,近年被作为相对特异性指标用于细菌感染性疾病的诊断^[10-11]。有研究表明,PCT升高与细菌感染严重程度显著相关,且可作为预测细菌感染及严重程度的指标^[12-14]。但有些基础疾病也可能导致血清PCT水平升高,有研究显示,脑外伤患者24h时血清PCT水平显著升高^[15];胰腺炎患者血清PCT水平也可出现非特异性升高^[16]。董智强等^[17]指出,PCT能够及时地反馈急性脑梗死患者的病情,并指导治疗,能够用于判断患者的预后状况。对于存在其他因素可导致PCT升高的患者,单纯依靠PCT水平作为抗菌治疗的依据,可能会导致滥用抗菌药物,且不利于患者康复^[18]。脑梗死急性期作为一种急性相危重病,是否疾病本身会引起血清PCT水平升高尚无定论。

本研究在排除感染组与非感染组急性脑梗死患者一般资料差异后,对两组患者发病24、48和72h血清PCT水平进行监测,结果显示,感染组和非感染组患者24、48、72h时血清PCT水平均显著高于健康对照组,且感染组各时间点PCT水平进一步高于非感染组;与24h时相比,感染组患者48h和72h时血清PCT水平均显著升高,而非感染组则明显降低。表明急性脑梗死本身可能会导致血清PCT水平改变,且随着时间推移,非感染患者血清PCT水平呈下降趋势,72h时下降最为明显;而感染患者血清PCT水平则呈上升趋势,72h时最为显著。ROC曲线分析显示,急性脑梗死患者24h时血清PCT水

平对伴发感染不具有诊断作用;48h和72h时血清PCT水平对急性脑梗死患者伴发感染均有预测价值,且72h时PCT预测的曲线下面积(AUC)、敏感度和特异度均优于48h时,说明急性脑梗死发病72h时血清PCT水平在诊断患者是否合并感染中意义最大。

hs-CRP作为非特异性急性相反应蛋白,在应激、组织损伤、感染等病理情况下会出现血清水平升高。有研究显示,急性脑梗死患者血清hs-CRP水平显著升高,且与病情严重程度有关^[19];亦有研究表明,急性脑梗死患者急性期血清hs-CRP水平与脑梗死的预后相关,且对其预后的预测价值良好^[20]。本研究显示,感染组24、48、72h时血清hs-CRP水平较非感染组和健康对照组显著升高,而非感染组仅72h时血清hs-CRP水平较健康对照组显著升高;与24h时相比,感染组和非感染组患者48h、72h血清hs-CRP水平均显著升高。提示急性脑梗死本身可导致血清hs-CRP水平升高,且随病情进展逐渐升高^[21];感染患者更易出现hs-CRP水平升高,且24h时已出现显著升高。Okazaki等^[22]曾指出,急性脑梗死患者发病24h、48h时CRP水平升高与感染所致发热有关,与本研结论一致。ROC曲线分析显示,急性脑梗死患者24h时血清hs-CRP水平对伴发感染不具有诊断作用;48h和72h时血清hs-CRP水平预测感染的AUC、敏感度和特异度均不佳,说明单纯依靠血清hs-CRP水平变化并不能很好地鉴别急性脑梗死患者是否发生感染;48h、72h时将血清PCT与hs-CRP联合预测时,可进一步提高单一指标诊断伴发感染的敏感度和特异度。

综上所述,急性脑梗死伴发感染和非感染患者发病72h内均出现血清PCT水平升高,但在非感染

患者血清中呈进行性下降趋势;在利用 PCT 判定急性脑梗死患者早期是否发生感染时需考量疾病本身对 PCT 的影响,以发病后 72 h 时血清 PCT 水平高于 0.23 $\mu\text{g/L}$ 作为临界值,并参考血清 hs-CRP 水平,对判断急性脑梗死患者是否合并感染意义最大。本研究作为单中心研究,样本量有限,且观察时间点有限,尚需进一步开展大样本量、多中心的研究予以证实,但本研究给我们提供了一个重要信息,即急性脑梗死患者发病 72 h 内血清 PCT 水平变化规律对判断患者早期是否合并感染意义重大。未来随着分子生物学技术的不断进步,也可能发现更多的指标用于鉴别诊断急性脑梗死患者早期是否合并感染。

利益冲突 作者声明不存在利益冲突

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